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A SYSTEM OF VETERINARY MEDICINE

"Together let us beat this ample field,
Try what the open, what the covert yield."
POPE.

"Look to the essence of a thing, whether it be a point of doctrine, of practice, or of interpretation."
MARCUS AURELIUS ANTONINUS: *Meditations*, viii. 22.

A SYSTEM OF VETERINARY MEDICINE

BY VARIOUS WRITERS

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VOL. I

MICROBIAL DISEASES

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TO

WILLIAM HUNTING, F.R.C.V.S.

AS A SLIGHT TRIBUTE TO HIS LABOURS IN THE CAUSE OF VETERINARY SCIENCE,

HIS VALUABLE ADDITIONS TO VETERINARY LITERATURE, AND

HIS SKILL AS A CLINICIAN,

THIS WORK IS DEDICATED BY THE EDITOR

PREFACE TO VOL. I

To produce a work on veterinary medicine which fulfils the functions of a textbook for the student, and is likely to prove useful to the practitioner for reference, is by no means an easy task in the present day, for not only is the subject an extensive and progressive one, but also it includes the consideration of many diseases which, in former times, owing to a limited knowledge of their etiology, pathology, and diagnosis, could only be discussed within narrow limits. But at the present time, in consequence of the researches of the pathologist, combined with the observations of the clinician, these diseases have been so thoroughly investigated that each requires to be dealt with by an author who has devoted special attention to individual subjects, and has had considerable clinical experience in dealing with the various phases that are presented in the affection he undertakes to describe.

In fact, it can truly be said that no individual, even though he possessed the widest experience of diseases in animals and had unlimited time at his disposal, could, unaided, either compile or write a work on veterinary medicine so as to do anything like justice to the subject. Moreover, of late years the sphere of utility of the practitioner has been widely extended, and while formerly a knowledge of the diseases of horses and cattle was regarded as sufficient for the purposes of his calling, it is now essential that he should be acquainted with the ailments of the smaller animals, and also be proficient in the subject of preventive medicine and in public health duties.

It was after due consideration of the above facts that, acting on the suggestion of some professional friends, the present work was undertaken by the Editor. By securing the collaboration of a number of eminent British veterinarians, he has attempted to produce a work which, he ventures to hope, may prove useful to both practitioners and students of veterinary medicine in all English-speaking countries.

It may be considered by some that the subjects are treated rather too fully for the purposes of a textbook for students, but the day is past when a series of cut-and-dried facts concerning each disease sufficed for the needs of the student. It is necessary that he should be acquainted with the various phases presented by each affection, and with its atypical as well as its typical characteristics. The function of a textbook is to act as a guide to the clinical study and observation of disease, but, in addition to this, it should stimulate the student to think for himself, and to note every detail in connection with a case. To commit to memory a series of epigrammatic statements referring to etiology, symptoms, diagnosis, and treatment, is of little value to the student of veterinary medicine, as it leads him to the erroneous belief that every disease possesses a stereotyped list of symptoms, presents a characteristic clinical picture, and should be treated by hard-and-fast therapeutical rules.

The Editor does not consider that any apology is necessary for the amount of space devoted to diseases other than those of horses and cattle. The importance of canine and feline medicine in the present day has rendered such a course desirable and essential. Similar remarks apply to the articles on avian diseases, and the microbial affections of fish, etc. There was a time when such subjects were regarded as belonging to the sphere of the kennel-man, the gamekeeper, the empiric, and the patent-medicine vendor; but dog-fanciers and sportsmen are now beginning to appreciate the fact that the veterinary practitioner, by his scientific training, is the proper person to advise on all matters of this kind. Hence a modern work on veterinary medicine should supply full information on these subjects, so that a general practitioner may be enabled to refer to it with advantage when he is called on to investigate diseases which are outside the sphere of his ordinary practice.

In consequence of the rapid progress of veterinary medicine in America and in the Colonies, it has been deemed advisable to devote attention to diseases occurring in these countries. Tropical diseases are fully considered by specialists in the subject, and it is hoped that the work will prove of value to practitioners in India and Africa. The present volume is devoted to microbial diseases, as it seems desirable to class these affections together. In Volume II. the diseases of the various organs will receive consideration.

To all the collaborators the Editor begs to offer his sincere thanks. They have readily responded to his call for assistance, and, although busily engaged in professional duties, with increasing demands on their time, they have spared no efforts to render the work as useful as possible.

A large share of the task has fallen to Mr. Henry Gray, who has not only written on the subjects on which he has specialised, but also has read the proof-sheets as they passed through the press, and suggested many useful alterations and additions. His wide experience of canine medicine has enabled him to write authoritatively on this subject, and the sections on Canine Distemper, Canine Typhus, and other microbial affections of the dog, cannot but prove of marked value to practitioners interested in the diseases of this animal.

To Professor J. F. Craig, Royal Veterinary College of Ireland, the Editor is also deeply indebted for much assistance, many useful suggestions, and kind encouragement; also for writing the introduction to the section on Piroplasmosis, and arranging the subjects therein.

To Mr. W. Jackson Young, F.R.C.V.S., D.V.S.M., he is indebted for many valuable suggestions in connection with several of the sections.

The onerous task of preparing the index has been carried out by Mrs. Hoare with care and ability.

To all those professional friends who by their advice and encouragement helped in the preparation of the work the Editor desires to express his thanks, and to acknowledge their valuable assistance.

In the preparation of a work of this kind it was necessary to consult freely the various professional journals, and the Editor gladly acknowledges the large amount of information derived from the *Veterinary Record*, the *Veterinary News*, the *Veterinary Journal*, the *Journal of Comparative Pathology and Therapeutics*, and the *American Journal of Veterinary Medicine*.

For errors of omission and commission the Editor craves the indulgence of his readers. He is fully aware of the magnitude of the task he has undertaken, and the difficulties to be overcome. To those who expect perfection in a work of the kind he would point out that, being engaged in the daily duties of a general practitioner, he has not been able to devote as much time and energy as he would desire to the preparation of the sections for which he is responsible and to the editing of the entire material; and if, in this attempt to make a useful addition to existing veterinary literature, he has only succeeded in laying the foundation on which some abler hand may in the future erect a structure destined to be a reflex of current knowledge in veterinary medicine, he will feel that his labour has not been in vain.

E. W. H.

18, Cook Street,
Cork.
February, 1913.

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SYSTEM OF VETERINARY MEDICINE

SEPTICÆMIA

BY H. TUDOR HUGHES, B.Sc., M.R.C.V.S.,
Oswestry.

Synonyms.—Septic infection; Bacteræmia; Bacterial septicæmia.
French: Septicémie.
German: Septicämie; Septichämie.

GENERAL REMARKS.

In the first place, we may point out that there is no one particular organism specific to this condition or to pyæmia; various organisms may produce them, and more than one organism may be present at the same time. Also, the same organism may produce varying results under different circumstances—*e.g.*, a local abscess, multiple abscesses, or a general septicæmia. The result will depend upon the degree of virulence of the organism, the path of infection, and the age, species, and degree of resistance of the animal.

SEPTICÆMIA is the term applied to a bacterial infection in which the main seat of multiplication is the blood. Symptoms of general poisoning arise, but without the production of abscesses in the internal organs. In all cases of septicæmia the organisms are more numerous in the capillaries of the internal organs than in the peripheral circulation, and thus it may not always be possible to detect any in the blood during life.

In a surgical sense, the term "septicæmia" was formerly applied to a condition in which the blood and tissues of the body were invaded by pyogenic organisms; but in the present day the term is applied in a medical sense to any condition in which microbic invasion of the blood and tissues occurs, with or without a local site of infection, and without metastatic foci of suppuration.

BACTERIOLOGY.

The organisms most commonly responsible for this condition are the *Streptococcus pyogenes* and the *Staphylococcus pyogenes aureus*. Less common causes are the *Staphylococcus pyogenes albus* and the *Bacillus coli*.

1. STREPTOCOCCUS PYOGENES.—This organism is a facultative parasite having a ubiquitous distribution.

Microscopical Characters.—The individual cocci are about 1 μ in diameter, and arranged in linear series, forming chains of varying length, comparable to chains of beads. When rapid multiplication is in progress, the chain frequently appears as if it were made up of diplococci. In old cultures many of the cocci appear swollen up to twice the usual size, and are regarded as involution forms. It is non-motile.

Staining.—It stains by any of the basic aniline dyes, and also by the methods of Gram and Claudius.

Cultivation.—It grows rapidly at room temperature, but much more rapidly at body temperature. In gelatine a stab culture becomes visible about the second day as a thin whitish line, formed of minute rounded colonies. The growth does not spread on the surface, and the gelatine is not liquefied. On agar it forms very small translucent colonies. On potato there is no visible growth.

In bouillon the growth is almost entirely at the bottom, whitish, somewhat fluffy in appearance, and easily broken up by shaking.

2. STAPHYLOCOCCUS PYOGENES AUREUS.—Like the *Streptococcus pyogenes*, this is a facultative parasite with a ubiquitous distribution.

Microscopical Characters.—In size the individual cocci are just a little less than 1 μ (about 0·9 μ) in diameter. Growth takes place irregularly, so that the colonies have the appearance of irregular masses like bunches of grapes. It is non-motile.

Staining.—It stains readily with any of the basic aniline dyes, and also by the methods of Gram and Claudius.

Cultivation.—It grows readily at low summer temperatures, but much more rapidly at body temperature, and in or on all the common media. In gelatine a stab culture is visible as a streak along the whole needle-track within two days, and almost immediately liquefaction begins, starting at the top, and forming a funnel-shaped pool of liquid gelatine, turbid from suspended cocci. Liquefaction rapidly spreads throughout the whole of the medium, and the growth settles to the bottom as a bright yellow flocculent deposit. Liquefaction is due to a tryptic ferment elaborated by the cocci. On agar incubated at 37° C. a stroke culture is visible in twenty-four hours as a smooth, shining, yellowish line, which spreads over the surface and becomes bright orange in colour, resembling a streak of oil-paint. On potato it grows well, with a golden or orange yellow colour. In bouillon it produces a uniform turbidity, which eventually settles to the bottom as a brownish-yellow layer.

3. Staphylococcus Pyogenes Albus.—This organism is similar in character to the foregoing, except that its growth on all the media is white.

SYMPTOMS.

Septicæmia most commonly follows upon a local infection—*e.g.*, a wound, or a septic condition of the womb. At first the toxins alone are absorbed into the blood, the condition at this stage being designated a toxæmia, in contradistinction to the later stage, when the organisms are themselves present in the blood, the condition being then known as a septicæmia or a bacteræmia. When the toxins of putrefactive organisms are being absorbed into the blood, the condition is known as sapræmia (see p. 5). The symptoms are those of fever, sometimes ushered in by an attack of rigors. The temperature rises to 106° or 107° F., and remains high, with but slight remissions and no intermissions. In some cases there is no great hyperthermia, but these are the exception. The pulse is frequent, quick, and feeble, and the heart-sounds are weak.

Indications of general disturbance are present, there is complete loss of appetite, great debility and dulness, muscular tremors, and occasionally paralysis of the hind-quarters. The mucous membranes are dirty red, or may show icteric discoloration and ecchymoses. The urine is discoloured and albuminous. Towards the end there is continued diarrhœa, which may be blood-stained and accompanied by colicky pains.

As death approaches dyspnœa comes on, whilst the temperature may be extremely high; sometimes the temperature is subnormal, and this, with a very rapid pulse, is always a grave indication. The disease usually takes a few days to run its course, and recoveries are rare.

MORBID ANATOMY.

Decomposition sets in early, rigor mortis is feeble, and the tissues have a dark and livid appearance. The blood coagulates imperfectly, and is dark and tarry in colour; if allowed to stand, the serum which separates from the corpuscles is much stained from the breaking up of the red blood-corpuscles, which results from the action of the toxins. The endocardium and tunica intima of the larger vessels are stained dark purple or black. Most of the serous cavities contain a certain amount of blood-stained fluid, and most of the serous membranes bear well-marked petechiæ, especially under the pericardium and pleura. The lungs are deeply congested; the liver, spleen, and kidneys are enlarged, softened, and congested, particularly the spleen.

On microscopical examination bacteria are usually demonstrable in the blood and internal organs, especially in the spleen.

DIAGNOSIS.

Septicæmia is distinguished from pyæmia by the absence of repeated rigors and secondary abscesses, and by a more regular and continuous type of fever.

Sapræmia is always associated with an obvious focus of putrefaction, whilst septicæmia may occur with little local manifestation. Septic traumatic fever may be so severe as to give one the idea that septicæmia is setting in; but if only the former condition is present, it rapidly disappears on freely opening up and draining the wound.

PROGNOSIS.

The prognosis of septicæmia is always very grave, recoveries being quite exceptional.

TREATMENT.

Prophylactic measures against septicæmia consist in the thorough application of aseptic and antiseptic measures. Suppurating wounds, gangrenous centres, fistulæ, sinuses, and sloughing injuries, where discharges may become putrid, should be frequently cleansed with disinfectant solutions. If pus accumulates in a wound on account of the disposition and direction of the latter, a counter-opening should be made and drainage effected. Septicæmia and septic intoxications of uterine origin are prevented by antiseptic irrigations of the uterus, after detaching and removing the fœtal membranes, if these have not been expelled.

Curative treatment comprises attention to any local lesions, as well as internal medication. Any local centre of inflammation should first of all be dealt with; the part should be irrigated with or bathed in a strong antiseptic solution, or thoroughly soaked with hydrogen peroxide solution, and then dusted over with an antiseptic dry dressing. In some cases free incisions, evacuation of pus and putrid discharges, and provision for drainage may be necessary.

In cases of puerperal septicæmia the uterus should be irrigated at once with a disinfectant solution, which will need to be repeated once or twice a day. The best agents for disinfecting the womb are: lysol, 1 in 100; creolin, 1 or 2 in 100; chinosol, 1 in 500 to 1 in 1,000; hydrogen peroxide, 5 to 10 vols. per cent. After washing out the womb, it is a good plan to introduce an antiseptic pessary, which may be made up of chinosol, salol, boric acid, iodoform, or a combination of these, either in gelatine or cacao butter. In this way the uterus is rendered antiseptic for the time between the irrigations, and it is not necessary to irrigate so frequently.

Often it is impossible to apply surgical treatment in time, as infection of the blood has taken place already.

When the streptococcus is responsible for the trouble, as is most frequently the case, the polyvalent antistreptococcic serum may be tried.

Another method of treatment that has been put forward is the intravenous injection of large quantities of normal saline solution repeated two or three times a day; diuresis and diarrhœa are induced, and it is hoped that by this means the organisms and their products may be eliminated.

This treatment is likely to give better results in cases of sapræmia than in cases of septicæmia. If the intravenous injections of normal saline solution cannot be carried out, subcutaneous injections of a similar solution or saline enemata, repeated two or three times a day, may be adopted instead. Internal antiseptics may also be administered, such as carbolic acid, or large doses of solution of acetozone (1 to 1,000), but their value is doubtful.

It is important that the patient should be placed in hygienic surroundings and made comfortable; his mouth and nostrils should be sponged, plenty of suitable and easily digested food given, and everything possible done to coax him to feed.

Tonics and stimulants are the medicinal agents indicated. It is difficult to control the fever, and quinine is probably better than antifebrin or antipyrin for this purpose.

SAPRÆMIA.

Sapræmia is also known as septic intoxication. It arises from the absorption of toxins of putrefactive bacteria into the blood, and is essentially a toxæmia. It differs from septicæmia in the following respects:

In sapræmia micro-organisms are not present in the blood, and the blood is not infective—*i.e.*, if a small amount be inoculated into another animal, the disease is not reproduced.

In septicæmia the blood is infective, it contains micro-organisms, and if a minute amount taken even at a distance from the local focus be inoculated into another animal, the disease is transmitted thereto.

Sapræmia results from the presence of a mass of putrefying tissue or a collection of putrid fluid in the body, from which absorption of toxins may occur. Examples are found in retention of a decomposing placenta, punctured wounds accompanied by putrid suppuration of the underlying tissue, septic pneumonia, the presence of a putrefying blood-clot in the pleural or peritoneal cavity resulting from a wound, etc.

It must be remembered that a clinical distinction between sapræmia and septicæmia is not always possible; the former may lead on to the latter, or both may be combined.

The symptoms of sapræmia depend on the amount of the toxins absorbed. Rigors are observed, followed by a continuous high temperature, the constitutional symptoms being in proportion to the severity of the case.

The toxins in some instances act chiefly on the alimentary canal, causing diarrhœa and evidences of gastro-enteritis; in others the nervous system is acted on, marked prostration and a comatose condition resulting. The lesions resemble those described as occurring in septicæmia. Sapræmia tends to recovery if treatment be adopted early. Treatment is to be carried out on similar lines to those advised for septicæmia.

PYÆMIA

By H. TUDOR HUGHES, B.Sc., M.R.C.V.S.,
Oswestry.

Synonyms.—Septico-pyæmia.

French : Pyémie.
German : Pyämie.

Pyæmia is a condition the essential feature in which is the production of multiple abscesses in various parts of the body, the result of metastasis. It is not of such frequent occurrence as septicæmia.

Metastasis may occur by way of the lymphatics, in which case the glands are usually involved, or by way of the bloodvessels. In the latter case a few organisms may gain entrance into the blood from a local lesion, and settle in a favourable part or in a damaged tissue ; or a septic phlebitis may take place, with suppurative softening of the thrombus and resulting embolism ; or there may be direct extension along a vein, producing spreading thrombosis and suppuration within the vein.

In suppuration the two main features are—

1. The immigration of leucocytes—chiefly polymorphonuclear—owing to the positive chemotaxis or attraction exerted by the bacteria and their toxins.

2. The liquefaction of the histological elements of the affected tissue, with necrosis of the cells of the part.

The result is the replacement of the affected tissue by a creamy fluid called "pus," which varies in character according to the variety and virulence of the bacteria present.

The BACTERIOLOGY is the same as that described under Septicæmia, the *Streptococcus pyogenes* and the *Staphylococcus pyogenes aureus* being the causal organisms most frequently met with. When these organisms are of an exalted degree of virulence, they bring about septicæmia, and when of a lesser degree of virulence, pyæmia.

The cause of pyæmia may be said to be any condition which leads to the formation and detachment of infective emboli in the circulation ;

the commonest conditions occur in the veins from disintegration of a thrombus. Acute infective inflammation of the cancellous tissue of bones is most to be feared, owing to the veins being abundant, thin-walled, and under pressure, on account of the bone being hard and unyielding. When an infective embolus becomes jammed in any of the smaller vessels, a thrombus is formed, in which the bacteria rapidly multiply, and pass through the vessel wall into the surrounding tissues, setting up localised inflammation, followed by suppuration and abscess formation. The lungs, being the first filter for emboli in the systemic circulation, are usually affected first, and infarction is brought about when the emboli have lodged in a terminal artery, the infarct being at first red, and then greyish-yellow when the formation of pus has taken place.

When the thrombi in the lungs start to disintegrate, and infective emboli are carried through the heart into the systemic circulation, abscesses may follow in any part of the body, the most usual regions being the kidneys, spleen, liver, and joints.

If the emboli are large in number and the symptoms severe, the condition is known as *acute pyœmia.* Sometimes along with the pyæmia there is development of the bacteria in the blood, constituting *septico-pyœmia.* If the emboli are few and the symptoms less severe, the disease is known as *chronic pyœmia.*

Conversely to malignant œdema, which is an early complication of wounds, pyæmia is a complication occurring at a later stage, though it may be no less formidable.

Purulent infection may show itself at any time during the course of deep irregular wounds which suppurate profusely, in the course of fistulæ following upon necrosis of the aponeuroses, tendons, or bones, and in the course of purulent inflammation of the synovial membranes, veins, or lymphatic trunks.

In the HORSE, phlebitis of the jugular vein, diseases of the poll, neck, and withers, quittor, purulent inflammations of the joints and tendon-sheaths, gangrene of the keratogenous membrane, and caries of the os pedis, are the affections most commonly associated with pyæmia. We may also include the irregular form of strangles as an affection in which pyæmia is not uncommonly met with.

In CATTLE pyæmia is most commonly seen as a complication of contused suppurating wounds and gangrenous or necrotic lesions, and the same applies to the smaller ruminants.

In the DOG the usual causes are compound fractures, contusions, and other serious traumatic injuries of the limbs.

In the females of the different species puerperal pyæmias are met with.

In young subjects, particularly foals and calves, purulent infection is frequently determined by a suppurative infection of the umbilical vein—"navel ill."

The etiology of pyæmia may be summed up as depending on the existence of a suppurating injury or focus. Various conditions inherent to this injury, such as the vital resistance of the animal and the means of injury, favour its development. The principal are—the depth of the wound, the irregularities and crevices with which its walls are riddled, sloughs in which pus becomes stagnant, the poor general condition and exhaustion of the injured subject, and the insanitary surroundings in which animals are so often kept.

The method of infection is often complex. In purulent phlebitis the intravenous clot, infected by pyogenic bacteria, detaches infective emboli, which are carried by the blood into the lungs and other organs. In the walls of fistulæ, caused and kept going by necrosis (*e.g.*, poll-evil, fistulous withers, and quittor), multiple phlebitis and lymphangitis are present, from which infection may arise.

In old suppurating wounds, unaccompanied by phlebitis, the layer of granulations may be broken, and the blood and lymph streams thereby exposed to infection.

SYMPTOMS.

The onset of acute pyæmia is generally marked by the appearance of rigors, which may be repeated, and along with this a sudden rise in temperature occurs. The animal is dull and depressed, without appetite, but thirsty, and the excretions are diminished. The pulse becomes soft and weak. Patchy sweating is commonly seen.

In connection with the wound there is from the commencement a change in the character of the suppuration. This is always considerably checked, and the pus which issues from the wound is of an unhealthy liquid nature, greyish or blood-stained, and full of cellular débris and microbes. The granulations are purple and friable; later they are sloughed off, and the product of their destruction forms a sanious and infectious liquid in the wound. There is no swelling of the borders of the wound, as in traumatic gangrene.

As the disease progresses, the general symptoms become more serious. There is rapid wasting and exhaustion of strength. The coat is staring, and the hair of the mane and tail is easily pulled out; the ears and the extremities are cold.

Secondary abscesses appear, and the symptoms which reveal their localisation—whether in the lungs, liver, kidneys, or brain, or purulent inflammation of the joints, synovial cavities, or pleura—are observed. There is often a greyish or sanguineous purulent discharge from the nostrils, with a fœtid odour. Auscultation and percussion serve to disclose the lesions in the lungs. The temperature undergoes considerable and rapid fluctuations. A profuse diarrhœa generally sets in, and leads to rapid weakening and exhaustion. In some cases there appear in various superficial parts phlegmonous swellings, which rapidly develop into abscesses. In strangles pyæmia there are often multiple abscesses of the lymphatic glands.

The course of pyæmia is generally slower than that of traumatic gangrene. According to the localisation and number of the secondary abscesses, the duration of the disease varies from a few days to several weeks.

Pyæmia is considered less grave than septicæmia, but the generalised forms with numerous centres in the viscera are invariably fatal. Even when the causal organisms have only an attenuated virulence, or have been destroyed after becoming fixed in the organs, they kill by the visceral lesions thus set up.

In Cows puerperal pyæmia is chiefly characterised by arthritis of the knee, hock, and stifle joints, tendo-synovitis of the flexors, perimetritis, and progressive wasting.

TREATMENT.

Prophylaxis consists in the application of the usual antiseptic measures. In the case of extensive, deep, and irregular wounds, it is important to prevent the accumulation of pus. Frequent cleansing with antiseptics and continuous irrigation are the best preventive means. It is often necessary to scarify, make counter-openings, and insert drainage-tubes, although as a general rule one should avoid injuring the granulation tissue; but if the case is urgent, it must be done. For this purpose the thermo-cautery judiciously used is better than the knife, as the eschar remains adherent to the subjacent tissues until they are covered with granulations, which are not so liable to infection as are raw surfaces. Irrigations with hydrogen peroxide, strong antiseptic solutions, and the carbolic and sublimate spray, are useful. For injuries to the limbs warm antiseptic baths continued for twenty minutes or half an hour, and followed by moist dressings, are best, though dry dressings covered with absorbent materials are preferred by some.

When indications of pyæmia set in, the wound must be rigorously and thoroughly cleansed, the cavities mopped out with hydrogen peroxide (10 vols. per cent.), or with a strong disinfectant solution (chloride of zinc, 5 to 7 per cent.; carbolic acid, 5 per cent.; or biniodide or sublimate, 1 in 500 to 1 in 1,000) and then powdered with iodoform, or placed under continuous cold-water irrigation. If any excessive or fungous granulations are present in the wound, they are best destroyed by the thermo-cautery. It must be recognised that one cannot render the wound aseptic, but it should be cleansed and disinfected as thoroughly as possible. If superficial collections of pus appear, they should be opened early.

Internal treatment consists in the administration of agents capable of sustaining the animal's strength, checking the multiplication of the bacteria, and neutralising the effects of their toxins. Amongst the drugs most generally used are alcohol, caffein, quinine, nux vomica, strychnine, carbolic acid and creolin. Most of these can be absorbed from the alimentary canal, and may be given by the mouth or by the rectum; others, such as caffein and strychnine, can be given hypodermically. It is better to introduce the necessary remedies *per rectum* than to forcibly administer them by the mouth, as the latter course greatly upsets the patient.

Treatment has been attempted by injecting antiseptics directly into the circulation—solutions of carbolic acid, sublimate, or iodine, into the jugular vein two or three times daily. It is difficult to believe that these agents could in this, or any other way, be introduced into the blood in a sufficient degree of concentration to destroy the bacteria without irreparably and disastrously damaging the system in general.

In those cases due to streptococci, antistreptococcic serum should be administered early, and repeated at proper intervals.

Nursing is all-important, and a liberal diet of light, tempting, and easily digested foods should be prescribed.

In acute pyæmia, as in septicæmia, when the infecting organism has firmly established itself, it usually carries the day, in spite of all one can do and whatever antiseptics one may choose.

As proof of the possibility of recovery from pyæmia, cases in horses are cited which, on post-mortem examination at a later date, revealed caseous purulent foci in the internal organs; but on closer examination these have turned out to be cases of tuberculosis or glanders, and not pyæmia. Cadiot and Almy have had several cases of pyæmia in horses under treatment, but have not observed a recovery once clinical indications of secondary abscesses in the internal organs have presented themselves.

In cases of chronic pyæmia, where the sequence of events is much slower, the preparation of a vaccine from the particular strain of organism with which the animal is infected may be tried. In undertaking this little-tried method, one should proceed cautiously, and not introduce the vaccine in excessive doses, nor too frequently, until some more practicable method of control than the observation of the opsonic index is available.

MALIGNANT ŒDEMA

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Synonyms.—Traumatic spreading gangrene; Hospital gangrene.

French: Septicémie gangreneuse; Œdème malin; Gangrène traumatique; Gangrène gaseuse.

German: Malignes Œdem Gangrän.

Definition.—Malignant œdema is a virulent inoculable disease occurring in man and several species of animals, and due to the invasion of the tissues by the malignant œdema bacillus (the *vibrion septique* of Pasteur).

ETIOLOGY.

Chiefly met with in the horse, it constitutes one of the most formidable complications of surgical or accidental wounds. Although relatively frequent formerly, it has now become an uncommon disease, owing to the general practice of aseptic or antiseptic methods.

Bacteriology.—The malignant œdema bacillus is a facultative parasite, whose common method of existence is as a saprophyte, having a ubiquitous distribution on the surface of the earth, but being especially abundant in cultivated and manured soils. It is therefore necessarily ingested by the herbivora with their food, and may be said to be a normal inhabitant of the alimentary canal of these animals, and is present in their fæces. It is doubtful whether it multiplies in the alimentary canal.

In cases of malignant œdema the bacillus is confined to the lesions, and is never found in any considerable numbers in the blood during life.

In veterinary practice the principal importance of the bacillus arises from the fact that in the herbivora it is a constant invader of the blood and tissues of the unopened carcass, and, on account of its resemblance to the anthrax bacillus, it is likely to lead to errors in diagnosis. The post-mortem invasion of the bacillus starts from the alimentary contents, and frequently begins immediately after the animal's death, being especially rapid if the blood is in a deoxygenated or asphyxiated state. The invasion mainly follows the line of the veins, and in the abdomen and thorax is soon fairly complete. The extent of the invasion of the peripheral parts is chiefly dependent upon the rate of cooling of the carcass, and is therefore most extensive in hot weather; in the height of summer or in the tropics there may be extensive invasion of the peripheral blood within twenty-four hours. If the temperature is 70° F. or above, the bacillus may sporulate freely in the blood, but at a lower temperature than this sporulation does not take place.

In size the malignant œdema bacillus is a comparatively large organism, being slightly less than 1 μ in thickness, and in length the single rods vary from 3 μ to 10 μ.

Both in the tissues and in the blood and fluid culture media it frequently grows out into long filaments, which may be uniform throughout, but generally are segmented at irregular intervals, some of the segments being twice as long as others.

The bacillus is motile, but this feature is of little diagnostic value, as the filaments seldom exhibit movement, and the single rods, on exposure to the air, soon cease to move.

At a temperature of 70° F., or above, sporulation takes place, the spore, elongated in shape, being either in the centre or towards one end of the bacillus, and not greatly exceeding it in thickness.

Staining.—The malignant œdema bacillus is readily stained with any of the basic aniline dyes. With methylene blue in semi-putrid anthrax blood, the malignant œdema bacillus, unless preparing to sporulate, stains quite uniformly and more intensely of a deep dark blue colour than the anthrax bacillus, and, in addition, the ends are rounded; whereas those of the anthrax bacillus are slightly concave when stained in this way. There is no metachromatic reaction with methylene blue, as is the case with the anthrax bacillus, where the bacillus stains blue and the capsule takes on a purple tint.

The malignant œdema bacillus does not stain by the method of Gram nor by the methods of Gram-Weigert or of Claudius.

Cultivation.—The organism has always been regarded as a strict anaërobe, but it can be grown exposed to the air by the Tarrozzi-Smith method—that is to say, in broth-tubes containing small pieces of sterile healthy liver, or even small pieces of sterile turnip or potato. By this method a moderate amount of gas is evolved—not so much as in the case of the black-quarter bacillus—and by the end of twenty-four hours' incubation the whole medium is rendered turbid, and a slightly putrid, though not sour, odour is noticeable. At this stage the culture is usually composed of single rods. As the medium gets exhausted sporulation begins, and by the end of two or three days the broth clears up, the growth being on the bottom or walls of the tube.

Although growth takes place best at 100° F., it will occur freely at ordinary temperatures. With a stab culture in glucose-gelatine the growth appears some distance below the surface as a whitish line with minute processes. Liquefaction soon occurs, and bubbles of gas are given off. In deep tubes of glucose-agar incubated at 100° F. growth is extremely rapid, taking the form of a white line along the needle track, with short lateral projections here and there, and attended with the formation of gas.

Under anaërobic conditions growth takes place on solidified blood-serum, which is liquefied, and upon potato, without coloration.

Inoculation is pathogenic for the following animals: Mouse, guinea-pig, rabbit, sheep, goat, horse, pig, dog, cat, fowl, and pigeon. The ox is refractory. In the mouse multiplication in the blood-stream occurs, and the spleen is much swollen. To produce a fatal result in a healthy animal a considerable amount of pure material is required, and must be introduced deeply into the tissues, intramuscular inoculation being the most certain. Injected along with other organisms (*Bacillus prodigiosus*, etc.), a smaller dose proves fatal. Inoculation by scarification is followed by no result, nor is it possible to infect by ingestion or inhalation.

ANIMALS ATTACKED.

This disease is chiefly met with in the horse as a complication of surgical or accidental wounds.

The ox, although proving resistant to experimental inoculation, is said by some authors to contract the disease naturally. The only cases meriting attention are those described in the cow following parturition.

Cases are but rarely met with in the other domestic animals, although they are susceptible to artificial inoculation.

MORBID ANATOMY.

Lesions in the Horse.

1. **Traumatic Gangrene.**—The tissues in the region of the swelling are reduced to a putrid fœtid mass. The skin is separated, necrotic, and sloughed off in irregular pieces. The muscles involved are emphysematous, friable, of a pale ochry colour, spotted with deep red or brown patches. The subcutaneous and intermuscular connective tissue is infiltrated with a reddish or pale yellow serous fluid, which extends considerably beyond the limits of the swelling.

The visceral changes are not extensive. The spleen is engorged and much softened. The myocardium is friable, pale, and ecchymosed about the coronary fissures; the blood of the right ventricle is uncoagulated and dark in colour. The pericardium contains a small quantity of a clear transudate. The blood is much altered in the veins of the affected part; it is uncoagulated, pitchy, and exhales a putrid odour. The walls of the vessels are red in colour.

2. **Septic Peritonitis.**—This is met with as a sequel to the operation of castration. The lesions are slight and quite out of proportion with the gravity of the symptoms. The abdominal cavity contains a small quantity of a reddish serous fluid; the peritoneal vessels are injected, and their serous coats have a uniform reddish colour, but there are no false membranes, and but slight traces of inflammation.

The inguinal region, stump of the spermatic cord, and scrotal wounds, often show their natural appearance; the connective tissue is slightly infiltrated. In other cases a clear gelatinous œdema surrounds the cord in its abdominal course; the infiltration may reach the peri-renal connective tissue, whilst, on the other hand, it may spread to the sheath and to the subcutaneous tissue of the abdominal wall.

The abdominal viscera are normal, with the exception of the spleen, which is softened and somewhat swollen. The pleuræ and pericardium contain a serous transudate; the myocardium appears parboiled and ecchymosed; the blood is dark and diffluent.

The organisms are met with in the blood and peritoneal exudate in the form of rods and filaments.

SYMPTOMS.

A. In the Horse.

In French literature two clinical forms of the disease are described as affecting the horse:

1. A form characterised by its traumatic origin, and the appearance from the first of crepitant swellings, and called *Traumatic Gangrene.*

2. A second form, analogous to the foregoing in its pathogenesis, but differing in its manifestation, consists in the invasion of the peritoneum following upon castration, and is called *Septic Peritonitis.*

Traumatic Gangrene.—The symptoms arise in connection with a wound, surgical or accidental, characterized at first by a small, hot, œdematous focus, which spreads in every direction with extreme rapidity. At the same time the appearance of the wound is changed; the tissues take on a livid, shining colour; suppuration is replaced by the running of a pale yellow or reddish serous discharge. The temperature rises to 103° or 104° F.; the pulse is rapid; respiration is accelerated; the mucous membranes are injected; the appetite is impaired. In twenty-four hours the œdema extends to a limited area of the body. If it does extend chiefly towards the dependent parts, it also spreads above the wound. The swelling, still hot and tender at the periphery, is softened, colder, and less painful towards the centre. A reddish-brown, frothy liquid with a putrid odour runs from the wound. The face is drawn. The animal, downcast and "at the end of its tether," from time to time shows periods of excitement. Certain parts are covered with sweat. The temperature remains at about 104° F.; the pulse is rapid, feeble, and thready; whilst the heart-beats gradually become violent, and to such a degree that they shake the thorax and can be heard at a distance. In some animals cerebral symptoms are seen, with violent excitement, muscular contractions, and a disposition to bite.

At a later period the œdema, spread to a tremendous extent, is softened, cold, and painless. On manipulation the tissues give a sensation of crepitation, indicating emphysema. The interstices of the wound contain a sanious and fœtid liquid; the skin, separated and withered, comes off in pieces; the muscles laid bare are friable, brown or purple, and their tissue spaces infiltrated with a frothy pale yellow serosity. The patient remains without feeling or action; the head is hung low or rested on neighbouring objects; the eyelids are half closed; the gait is laborious and unsteady; wasting is rapid; the flank is hollow; the hair of the mane or tail is easily pulled out. The temperature sinks below normal; the pulse is imperceptible, whilst the heart-beats preserve their force; respiration is slow, deep, and tremulous.

Sometimes indications of pulmonary gangrene appear. The animal falls and dies in a state of excitement.

In cases affecting the head, the emphysematous condition of the skin may not always be apparent.

The course is always rapid; the mean duration is from three to five days.

Septic Peritonitis.—The cases recorded were all consecutive to castration.

The symptoms of infection appear some days or several weeks (up to two or three months) after the operation. The animal suddenly appears listless and refuses to eat; colicky pains are noticed; he lies down with care, and rests extended on his side. In the course of some hours the symptoms are aggravated. Prostration and trembling of the whole body has set in; the face is drawn; the eyes are fixed and the pupils dilated; the temperature rises to about 104° F.; the pulse is small and rapid; respiration is slightly accelerated; the colicky pains continue. The animal remains motionless, stretched out on his side. The abdomen is distended with gas, and tender upon pressure.

Examination of the inguinal region often reveals nothing in particular; the operation wounds are covered with pus, but otherwise their appearance is normal. Only occasionally does an œdema appear in the region of the inguinal canal, and quickly spreads to the sheath, perineum, and abdominal wall.

Matters quickly go from bad to worse. Prostration is complete; various parts of the body are bedewed with sweat; the temperature falls to 100° F., or below; the patient remains outstretched; the abdomen is greatly distended; the mucous membranes have a yellowish colour; the pulse is imperceptible. Death takes place in the course of twelve to thirty-six hours on an average.

B. In the Ox.

The only cases worthy of notice are those *post-partum* ones recorded by German authors under the title of "Geburtsrauschbrand," and even regarding these an element of doubt occurs. The symptoms described are briefly as follows: The infection always takes place subsequent to parturition. It is preceded by symptoms of septic metritis. The first signs are loss of appetite, with rigors, diarrhœa, and slight elevation of temperature. Swellings develop about the perineum, buttocks, thighs, and sometimes from the commencement in the region of the shoulder, neck, or head. The swelling is at first hot and œdematous; it extends rapidly, and becomes cold, emphysematous, and crepitant. Death takes place in one to three days from the onset.

Post mortem one finds indications of metritis. The connective tissue of the pelvis and around the womb is œdematous and infiltrated with blood and gas. The subcutaneous connective tissue is emphysematous in several places, and at the same parts the muscles appear parboiled, friable, and spongy, of a dirty brown colour, and dissected by a sero-sanguinolent exudate and gas. The blood is normal. The viscera are but little altered.

C. In the Sheep.

The cases cited in sheep are those occurring in connection with the operation of castration, or with wounds inflicted during shearing or inoculation for sheep-pox.

The symptoms develop in connection with the wound, from which there quickly spreads an œdematous swelling, which is hot and painful, whilst from the wound there issues a clear serous discharge. The sheep loses its appetite and becomes dejected and sleepy.

If the swelling develops upon the body or upper parts of the limbs, death is the constant result; only in cases where the infection is towards the extremities is there any hope of recovery.

Cases of intestinal origin are quoted as the result of certain methods of feeding (fermented beetroots). The symptoms were those of peritonitis with extensive œdema.

D. In the Dog.

Infection follows bites inflicted by other dogs, or extensive wounds involving the subcutaneous or intermuscular connective tissue. There is a diffuse swelling, with gaseous infiltration of the tissues. Death follows in the course of a few days. Nocard met with an epidemic affecting young beagles fed on raw flesh, which ceased on substituting cooked foods.

DIAGNOSIS.

A. Clinical Diagnosis.

1. **The Horse.**—Traumatic gangrene is clearly indicated by the presence

of a swelling that is rapidly extending, œdematous, hot, and painful at the onset, then emphysematous, cold, and painless in the parts first affected.

Simple œdemas, inflammatory or passive, never become crepitant; their tendency always is to spread towards the dependent parts.

Certain swellings following castration, probably due to a streptococcic infection, appear in the groin and sheath, and extend towards the perineum; they are cold and somewhat tender, but never become emphysematous; they nearly always pass off, and the general symptoms are slight.

The septic nature of the peritonitis is to be suspected when the symptoms of peritonitis appear in subjects bearing castration wounds that have not yet cicatrised. Streptococcic peritonitis seen under the same conditions is only indicated by grave general symptoms; its termination is in other respects the same.

Malignant œdema is distinguished from erysipelas by the more grave general disturbance and the rapid development of the swelling. The latter in malignant œdema becomes emphysematous, and soon displays an outer œdematous zone, an intermediate gangrenous zone, and a central putrefying area.

Traumatic emphysema is differentiated from malignant œdema by the fact that in the former no grave constitutional symptoms are shown as a rule, and there is an absence of severe œdema and gangrene of the swelling with putrefactive changes.

2. **The Cow.**—The forms of uterine origin are indicated by crepitant external tumours. The diagnosis ought to be confirmed by finding the bacillus and by inoculation experiments.

3. **The Sheep.**—The presence of emphysematous swellings of traumatic origin, coinciding with a severe general condition, may be assigned to this infection.

B. Experimental Diagnosis.

For this purpose direct microscopical examination, cultivation and inoculation of the fluid obtained from the affected tissues, are employed. The bacillus of malignant œdema kills the guinea-pig, rabbit, and fowl, whilst the anthrax bacillus kills the guinea-pig and rabbit, and the black-quarter bacillus kills the guinea-pig only. The examination of the lesions in these inoculated animals is instructive. In the case of anthrax one finds in the guinea-pig and rabbit, in the connective tissue at the seat of inoculation, a clear gelatinous œdema; in the case of malignant œdema a red sanguinolent and frothy œdema. Black-quarter in the inoculated guinea-pig presents close analogies, and the finding of morphologically similar organisms has no weight.

IMMUNISATION.

Immunisation is effected—

(*a*) By the intravenous inoculation of virulent material. In the ass, by repeating the injection with large doses on two or three occasions, a high degree of immunity can be established, and a subsequent subcutaneous inoculation results only in the development of a local swelling and abscess. By means of a series of inoculations of increasing strength hyper-immunisation may by this method be obtained in the horse or ass for the purpose of producing an anti-serum.

(*b*) Immunisation by means of attenuated viruses. Satisfactory results have not been obtained in this way.

(*c*) Immunisation by means of toxins. This can be effected by the injection of cultures or virulent organic liquids, sterilised by heat or filtered through porcelain.

(*d*) **Serotherapy.**—An immunising serum is obtained by means of method (*a*), and has an antitoxic effect, as well as a direct action, on the bacteria (agglutination), and is considered as promoting phagocytosis.

TREATMENT.

An early degree of infection may be arrested by energetic surgical intervention, amputation of the infected region, cutting widely, being the best method, but is rarely applicable to animals. Total destruction of a centre of infection by excision or cauterisation is difficult to carry out, as it is always to be feared that the organism may have penetrated deeply at some point of lowered resistance. Deep scarifications of the swelling are indicated, and should be done with a bistoury, as the slough produced by the actual cautery protects the organisms from the action of the air or antiseptics. Drainage of the deep parts or periphery of the infected area is equally useful. The wounds should be irrigated copiously with a warm antiseptic solution, or with a solution of hydrogen peroxide, and then a dressing applied, containing some antiputrescent agent—*e.g.*, iodoform or tincture of iodine. Constitutional treatment includes the administration of diffusible stimulants and tonics. Digitalis and hypodermic injections of ether or caffeine are required in special cases.

Curative serotherapy is indicated in the horse. The results which have been obtained foreshadow the general efficacy of the method.

PROPHYLAXIS.

Prevention is assured by prompt antiseptic treatment of accidental wounds and the asepsis of surgical wounds.

Small deep wounds intersecting the connective tissue, and soiled by earth, dung, or dust, are chiefly exposed to septic complications. The indications are to cleanse the surfaces of the wound by prolonged irrigation with an antiseptic solution, and, if necessary, to open up or drain deep wounds. Any accumulated blood-clots are to be removed. An antiseptic dressing must be applied to the part, and be renewed often enough to prevent putrefactive changes from taking place.

The rules for surgical asepsis need hardly be impressed, for it is deep infection from hands or instruments that is to be viewed with apprehension, rather than superficial soiling from dust or simple contact with unsuitable objects.

Septic infection subsequent to parturition is to be avoided by antiseptic irrigation of the uterus and vagina, and appropriate treatment of any deep wounds of the parts. In the case of accidental wounds in parts rich in connective tissue, or castration wounds becoming soiled with infective material, preventive serotherapy should be adopted for valuable animals. In order to prevent infection occurring from the employment of hypodermic injections, it is necessary to use sterilised water to dissolve the hypodermic tablets, and to carefully sterilise the syringe and needle prior to use. Cases of malignant œdema have been traced to neglect of these precautions.

ANTHRAX

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Synonyms.—Splenic fever; Splenic apoplexy; Malignant pustule.

French: Charbon.
German: Milzbrand.
Human Pathology: Woolsorter's disease.

In India anthrax is known as "Loodianah disease" and "Manipur horse disease."

Definition.—Anthrax is a specific infective bacillary disease caused by the entrance into the body of the *Bacillus anthracis*.

ANIMALS ATTACKED.

Briefly it may be said that anthrax attacks all animals, including man. In man, however, if we except the possibility of cases arising from the ingestion of anthrax flesh, it does not occur as a natural infection. To man it is communicated directly or indirectly by animals, and takes the form either of a local infection (*malignant pustule*) or of a combined pleurisy and pneumonia (*woolsorter's disease*).

But it is the herbivora that are the most susceptible, and the commonest subjects are the ox and the sheep. Next in order of importance and susceptibility come the horse and the pig, the latter animal contracting the disease almost invariably by the ingestion of anthrax-infected flesh. Dogs, too, and foxes become infected in the same way. Cats, tame rabbits, hares, and wild rabbits are all subject to the disease, as also are guinea-pigs, mice, and rats.

Birds are not readily infected, while white rats, old dogs, and amphibians also show a marked resistance to anthrax. These remarks apply to inoculation tests, and inasmuch as it is certain that such methods would insure infection if susceptibility existed, it is safe to assume that naturally these animals are immune.

In this connection it is interesting to note that, despite the readiness with which other breeds become infected, Algerian sheep possess a natural immunity.

ETIOLOGY AND GENERAL REMARKS.

Among the diseases due to a specific organism anthrax is probably the best known of all. The reason is this: compared with other organisms of the class, that of anthrax is of considerable size. For instance, examination of a droplet of anthrax blood with only a moderate power of the microscope reveals the presence of the bacillus quite easily, and this in the unstained state. Add to this the fact that it multiplies with almost inconceivable rapidity in the blood of the more susceptible animals, and the further fact that with comparative ease it is cultivated outside of the animal body, and it is at once clear that facts concerning its life-history, morphology, etc., quickly followed on its initial discovery.

It was in 1849 and 1850 that Pollender, and afterwards Reyer and Davaine, first described the rod-like organism, which we now know to be the *Bacillus anthracis*, as occurring in the blood of animals dead of splenic fever. This was supplemented later by Pasteur's work on fermentation, so ably continued by Hansen, which works in their turn stimulated Davaine in 1863 to further consideration of the subject as affecting anthrax, and to a series of observations which ended (in 1873) in demonstrating, just short of actual proof, that the anthrax bacillus was the actual and existing cause of the disease. He, in fact, originated the name *Bacillus anthracis*.

Here Koch took up the running. He it was who observed in anthrax blood the appearances of division, and suggested that multiplication of the bacilli took place in the living body. Moreover, he was able to note this same division occurring outside the body, and to record the fact that spore formation here took place, crowning his work by isolating the bacillus in pure culture, and, by inoculating animals with the products, artificially producing the disease.

That the bacillus was the cause of the disease was thus proved. The mode of natural spread of anthrax, however, was still obscure. Koch himself, while surmising that infection by way of the intestinal tract was to be looked for, signally failed in his earlier experiments to reproduce the disease by feeding animals on the bacilli and their spores. This, however, followed later. Animals fed on the spores succumbed to the disease, demonstrating clearly enough the way in which anthrax might spread in a natural manner.

In the section on the bacteriology of the disease (p. 28) it will be seen that these spores of anthrax stand in much the same relationship to the bacilli as do the seeds to the higher plants. They are thus far more resistant to adverse circumstances (*e.g.*, excessive heat or cold, abnormal dryness or undue damp, chemical agents, antiseptics, etc.) than are the bacilli themselves.

This accounts for much that we now know regarding the manner of outbreak and the distribution of the disease. It explains, for instance, how it is that while anthrax may occur in a sporadic or even in an epizoötic manner, it occurs far more frequently as a marked enzoötic, outbreaks being limited to special districts, and showing no tendency to spread therefrom. It explains, too, how the occurrence of the disease will depend on varying conditions of the soil and climatic influences. It is a well-known fact that marshy, boggy, or swampy land particularly favours the conservation of the spores and the development of the bacilli. Especially does this apply when the land is flooded periodically, and good instances are to be found in the low-lying lands of the Kent and Essex marshes, and the black, loose moulds of the Lincolnshire and Cambridgeshire Fens. The spores in such soil, while the land is in flood and during winter's cold, lie inactive. With the advent of summer, however, with the draining away of the waters and the steady rise in temperature, activity commences and bacilli develop. It is in the months of June, July, August, and September, therefore, that outbreaks may be looked for and most frequently occur.

In passing it may be mentioned that under these circumstances the organic deposit remaining after subsidence of the waters is an extremely suitable medium for the growth and development of the organism. From this it follows that manuring the land with material of a like nature, and with the dung from stables and byres, will have an equally favourable effect in promoting the growth and sporulation of the bacillus.

While this occurs in a temperate climate during the months of summer, it must not be forgotten that spore-bearing material, in the shape of fodder grown on infected areas, is quite capable of giving rise to cases at other seasons. This is, in fact, the only way in which one can account for outbreaks in the months of winter, when the animals are being stall-fed, and very often on premises without a previous history of the disease. And this applies not only to home-grown produce, but to oats and to made-up feeding material in the form of cake from a foreign source.*

McFadyean has isolated anthrax bacilli from imported oats, in the important case of Denny *v.* Covington. Dunstan has demonstrated experimentally that feeding cake may contain the spores of anthrax.

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* According to Stockman, a summary of the quarterly incidence of anthrax from 1906 to 1910 shows "that there is a marked and constant drop in the number of outbreaks in the third quarter of each year—*i.e.*, at the time when most stock are on grass—while there is a decided rise when the animals may be assumed to be running in and receiving artificial food" (*Journal of Comparative Pathology and Therapeutics*, June, 1911).—Ed.

Reverting to the subject of manuring, a very real danger of infecting a pasture previously free from the disease lies in using a manure composed of animal matter such as blood or bone. One such case is recorded in the Board of Agriculture Report (Diseases of Animals) for 1908. This outbreak, occurred on a farm where anthrax was before unknown. The owner was able to state that no cattle or sheep had died during the ten years previous, and the veterinary inspector stated that no case of anthrax had recently occurred in the district. About a year before the outbreak bone-manure was applied to one field of grass which had been kept free from animals for the best part of a year. After a crop of hay had been removed from this field, nine cows were admitted for grazing purposes, and the grass formed their sole diet. Three out of the nine cows died of anthrax about a week after being admitted to the field.

What has been written here applies to anthrax as it occurs in a temperate clime and as we know it in Great Britain. But from what has been said it may readily be understood how in a hot climate, with the longer periods of temperature suitable for the development of the organism, anthrax may become a positive scourge.*

Below are the statistics of anthrax in Great Britain for the ten years 1899 to 1908:

Years.	Animals attacked.					Total.
	Cattle.	Sheep.	Pigs.	Horses.	Deer.	
1899	634	69	253	30	—	986
1900	668	40	204	44	—	956
1901	708	76	152	35	—	971
1902	746	50	192	44	—	1,042
1903	809	48	234	51	1	1,143
1904	1,115	62	365	47	—	1,589
1905	1,001	53	210	53	—	1,317
1906	999	83	231	35	—	1,348
1907	1,163	66	190	37	—	1,456
1908	1,107	34	220	58	—	1,419

When this is compared with the statistics for the ten years 1889 to 1898 it is found that there is a steady, if not a large, increase in the

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* Stockman points out that, according to available evidence, anthrax does not arise mainly, or even to a great extent, from previous cases of the disease on the same establishment. In great Britain statistics from 1902 to 1906 show that 83·5 per cent. of the outbreaks occurred in farms which had not been previously infected ("Epizoötology of Anthrax," *Journal of Comparative Pathology and Therapeutics*, June, 1911).

The same authority is of opinion that from this fact, and also from the information derived from a consideration of 1,257 outbreaks of anthrax which occurred on farms not previously infected, and where ground infection could not number of cases returned, the average number per year for the first decade being approximately 783, while for the second decade it is 1,222, or an increase of 439 per annum.

On this we need not speculate very largely. We would say, however, that it does not necessarily point to any great spread of the disease, nor to failure of the regulations which deal with it. Rather, we believe that with more modern methods of diagnosis such cases as have hitherto escaped detection are now recognised. It is probable, too, although the efforts of the Board of Agriculture have not yet confirmed the suggestion, that anthrax may be as prevalent among sheep as it is among cattle. If that be so, and the same attention is paid to the reporting of sudden deaths in these animals as is now paid to those among cattle, then we may look for a further increase in the total number of reported deaths from anthrax.

Regarding the geographical distribution of the disease, we may say briefly that it is world-wide, and that in nearly every country, under one of its many names, anthrax has been recognised.

BACTERIOLOGY.

1. The Bacillus.

(*a*) *In the Blood.*—Anthrax bacilli are found in the blood of the living animal only a very short time previous to death. A minute drop of fresh anthrax blood spread on a slide and examined microscopically reveals the bacilli in large numbers. They appear in this as small, transparent, non-motile rods, and may be observed with only a moderate magnification. In this manner, however, if we except the evidence furnished by a clinical history of the case, they may be mistaken for other bacilli, notably those of malignant œdema or of putrefaction.

In size the bacilli range from 1·5 to 2 microns long, and from 1 to 1·5 microns broad. These are the individual segments. It is characteristic of anthrax, however, that the multiplying bacilli after division show no tendency to separate, but to hang together end to end in chain-like form. Even in the blood, whose circulatory movements interfere with the formation of chains of any great length, this tendency is still maintained. It is thus that in the examination of anthrax

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have operated, that the evidence is strongly in favour of the view that the great majority of outbreaks in Great Britain are due to infection from without, particularly by material brought in from countries where anthrax is very prevalent.

He also points out the difficulty of obtaining definite evidence that foodstuffs carry infection, this being due in part to the circumstance that the infected portions may have been ingested by the animal prior to samples being taken, and also that it is only possible to examine small samples of the suspected material experimentally.

As regards the most likely explanation of infection in grain or meal, he is of opinion that during transit from infected countries these foodstuffs "become contaminated by infective material from dry hides, which on account of their light weight are often stored on the top of other cargo." He also points out that anthrax is "exceptionally prevalent in certain districts where imported hides are tanned and wool is washed, the assumed explanation being that the drinking-water becomes contaminated by the sewage from the factories. In the course of inquiry it has also been found that the disease is exceptionally prevalent on certain sewage farms which are known to receive tannery and knackery drainage."—Ed.

blood we are able to observe chain-like forms of from 5 to 20 microns in length. The fact that these 20-micron lengths are not individual organisms, but are composed of several bacilli end to end, is revealed by close examination. Along the length of the chain will be noted numerous fine, transparent, transverse markings. These indicate the points of division between the individual bacilli. While at times the line of demarcation between the adhering bacilli appears to be perfectly straight, at others we get a biconcave or biconvex marking, the two latter appearances depending on whether the ends of the bacilli are dimpled, or are rounded off after the manner of the proximal end of a phalanx. This variation in the shape of the ends of the bacilli appears to have some connection with the different methods of staining.

The body of the bacillus is composed of a finely granular protoplasm, while around this, and revealed by appropriate staining, is a well-marked capsule. When several of the bacilli are end to end, this capsule sometimes appears to be common to the whole chain.

Such are the appearances of the bacilli in the blood. From a sample of such blood it is easy to isolate the bacillus by making gelatine or agar plates, and so observe their characteristics in culture.

(*b*) *In Plate Culture.*—After twelve hours' incubation of the inoculated plates at 37° C., growth may be looked for, the separate colonies appearing as minute rounded points, around which the gelatine is liquefied. The plate should be placed on the table of the microscope and the colonies examined under a low power. They may then be readily recognised. Each has the appearance of a light grey, shining mass, consisting of numerous wavy bundles, the latter being likened by several writers to curling locks of hair.

From one of these colonies an impression preparation should be made. This is done by simply dropping a cover-glass on to the colony, and afterwards staining and mounting the adhering growth. Under examination with a high power this preparation reveals a characteristic of anthrax, which it always exhibits when grown under artificial conditions—viz., its tendency to form unbroken chains. The wavy locks of hair are seen to be composed of innumerable interlacing filaments of a wonderful delicacy of form and arrangement, the filaments themselves consisting of anthrax bacilli placed end to end without a break. This appearance is very nearly, if not quite, diagnostic of an anthrax culture, no other pathogenic organism growing in a similar manner.*

(*c*) *In Bouillon.*—In this medium irregularly spiral threads are seen suspended in the liquid after twenty-four hours' incubation at 37° C. Later, as growth becomes more abundant, a flocculent mass is deposited at the bottom of the flask or tube. Here, again, the growth is composed of interlacing chains of the bacilli. The bouillon is not rendered turbid.

(*d*) *In Gelatine* (*Stab Culture*).—Cultivated in this way, anthrax grows as a white granular line along the needle-track. If the proportion of gelatine in the medium is low—that is, if the medium is not too stiff—a characteristic appearance is presented. In about forty-eight hours numerous delicate feathery growths radiate out from the inoculating stab. From these other lateral rays are given off, and the whole has a feathery appearance, somewhat resembling thistle-down. The radiating rays are always longer near the surface of the medium. On the immediate surface of the gelatine, however, little or no growth takes place. In this position liquefaction of the medium occurs, gradually spreading down the stab and into the medium. In this way the whole of the gelatine may become liquefied, and the anthrax growth finally deposited at the bottom of the tube.

A slope culture on gelatine shows a thick felted growth, on the edges of which the hair-like appearances, as described in plate cultures, may be recognised. Along the line of inoculation the gelatine is liquefied.

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* By some workers it is claimed that this characteristic growth of anthrax is obtained in sufficiently large degree after only five hours' incubation, thus rendering the making and examination of gelatine plates a useful adjunct to other methods in cases where a ready diagnosis is required concerning a specimen of blood showing a mixed infection.

(*e*) *In Agar.*—In this medium, both in stab and slope preparations, much the same appearances are presented as in similar preparations in gelatine. There is, however, no liquefaction of the medium.

(*f*) *On Potato.*—On this medium a dull white felted mass occurs along the line of inoculation, but shows no tendency to spread therefrom. It shows no special characters.

In or on all these media the manner of growth is that specially characteristic of the organism—namely, that of long interlacing leptothrix filaments.

(*g*) *Biology of the Bacillus.*—Under this heading probably the first point of interest to the veterinarian is the fact that the *Bacillus anthracis* is aërobic. For its complete development free exposure to the oxygen of the air is indispensable. In this we find explanation of the fact that, while the anthrax bacillus rapidly multiplies in the blood-stream, depending for its existence on the oxygen there present, yet it never there forms spores. For this further phase in its development exposure to the air is necessary. It further explains the fact that an anthrax carcass rapidly putrefies. The blood, robbed of its oxygen by the rapidly multiplying bacilli, is quickly invaded by anaërobic organisms from the intestine, while under this invasion, and with all available supply of oxygen exhausted, the anthrax bacilli degenerate. This point has a twofold practical bearing. We understand from it how it is that in the blood of an anthrax carcass allowed to lie long after death no single bacillus can be demonstrated where previously they existed in untold numbers. We understand, too, the need for disposing of anthrax carcasses intact, recognising the fact that spilling the blood, and consequently exposing it to the air, is assisting the spread of a highly virulent material.

The question of temperature, too, is important. Anthrax bacilli thrive best at 35° C. Below 12° C. no growth takes place.* Neither does growth occur above 45° C., while prolonged exposure to a temperature of 60° C. kills them outright.

And in other respects the spore-free bacilli exhibit comparatively low powers of resistance. Light, and more especially sunlight, impedes their growth. The action of the gastric juice is absolutely inimical to them, and they are accordingly destroyed in the stomachs of healthy animals. Even if kept at ordinary temperatures for a few days, they are usually found to be dead. They may, however, be subjected to cold at the freezing-point without dying.

That they are rapidly killed off under the process of putrefaction we have already intimated. Also, we have pointed out that in the absence of oxygen, degeneration takes place. It remains to be said, however, that, while certain of its vital functions are best carried on in the presence of this gas, the *Bacillus anthracis can* grow in its absence.

(*h*) *Staining Reactions of the Bacillus.*—The *Bacillus anthracis* stains easily with solutions of the basic aniline dyes. The method, however, which is of great interest to the veterinarian is that introduced to English practitioners by Sir John McFadyean. In this method the stain used is a 1 per cent. aqueous solution of methylene blue, rendered slightly alkaline by the addition of ½ per cent. of sodium bicarbonate. With this the film of suspected blood is stained for half a minute to a minute, and then afterwards simply washed in water. The picture presented by an anthrax film stained in this manner is such that in animals recently dead of the disease—in cattle, at any rate—a means of diagnosis is provided that is nearly constantly reliable. The appearance of the film is, in fact, remarkably characteristic. Not only have we the bacilli stained a deep blue, but we have also other elements in the film stained a pronounced violet or reddish-purple.

Close inspection of such a film reveals the fact that the violet-stained portions have some close connection with the envelope or capsule of the bacillus; for, while such violet-stained portions are found detached and plainly apart from the bacillus itself, we note also that the coat or envelope of the bacillus is traceable as a thin violet line immediately round the body.

This peculiar double-staining reaction with alkaline methylene-blue solution appears to be a characteristic of anthrax alone, and is of the utmost service as an aid to diagnosis.

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* On this knowledge is based the Board of Agriculture's regulations dealing with deep burial of the carcass. The temperature of the ground, save at the surface, is always below 12° C.

An additional point of interest in the staining reactions of the bacillus is that it takes the stain of Gram, or, in other words, is Gram positive. Briefly stated, Gram's method is to stain with aniline gentian violet, and afterwards to decolorise with a solution of iodine, finally rendering the decolorisation complete by treating with absolute alcohol or methylated spirit.

2. The Spores.

As we have said before, just as the seed is to the higher plant, so is the spore to the bacillus. Now, the spore of anthrax may first be seen in a bacillus about to sporulate as a small, highly refractile speck in the centre of the protoplasmic body. This gradually increases in size until it forms an oval body about the thickness of the bacillus. The bacillus itself gradually loses its staining properties, and finally disappears, allowing the spore (an oval, highly refractile body) to lie free.

Most observers are agreed that sporulation only occurs outside the animal body. This, therefore, as well as the marked resistance the spores show to adverse circumstances, furnishes us with another and a weighty reason for the necessity of disposing of anthrax carcasses intact.

As a result of the high resistance they show to ordinary outside circumstances, the spores of anthrax, when they are set free from an infected animal and have got into the soil, may remain there for a considerable time. By some observers this time has been put at twelve months, while others say it may be many years—ten or even more. It is sufficiently long, at any rate, to account for the regularly-occurring cases on infected pastures, and to explain how the disease may be spread by means of feeding materials, such as cake, fodder, or roots, and by soil and running water.

In this connection it is important to remember that the gastric juice, which is able to kill off most of the bacilli taken into the stomach, is unable to injuriously affect the spores. These are able, therefore, to pass along to the intestines, and so give rise to that form of anthrax with which the English veterinary surgeon is, perhaps, most familiar.

From what has already been written, it will be understood that for vegetating, the spores find a favourable medium in the animal body. Under certain circumstances, however, they may do so outside of it. Where temperature and other conditions are favourable they may spring into activity, resume the bacillary form, and again sporulate in the soil and in surface-water. This explains how, in tropical climates, the contagium may exist in all virulence for many years without the occurrence of fresh cases to maintain the running.

For the formation of spores free oxygen and a suitable temperature are required. The temperature most favouring sporulation is 32° to 35° C. It may, however, take place at a temperature as low as 14° C. or as high as 43° C. Thus it follows that abundant sporulation occurs at a temperature varying from 20° to 25° C., or, in other words, at the ordinary temperature of summer heat, or at that of a dwelling-room—a point of high practical importance. Above 43° C. sporulation ceases. Further, if the bacilli are kept at this temperature for eight days, they fail afterwards to sporulate when grown again at a lower temperature. The spores are killed by boiling for five minutes; but the application of dry heat at 140° C. must be maintained for several hours to destroy them with certainty.

Experience has shown that the bacilli themselves are killed by cold only in rare instances, resisting a temperature of 10° C. for three days, from which it follows that the spores, with their higher powers of resistance, retain their vitality for an indefinite time under the same conditions.

As bearing upon the important rôle played by the spores in the spread of anthrax, we would refer to the case of Denny *v.* Covington. Here the deaths of certain horses were plainly traced to feeding on a quantity of oats to which anthrax spores were adherent. More than that we need not say here. The whole of the evidence in the case, however, more particularly that of Sir John McFadyean in describing his experiments, makes most interesting and instructive reading.*

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* *Vide* the *Veterinarian*, vol. lxix., p. 62.

The Action of Germicides on the Bacilli and their Spores.

Those agents in common use for this purpose are rapidly effectual in destroying the bacilli. Thus they are killed by perchloride of mercury (1 in 30,000), by creolin (1 in 15,000), and by carbolic acid (1 in 100).

The same agents, with the exception of carbolic acid, are equally effective in disposing of the spores. A 1 in 1,000 solution of perchloride of mercury kills them in ten minutes, and a 3 per cent. solution of creolin in forty-eight hours. It is important, however, to note that the spores remain virulent after immersion for twenty days in a 5 per cent. solution of creosote, and for nineteen days in a 10 per cent. solution of carbolic acid. On the other hand, they rapidly succumb to 2 per cent. watery solutions of chlorine, iodine, or bromine.

Light, and more especially sunlight, has also a modified germicidal effect on both the bacilli and their spores, impeding, and even entirely arresting their development.

MORBID ANATOMY.

The morbid changes in anthrax are those brought about by (1) the engorgement of the capillaries by the bacilli; (2) the deprivation of the tissues of oxygen; (3) the action of the toxins elaborated by the bacilli. Accordingly, we find on post-mortem, hæmorrhages in practically all the organs of the body; a tar-like, non-coagulating condition of the blood; serous, gelatinous, and hæmorrhagic infiltrations of the subserous, submucous, and subcutaneous tissues; considerable swelling of the spleen, and a diffluent condition of its pulp, together with inflammatory changes in the substance of the lymphatic glands generally, and in the liver and the kidneys.

On removing the skin the first thing that strikes one is the amount of yellow, gelatinous exudate. This in some positions is very considerable, more particularly along the neck and down the trachea. The vessels of the skin itself are tensely filled with blood, while on the inner surface of the skin and in the subcutaneous tissues generally, appear well-marked blood extravasations up to the size of a shilling. These same extravasations are found also in the substance of the gelatinous exudate wherever that appears.

Such muscular tissue as is now exposed shows considerable softening, is flabby with contained inflammatory exudate, is considerably darker than normal, and is spotted with blood extravasations. It may be taken, too, that the whole of the muscular tissue of the body, including that of the heart, is in a like condition.

On opening the abdominal and the thoracic cavities there is found, in place of the normal straw-coloured lymph, a blood-stained, inflammatory exudate—this in considerable quantity; while the gelatinous exudate already noticed beneath the skin is found to be also infiltrating the subserous and submucous connective tissues. This is particularly noticeable beneath the serous coat of the mesentery, in the neighbourhood of the kidneys, and in the mediastinal cavities, notably the anterior. The glands of the mesentery and the mediastinal glands are swollen, contain blood extravasations, and are infiltrated with the gelatinous exudate.

The spleen calls for special mention. Almost constantly it is greatly enlarged (up to four or five times the normal size), and may reach the weight of 30 or 40 pounds. Its capsule is tense and blood-stained, and often raised in tumour-like swellings; while its pulp, abnormally soft, is dark red, almost chocolate in colour. In cases of considerable enlargement softening of the pulp occurs until it is actually diffluent. In fact, a spleen in this condition, if incised and suspended, will drain itself of its pulp almost entirely.

With regard to the other internal organs, it may be put briefly that they all show signs of intense congestion and actual blood extravasations, with degenerative changes as a consequence. Thus we get degenerative changes in the parenchyma of the liver and the kidneys. Hæmorrhages and patches of necrosis occur along the course of the alimentary canal, the hæmorrhages being sufficiently extensive to render the intestinal contents bloody. The lungs are engorged with blood, and the whole of the mucous membrane of the air-passages reddened and hæmorrhagic, while the trachea and bronchi contain a frothy, blood-stained discharge.

All the other organs of the body—the sexual organs, the urinary organs, the salivary glands, bones, articulations, etc.—in fact, all structures supplied by the infected blood—participate in the changes mentioned, hæmorrhages and blood-stained gelatinous exudations being the main naked-eye characteristics.

Everywhere the blood is abnormally dark in colour, exhibits a peculiar iridescence described as tarry or varnish-like, and shows little or no tendency to coagulate. Microscopic examination shows numberless anthrax bacilli in the plasma, considerable alterations in the size and shape of the red cells, and a marked degree of leucocytosis.

In this country it should not often happen that the veterinary surgeon should be able to observe the foregoing post-mortem appearances, for the prophylactic measures insisted on by the Board of Agriculture demand that the carcasses of all animals dead of the disease be buried or burned intact.

Before proceeding to an actual post-mortem, however, an anthrax carcass often offers a few points for consideration that may, perhaps, be best mentioned under this heading of morbid anatomy. In cattle, for instance, who usually die from the so-called apoplectic form of the disease, the cadaver is markedly tympanitic, while from the natural orifices there is considerable exudation of a blood-stained, frothy discharge. Usually, too, the last few evacuations of fæces (probably passed involuntarily and nearly always found near the carcass) are distinctly hæmorrhagic.

Much the same may be said in the case of sheep. Here, again, the carcass becomes rapidly tympanitic, and from the natural orifices issues the same frothy, hæmorrhagic discharge.

In horses and in pigs, where the acute, apoplectic, or intestinal form of anthrax is probably not the common one, there may usually be found diffuse swellings (the "anthrax carbuncles" of some writers) about the throat and down the neck, which swellings when incised exude the blood-stained, gelatinous exudate so characteristic of the disease.

Finally, it may be mentioned, with reservations which will be given under the heading of diagnosis, that all the lesions, the blood, and the exudates contain the anthrax bacilli.

SYMPTOMS.

In Cattle.—It is not often the veterinary surgeon is afforded an opportunity of noting the symptoms of anthrax in cattle. Usually the first intimation he has that he may be dealing with such a case is that an animal is reported to him to have been found dead, either at pasture or in the house, without previous noticeable illness. Or it may be that the veterinary surgeon will receive with the report of the death a history of slight "hoven" or staggers—nothing more than that.

Even in cases sufficiently protracted to allow the symptoms to be observed, anthrax in cattle runs an extremely rapid course. Infection has taken place by ingestion, and in a short time symptoms attributable to the invasion of the blood-stream by the bacilli make their appearance. In other words, we have all the evidence of acute fever, coupled with the effects of the deoxygenated blood and of the toxins of the bacillus on the higher nerve centres. The temperature runs up to 106° or 107° F., and the pulse, much increased in its number of beats (80 to 100 or 120 per minute), is thin and hardly perceptible. Rumination is suspended, the horns and other extremities are cold, and occasional rigors manifest themselves in various parts of the body. The animal separates itself from the rest of the herd, and is found to be standing in a dazed, stupefied condition, labouring hard for breath, and reeling and staggering in its gait if asked to move. The visible mucous membranes are intensely injected. Often there are symptoms of colic, and nearly always there is marked irritability of the intestines, manifested by the evacuation of more or less fluid and blood-stained fæces.

The condition of coma rapidly increases, until finally the animal gets down and dies in convulsions.

Death may occur thus suddenly, even as the veterinary surgeon is watching the case; or in a few instances, though few indeed, the animal may linger on for a period of twelve hours or so.

The above is the usual run of the symptoms. It must not be forgotten, however, that cases may be met with in which there is nothing more to note than the abnormal temperature, other signs of disturbance, such as the diarrhœa and marked dyspnœa, being wanting. These cases, as we shall afterwards mention (see Diagnosis, p. 38), are extremely liable to be mistaken for an ordinary acute attack of pneumonia, and may linger on for so long as forty-eight hours.

It should here be noted that usually the first case in an outbreak runs the course first described. If, however, the temperatures of the other cattle of the herd be taken at this time, it will be found that perhaps one or two, or even more, of them are showing a rise. Such cases—for cases of anthrax no doubt they are—usually pass off with nothing further to note.

By some writers the appearance of anthrax "carbuncles" is referred to as occurring in cattle. Using the term "carbuncle" as applied to the local inoculation form of anthrax as seen in man, then the existence of such a lesion in cattle must, to say the least of it, be most rare—in this country, at any rate. Occasionally very diffuse, painless swellings may be noted, commencing in the neck, and extending from there to beneath the chest, together with like swellings in other parts of the body. These, however, are very far from approaching the size reached by similar swellings, to be afterwards described, in the case of the horse, and very often only succeed in making their presence known at post-mortem. Patches of degenerated tissue implicating the deeper structures of the skin of course occur. That any great evidence of this, in the shape of discoloration and swelling, is given during life, however, is doubtful. It is better, therefore, to reserve the term "carbuncle" for such forms of anthrax as are purely "inoculation" forms.

Before leaving this section it will be well to refer here to those cases of submaxillary enlargements suddenly occurring in cattle in this country, implicating the tongue, sometimes extending down the neck to the dewlap and chest, and variously known as "blain," "gloss-anthrax," or "hawks." It is permissible to doubt that these cases are anthrax at all. Certainly the existence of the anthrax bacilli in the swellings has never been demonstrated. Moreover, it is a matter of ordinary clinical knowledge that such cases very frequently recover.*

In the Horse.—Equine anthrax may manifest itself in one of two ways: It may occur with a large amount of swelling round the throat, which swelling gradually increases in size, and extends down the neck to the chest; or it may occur without this lesion, and simply run the course of an acute enteritis, accompanied by the exhibition of severe abdominal pain.

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* See note on Gloss-Anthrax at end of chapter, p. 46.

In the first instance the symptoms offered for observation are such as to be almost diagnostic, the head and neck swelling, with its tendency to gradually enlarge, and with the exudation therefrom, when pricked, of a thin serous discharge, being the main characteristic.

With this, as in cattle, there is also all the evidence of an acute febrile condition. The temperature runs up to as high as 105°, 106°, or even 107° F. The pulse is increased in number up to as many as 120 per minute, and is weak and thready. The mucous membranes are deeply injected, and this is particularly noticeable in the conjunctiva and in the cavities of the widely distended nostrils. The surface temperature is irregular. The coat is hard and staring, and muscular tremors affect various parts of the body in turn. Later, patchy perspirations bedew the body; the breathing becomes laboured and largely abdominal; and very evident interference with the higher nerve centres, as manifested by stupor and a reeling gait, makes its appearance.

The fæcal evacuations, at first normal in consistence, become sloppy, have a peculiar greasy appearance—suggesting, as near as description will allow, an admixture of linseed oil—assume a dark, nearly black, colour, and sometimes, though not always, become watery and blood-stained.

The swelling about the head and neck increases in volume and in tenseness, setting the head and neck almost immovable on the trunk, and giving the animal the appearance more of a hippopotamus than a horse. Respiration grows more and more laboured; a thin hæmorrhagic discharge issues from the nostrils; the patient is unable to swallow, while strings of tenacious saliva hang from the mouth; perspiration becomes more profuse, and the pulse grows weaker and weaker, until at last it is imperceptible. Finally the animal goes down, and expires in convulsions.

With a train of symptoms such as these, death may occur in from twelve to thirty-six hours. On the other hand, the case may, and often does, linger on for four or five days.

The second set of symptoms exhibited in equine anthrax are, as we have just said, simply those of an acute enteritis. In fact, it may be stated pretty definitely that infection has commenced in this instance in the intestine, and that the case will offer nothing more for observation than the ordinary symptoms of rapid inflammation of the bowels.

An attack of shivering ushers in the symptoms. The pulse becomes enormously quickened in number—hard and wiry at first, later weak and thready. Abdominal pain is manifested by the animal continually going down and rising, and these pains, all the while continuous, grow gradually in intensity. The intestines commence to empty themselves in an irritable manner, small quantities of fæces being passed at frequent intervals. The tail is outstretched and quivering, muscular tremors affect various parts of the body, and intense restlessness, almost approaching mania, is manifested as the case runs its course.

Later, profuse perspirations bathe the body, the respirations grow quicker and quicker, and the pulse, save at the radial, becomes imperceptible.

Later still the excitement and the mania give way to a dull stupor. The animal reels in his walk, the eyes grow amaurotic, and the sweats grow cold. Finally the animal drops and dies in convulsions, in from five, six, or seven hours after the commencement of the symptoms.

In Sheep.—As in cattle, the course of anthrax in sheep is rapid. In most instances, beyond what appears to the shepherd an apoplectic seizure, suddenly followed by convulsions and death, nothing is seen. At times, however, a case may be slightly more prolonged, although even then it is rapid. In such instances, then, much the same symptoms as are shown by cattle will be noticed here—namely, a great rise in temperature, tremendously increased respirations, rapid and quickly failing pulse, irritability of the bowels and blood-stained fæces, together with periods of excitement, succeeded by stupor, convulsions, and death.

In Pigs.—Anthrax in the pig is most rare without a history of the animal being previously fed either on slaughter-house offal or on the flesh of an animal dead after only a brief illness. Add to this the fact that there is in nearly every case a most marked and characteristic swelling of the throat and neck, and little more need be said. We give here, however, the symptoms attending the deaths of twenty-eight pigs at Witham:*

"Swelling of the throat, dulness, inclined to lie about, but movements fairly brisk when disturbed; appetite and fæces normal at outset, and later remained so. Conjunctivæ deeply injected; no discharge of froth or blood from nostrils, eyes, or anus. No cough, but a choking, stertorous noise occasionally heard when an attempt was made to swallow food. Some difficulty in breathing as death approached. Temperature, 105° to 106° F., declining before death. The swelling, beginning in the intermaxillary region, rapidly extended over the jaws, face, and parotids, and down the lower aspect of the neck. It attained a large size, was tense, painful on manipulation, did not pit, closed the eye (usually only one), and gave the skin of the snout a glistening, shiny appearance."

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* *Vide Journal of Comparative Pathology and Therapeutics*, vol. xix., p. 247.

In this outbreak the pigs died in from eight to twelve or sixteen hours after the appearance of the swelling, the younger pigs, as might be expected, dying most quickly. Several attempted to feed an hour or so before death, which occurred very quietly and quickly.

DIAGNOSIS.

Post-Mortem.—Seeing that the disease in most animals is nearly always rapidly fatal, the diagnosis of anthrax will depend on demonstrating the existence of the bacilli in a stained film prepared from the suspected blood or exudate from the dead animal. In fact, no case in which anthrax is in any way suspected should be allowed to pass unless this examination is made.

Fortunately, in the case of cattle and sheep, the animals in which we most frequently meet with anthrax, this method is productive of reliable results. The most suitable stain for this purpose is the alkaline methylene blue already referred to on p. 27. The peculiar double-staining reaction with alkaline methylene-blue solution there described appears to be a characteristic of anthrax alone, and a film exhibiting it in marked degree may be diagnosed as anthrax with an amount of certainty almost approaching the absolute—that is, we repeat, in the case of cattle and sheep.

Practically the only objection of any weight to be urged against this method is to be found in cases where the carcass has been allowed to lie for a long time after death before an examination is requested. In the deoxygenated blood the anaërobic bacteria from the intestine commence to multiply, while the anthrax bacilli under this invasion rapidly degenerate and die off. Proportionate, therefore, to the degree of putrefaction, anthrax bacilli have become fewer in the blood, while others, from which they have now to be distinguished, have taken their place. This condition, if far advanced, may of course render diagnosis with the microscope impossible, for a stage is finally reached where the blood is occupied by none but putrefactive bacteria.

If, however, the putrefactive process is not too far advanced, if anthrax bacilli, though only few in number, still exist, then a diagnosis may yet be made, even in these cases of mixed infection. *Putrefactive bacteria*, or the *bacilli of malignant œdema*, may be fairly readily recognised. They are not nearly so numerous in the film, as the bacilli in a case of anthrax examined shortly after death. They are further recognised by their relatively greater length and thickness as compared with those of anthrax, by their evident inclination to form chains, and, above all, by the intensity with which they stain. Moreover, the well-marked envelope, so noticeable in anthrax, is absent. It follows, therefore, that if with bacilli showing these appearances, other bacilli are discovered comparatively shorter, with a violet-stained contour, and with an appreciable amount of violet-stained débris external to the bacilli themselves, but immediately adjacent to them, we are dealing with a case of mixed infection ; that the occasional bacilli showing the double-staining reaction, and perhaps assisting in the diagnosis by showing a more or less regular segmentation, are those of anthrax, and that invasion of the blood-stream by the putrefactive bacteria has been subsequent to this.

As the putrefactive process above referred to commences in the intestines, it follows that the peripheral vessels are the last to be invaded. This is a point of practical importance, for it sufficiently indicates the wisdom of always selecting blood from the extremities for the purposes of examination.

A further point of importance in making a diagnosis by microscopic examination of the blood is to remember that in cases of sudden death from causes other than anthrax (notably lightning-stroke, sudden heart failure, or rapidly fatal pulmonary congestion) the bacilli of malignant œdema may be found in relatively large numbers in the blood very shortly after death. In fact, they follow decease so early that one may possibly be led to assume they were in the circulating blood during life, and that therefore they must be those of anthrax or other specific disease. Again, however, a reference to their characters as just described should render error unlikely. Moreover, in any film quite innocent of anthrax, even where putrefactive bacilli or those of malignant œdema are present in large numbers, it will be noticed that the film considered as a whole—that is, before being placed under the microscope at all—has not stained with the uniform density that marks a film of anthrax. In such a case the merest flooding of the stained film with the water necessary to decolorise it, is successful in immediately removing the stain to the extent of leaving a barely discernible film for examination. In anthrax, on the other hand, comparatively lengthy washing leaves a film that is deeply stained.

Should any great doubt exist, and a post-mortem be deemed inadvisable, then this method of blood-film examination should be succeeded either by plating out gelatine inoculations of the suspected material, or by inoculation of a susceptible animal. As these are purely laboratory methods, we do not propose entering into them here. In such a case the practitioner will be well advised in forwarding samples of the suspected material to an expert he can trust.

Although in cattle and in sheep this method of blood-film examination is nearly constantly reliable, in the case of the horse and the pig it is not so. While in some instances the blood may swarm with typical anthrax bacilli, in others the bacilli are quite undemonstrable, notwithstanding the fact that the clinical symptoms unmistakably point to anthrax. Here examination of the blood should be followed by examination of films prepared from the exudate in the throat swellings, where these exist and this may need to be further followed by examination of section prepared from the throat glands or the spleen before demonstration of the bacilli is possible.*

In some cases where the existence of the disease has not even been suspected,-as, for instance, where the ordinary clinical symptoms have been aberrant or altogether wanting, an actual post-mortem is sometimes proceeded with. In the case of the ox this is not carried very far before the true nature of the case is realised. The changes described in the section relating to morbid anatomy afford unmistakable, almost indisputable, evidence, more especially the condition of the spleen. The same remark applies to sheep.

In the case of the horse dead of the disease in its acute form, and without the characteristic throat and neck swellings, there is just the ordinary evidence of an acute septicæmia. The post-mortem of one of the horses as related in the case of Denny *v.* Covington is an instance of this :

" When the skin was stripped off there was a jelly-like substance beneath. On opening the abdomen there was a discharge of a large quantity of yellowish-red fluid—some 2 or 3 gallons. When the bowels were removed, it was noticed that the glands along the upper surface of the bowel were very much enlarged, and of a dark reddish colour, some of them being as large as an orange. Between the folds of the mesentery there was a gelatinous material similar to that under the skin. The large intestine showed discoloured patches, some about the size of a sixpenny bit, and others as large as half a crown. The small intestine showed a few discoloured, reddish-purple patches, but the discoloration was not so extensive as in the larger bowel. On opening the bowel there was a certain amount of reddish-coloured fluid mixed with the contents. It appeared like broken-down blood. The liver seemed normal in size, but in cutting across it the blood which flowed out was very dark in colour. The spleen was about the ordinary size, but its surface was mottled with spots, the colour of a mulberry. The kidneys contained a large quantity of muco-purulent material."*

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* *Journal of Comparative Pathology and Therapeutics*, vol. xix., p. 248.

Unsuspected before the post-mortem (the horse was supposed to have died from twisted bowel), microscopic examination of the blood showed the cause of death to be anthrax.

In the pig, too, if we except the characteristic throat swelling, the classical lesions are wanting. It so happens, however, that the throat swelling is so constant as to be pathognomonic, and on incision shows the changes usually occurring in an anthrax enlargement. Lesions elsewhere are very insignificant. As a matter of fact, the disease appears to localise itself in the throat and stomach. On removing the tongue, larynx, etc., large ulcers covered with necrotic tissue are found in front of the epiglottis, bilateral, but seldom the same on each side, averaging a penny-piece in size, and approaching the circular in shape. This process of necrosis appears to commence in the tonsils, and to spread therefrom, giving a greyish, diphtheritic appearance to the affected parts. The gastric mucous membrane is deeply congested, and the intestinal mucous membrane is congested also, but less in degree. The mesenteric glands are more or less swollen and congested. The spleen, however, and all other organs appear healthy, while there is quite a remarkable absence of the general effusion and blood extravasation so noticeable a feature in other animals.†

During Life.—If we except the subacute form as it occurs in the horse and the pig, the diagnosis of anthrax during life offers considerable difficulty. Here the protracted course of the disease and the characteristic swellings enable one to arrive at a more or less correct decision. The latter, in fact, may be said to be almost certainly pathognomonic. This notwithstanding, an exact diagnosis cannot always be arrived at, for microscopic examination of exudate or blood from the swellings nearly always gives a negative result.

When the disease runs an acute course, as in cattle and in sheep, or as in the intestinal form in the horse, diagnosis may be said to be impossible during life. The rapid nature of the cases, and the closeness with which they may simulate other conditions—*e.g.*, acute pulmonary congestion in cattle, apoplexy in sheep, and acute enteritis or intestinal twist in horses—absolutely prevent any definite opinion being given. At the same time, when, on any particular farm or pasture, a case has once occurred, and has been diagnosed by unequivocal methods, and when other suspicious cases follow in its train, then the veterinary surgeon is able accurately to base an opinion on the clinical symptoms. This particularly applies to suddenly occurring and rapidly fatal cases of apparent pulmonary congestion in cattle and sheep on known anthrax-infested pastures, and to equally sudden and fatal attacks of apparent twist or apparent pulmonary congestion in horses in like infected areas.

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* *Veterinarian*, vol. lxix., p. 65.

† *Vide Journal of Comparative Pathology and Therapeutics*, vol. xix., p. 247.

DIFFERENTIAL DIAGNOSIS.

In Cattle.—Anthrax in cattle may be confused with cases of poisoning by *narcotic* or *acrid-narcotic principles of vegetable origin*—*e.g.*, celandine, yew, cake manufactured from the husk of the castor-oil seed, colchicum, ergot, hemlock, and many others. In such instances it is not usual that a diagnosis is made of the first case. The first death is, perhaps, shown by the microscopic examination of the blood and the subsequent post-mortem *not* to be anthrax. In possession of this negative evidence, the veterinary surgeon is able to proceed, and, should further cases occur, is led by their history—by the connection between the symptoms and the food taken; by the simultaneous sickness of many animals, without demonstrable contagion; and by the complication of gastric and nervous symptoms—to look farther afield.*

Death from *lightning-stroke*, *quarter-ill*, *cerebral apoplexy*, and *acute pulmonary congestion*, may also simulate anthrax. In the main, diagnosis in these cases also rests on proving the absence in the blood of the *Bacillus anthracis*, together with such evidence of a clinical nature as each case affords. Particularly should *quarter-ill* be differentiated by the presence of the black-quarter tumour. The characteristic crackle and feather-like feel of this lesion is almost unmistakable, and are further dealt with in the chapter relating to that disease. Moreover, smears taken from the incised quarter-ill muscle may be easily distinguished bacteriologically from anthrax. In death from *lightning-stroke*, too, there is usually evidence of superficial singeing of the coat, generally commencing at some high position—*e.g.*, the tips of the ears, base of horns, or top of lumbar region—and traceable down one or more of the limbs to the horn of the feet. Additional evidence in these cases is also often found in the fact that the animal is nearly always found with a tuft of grass held tightly between the incisor teeth—just proof that death was instantaneous and the animal in a fair state of health when taken. The malignant œdema bacilli rapidly appearing in the blood in these cases must be differentiated from those of anthrax by the characteristics already given (p. 35).

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* *Vide* "Poisoning of Cattle by Common Celandine," *Journal of Comparative Pathology and Therapeutics*, vol. xvi., p. 367; and "Toxic Gastro-Enteritis," "Veterinary Pathology" (Friedberger and Fröhner), vol. ii., p. 179 (1905 edition).

In Horses.—We have already pointed out that anthrax in the horse may be mistaken for *twist of the intestines* and for *acute congestion of the lungs.* It may also be confused with other septicæmic conditions, more particularly *purpura hæmorrhagica*, especially if in the latter instance the swellings of this disease make their appearance in the region of the throat and neck. Here, again, diagnosis must entirely depend on a search for the bacilli of anthrax in the blood. In the case of purpura, however, a little help in diagnosis is furnished by the character and position of the swellings during life. Unlike those of anthrax, they tend to affect various parts of the body, more especially those parts that are dependent, not confining themselves to the throat and chest, but appearing in several positions rapidly and simultaneously. Moreover, the swellings in purpura show a much more decided tendency to become gangrenous, particularly where there is slight pressure from such articles as the noseband of the halter or from the surcingle holding the rug. A further point of differentiation, too, is the fact that in purpura there is very often the discharge of a clear serous exudate from the skin in the bends of the hocks and in the hollows of the heels, with, later, gangrene and exfoliation of the surface layers of the skin in those positions.

In differentiating equine anthrax from death from lightning-stroke the same search for marks of singeing must be made as in the case of cattle; while in this, and in death from equally sudden cause while the animal is in possession of full health, the same care must be taken as in cattle to distinguish between the bacilli of malignant œdema and those of anthrax.

A disease which very closely simulates anthrax clinically is *South African horse sickness*—so much so that it was at one time actually thought to be anthrax. Differentiation, however, is easy. If fresh, the blood in South African horse sickness shows no bacteria of any kind, and yields nothing on culture. Moreover, points of differentiation are found as follows: That South African horse sickness appears at the end of the South African summer, and is extremely rare in winter; that the disease is peculiar to equines; and that the spleen in horse sickness is unchanged.

In Sheep.—The conditions with which anthrax may be confused in the sheep are malignant œdema, braxy, and louping-ill. Again, diagnosis will depend on a microscopic examination of the blood for the bacilli, followed, if necessary, by cultivation or inoculation methods in the laboratory.

In Pigs.—In these animals the appearance of the throat swelling is so characteristic as to be diagnostic, for, so far as is yet known, there is no other disease of pigs in which this swelling occurs. If, after death, verification of the diagnosis by demonstrating the bacilli is deemed necessary, then it must be remembered that the spleen and the blood contain but few of them, and that it may be further necessary to proceed to examination of sections from the glands about the throat. According to Stockman,[*] "a fairly common lesion in pigs, and one of great diagnostic value, is the presence of necrotic areas on the mucous membrane of the pharynx. These vary in size, but are commonly as large as a shilling. The centre is dark, almost black in colour, slightly raised, and the edges are level with the mucous membrane. There is seldom any difficulty in finding anthrax bacilli in preparations made from these areas."

PROGNOSIS.

While in the course of an outbreak cases of spontaneous recovery may, and no doubt do, occur, the prognosis in anthrax must always be grave. The average mortality ranges from 70 per cent. to as high as 100 per cent., and once symptoms of any gravity set in, then death invariably results.

PROPHYLAXIS.

If we except protective inoculation, which is dealt with in the following section, then the most important prophylactic measure is the thorough destruction of the anthrax-infected carcass. This may be done either by deep burial or by cremation. In the former case the Board of Agriculture's regulations direct that the carcass be buried intact at a depth of not less than 6 feet below the surface of the earth, and with a layer of lime not less than 1 foot deep both beneath and above it. They further direct that such burial should take place in a spot to which animals have not access, removed from any dwelling-house, and at such a distance from any well or watercourse as will preclude any risk of contamination of the water therein.

If cremation is decided on—and it is undoubtedly the most effectual method—then it is conveniently carried out by burning the carcass over a cross-trench.[†]

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[*] *Journal of Comparative Pathology and Therapeutics*, June, 1911.

[†] *Cremation of Anthrax-Infected Carcasses.*—As the veterinary surgeon may be called upon to advise regarding this, we quote here the description of the method given by General Fred Smith in his "Veterinary Hygiene":

"I use a trench dug in the form of a cross about ¾ foot deep in the centre, and gradually becoming shallower as it reaches the surface of the soil. It is 7 feet long each way, by ¾ foot wide, and the earth taken out of the trench is thrown in the right angles formed by it; on this earth the wood is placed. If two pieces of stout iron can be obtained to place the wood on, so much the better. A layer of big

In either case, care should be taken to collect the last excreta passed by the animal, the spilled discharges from the natural orifices, and any infected portion of fæces or stable litter, in order to bury or to burn with the carcass.

These methods should each of them be followed by a rigid disinfection of the surroundings, and for this purpose either a 1 in 1,000 solution of perchloride of mercury or a 4 per cent. solution of creolin is suitable.

Should the cases arise in the centre of some large town, and neither of these methods be immediately available, then the carcass must be moved to some knacker's yard or other suitable place, and there either burned as above described, or subjected to compressed steam sufficiently long to break up all the softer structures. The bones should afterwards be destroyed. Before moving the carcass, the natural orifices must be plugged with tow or cotton-wool steeped in some reliable germicide, and a strict disinfection of the surroundings and conveyances used, should be afterwards carried out.

Anthrax in Dogs, Cats, and Farmyard Poultry.—Although ordinarily the carnivora and birds are not easily infected, the possibility of anthrax occurring in these animals must not be overlooked by the veterinarian. Dogs have been known to contract anthrax by ingestion of anthrax-infected flesh, as also have fowls. It is important, therefore, to remember this when disposing of the larger animals dead of the disease.

PROTECTIVE INOCULATION.

In this country, for reasons which we shall afterwards discuss, protective inoculation against anthrax has not been largely attempted. Elsewhere, however, in nearly every country in Europe, notably Germany and France, and in America (Argentina and Uruguay), these inoculations have been carried out on a large scale. The practitioner should therefore become acquainted with the methods employed and the technique of the operation.

Practically, there are two methods which have come into prominence. The one consists in subcutaneous injections of attenuated anthrax

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wood comes first; then the trunk, which has had all the limbs and viscera removed; next more wood, and on this are placed the limbs, covered by wood; and, lastly, the viscera, also well covered with wood. The pile is then set fire to, and the body of a horse can in this way be readily consumed in five or six hours. . . . If the body be placed whole on the fire, it will take about twelve hours to consume."

To this we would add that in districts where wood is scarce coal may be added to the fire with advantage. The method of cremation is expensive and tedious (careful turning being required during consumption of the carcass), but, as we have mentioned in the text, is certainly the most effectual.

cultures; the other in the simultaneous injection of an attenuated culture and an anti-anthrax serum.

We will take first *the method of injecting attenuated cultures.* It is now well known that the virulence of an anthrax culture may be reduced by one of several means. Touissant, for instance, first showed that the virulence of anthrax blood was lessened by the application of heat. Attenuation of the virus was also obtained by cultivating the bacillus in media containing weak antiseptics (Chamberland and Roux); again, by the application of heat (Pasteur and Chauveau); by growing in compressed oxygen (Chauveau); by cultivating the organism in the blood or serum of animals that are immune—*e.g.*, white rats, frogs.

There are still other methods, and the most of them consist in cultivating the organism in conditions slightly unfavourable to its growth.

In obtaining the necessary attenuation, whichever method is adopted has for its primary object the prevention of spore formation. The method which has been found the most suitable, and has therefore become general, is that of heating. This is associated with the name of Pasteur.

By cultivating anthrax bacilli in the presence of oxygen at a temperature of from 42° to 43° C., Pasteur found that they ceased to form spores, although still multiplying by fission. He further found that varying degrees of attenuation were to be obtained proportionate to the length of incubation. The longer the cultures were subjected to the temperature above stated, the greater the degree of attenuation.

By then inoculating laboratory animals with the attenuated cultures, two vaccines were arrived at suitable for the protective inoculation of the larger animals.

The First Vaccine (the weaker) consists of a bouillon culture of non-sporulating anthrax bacilli attenuated by cultivation for twenty-four days at a temperature of 42·5° C. This, when injected, must be fatal to mice, but not to guinea-pigs.

The Second Vaccine (the stronger) is a similar cultivation incubated at the same temperature, but for only twelve to fourteen days. When injected, this should be fatal to both mice and guinea-pigs, but not to rabbits.

The animal to be protected is then inoculated with the *first* or weaker vaccine. Ten to fourteen days later it is again inoculated, this time with the *second* or stronger vaccine.

Immunity is said to be established in ten days to a month after the second injection, and to last for one year.

Instructions are generally issued with each lot of vaccine sent out, and the operator is directed to make the injections subcutaneously behind the shoulder in the ox, and in horses and mules in the region of the neck. He is further instructed to make the first injection on one side of the animal, and the second injection on the other.

The dose of each vaccine for horses, cattle, and mules is 1 to 4 c.c., and for sheep and goats 1 to 8 c.c.

The injection should be made with a small Pravaz syringe supplied for the purpose.

The second method—viz., that of injecting simultaneously an attenuated culture and an anti-anthrax serum—has been largely employed by Sobernheim. To obtain the necessary serum, animals already immunised by the Pasteur vaccines are taken, and further injected with large quantities of a virulent culture. Their blood then yields a serum which shows marked prophylactic and curative properties against anthrax when injected into rabbits. In other words, there has been obtained an anti-anthrax serum with marked bacteriolytic properties.

The serum thus obtained is injected in a dose of 10 c.c. into one side of the animal to be protected, while into the other side is injected at the same time an attenuated culture of about the same strength as Pasteur's second vaccine.

Both of these methods are successful in conferring immunity. Against that of Pasteur it has been urged by Koch that, while it protects against inoculated anthrax, it gives no immunity from natural infection by way of the intestinal tract. If this be so, then no stronger objection can be raised against its use in veterinary practice. Further, it may with truth be objected against this method that losses occur from the vaccination itself, and that these losses are sometimes heavy. It is in this way: While the losses, calculated on a large number of inoculations, are only 1 to 2 per cent., yet these same losses from inoculations on a particular farm may be as high as 10 per cent. Therefore, so far as Great Britain is concerned at any rate, the losses from inoculation itself may exceed the average natural loss from the disease. In other words, the Pasteurian inoculation is not to be advised except in those instances where the disease in a district is stationary, and where there are regular and heavy losses from it.*

The method of Sobernheim appears to be free from this element of danger. Moreover, if we take Sobernheim's own conclusions, which conclusions appear to be well founded, it possesses other advantages. It is quickly effective, and has not to be repeated. It protects from infection by way of the intestinal tract, and a more lasting immunity is conferred.

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* According to Stockman, observations on several millions of cattle in various parts of the world, submitted to the Pasteur method of preventive inoculation showed that accidents occurred in about 0·5 per cent. of the inoculated, taken all round.—Ed.

Sobernheim's published results, particularly of the inoculations in Argentina and Uruguay, certainly appear to offer proof of the efficiency of this simultaneous method of inoculation against anthrax. Although carried out on nearly 300,000 animals, it was not in itself productive of any loss, and, as shown by statistics, served to bring about the almost complete disappearance of cases of anthrax.

In districts, therefore, where anthrax is constantly present, and where the losses from the disease exceed 2 per cent., the simultaneous method of inoculation may be advised.

For the very reason, however, that in Great Britain anthrax is not the scourge it is in a hotter climate, it is doubtful whether any method of protective inoculation, with its expense and attendant risks, is warranted. The ordinary prophylactic measures may serve the necessary purposes.

TREATMENT.

Having regard to the rapidly fatal course of anthrax in the majority of the domesticated animals, it may be said at once that any ordinary treatment of the disease may be looked upon as useless. By "ordinary treatment" we here mean the administration of medicines. In passing, however, it is interesting to note that improvement and quick recovery has been reported after the administration of 50 to 200 grammes of creolin (Meier), and that such agents as perchloride of mercury (subcutaneous injection), carbolic acid and salicylic acid in 10 to 15 gramme doses, Lugol's solution in 2-ounce doses, chlorine, arsenic, phosphorus, hydrochloric acid, and ammonia, have all been recommended.

Recovery under treatment such as this, however, cannot fairly be attributed to the therapeutic measures alone. Far more likely is it to be one of the spontaneous recoveries that careful temperature records have shown to occur during the course of an outbreak.

The use of a reliable anti-anthrax serum, however, would appear to be indicated. Sobernheim has shown that animals inoculated with virulent anthrax cultures have successfully withstood this inoculation if subsequently injected with large doses of an anti-anthrax serum; that, therefore, the serum may be regarded as curative. With this in view, such cases as run a protracted course, and thereby offer opportunity to the veterinarian—*e.g.*, cases of the disease in the horse—may yet be proved to be amenable to a bacteriolytic serum.

Stockman states that if accidents threaten to occur from preventive inoculation, they can be avoided to a large extent by administering a dose of anti-anthrax serum. He also advises the use of the serum in cases where an animal has died of anthrax amongst other animals, and its blood has been spilt. The injection is then carried out on the remaining animals; also in cases where any in-contact animals show an abnormally high temperature the serum is indicated. The immunity from serum is said to last for about ten days. It can only be of use in cases where the blood-stream is not invaded by the bacilli.

NOTE ON ANTHRAX IN WILD ANIMALS IN A STATE OF CAPTIVITY.

In the *Veterinary Record* of February 25, 1911, an outbreak of anthrax amongst wild animals in a menagerie is reported by Mr. F. Somers, M.R.C.V.S.

The following animals died from the disease—viz., two pumas, one lioness, one leopard, two racoons, one coypu, one English badger, one ant-eater, and two Polar bears. A vulture also succumbed to the disease.

The following showed marked symptoms of anthrax, and recovered—viz., two lions, two leopards, one Polar bear, and one or two smaller animals.—EDITOR.

NOTE ON GLOSS-ANTHRAX.

In many respects the term "gloss-anthrax" is an unfortunate one, and save for the fact that it has crept into somewhat general use no mention would have been made of it in connection with the foregoing chapter. Simply it signifies anthrax affecting the tongue; and, inasmuch as we know that the tongue sometimes becomes the seat of anthrax lesions—notably in outbreaks among the carnivora and in pigs—that at first sight would appear justification enough for the retention of the term in our nomenclature.

Unfortunately, however, there is no reason to doubt that gloss-anthrax is far more often used in recording not cases of anthrax, but those conditions which only simulate it. For instance, the disease affecting cattle in this country, and known as "hawks," "blain," or "gloss-anthrax," very often appears much more like urticaria than anything else, in support of which statement we would point to the quick rise of the swelling, its equally rapid decline, the almost constant failure of the lesion to spread far from the seat of inoculation, and to the very frequent rapid recovery of the patient. If such a swelling were due to a local infection with anthrax, particularly as it occurs in an animal so susceptible to that disease as the ox, then one would most certainly expect it to spread with extreme rapidity, and the condition itself to show a high death-rate. In short, that the disease is more or less benign is opposed to the fact of its being anthrax at all.

Again, from continental writings we are able to judge that the term "gloss-anthrax" is used in reference to a condition in which bullæ appear on the tongue and buccal mucous membranes, which bullæ break and discharge a stinking fluid. While it is probable, especially in the case of the carnivora, that such a condition may indicate an attack of anthrax, it is far from being equally probable in the case of the herbivora. Malignant œdema, hæmorrhagic septicæmia, and ordinary cases of ulcerative stomatitis may give rise to somewhat similar lesions, and a diagnosis of anthrax in such a case is very wide of the mark.

With advantage to everyone it seems that the term "gloss-anthrax" might be conveniently dropped. In dealing with our case it would be better to say simply that we have "anthrax," making that statement dependent on demonstrating the anthrax bacilli in the blood or discharges; and then, if lesions occur in the tongue, simply to say so. Much confusion would thereby be saved.—H. C. R.

BLACK-QUARTER

Synonyms.—Black-leg; Quarter-ill; Symptomatic anthrax; Emphysematous anthrax; Struck; Strike; Carbuncular erysipelas; Merylen; Felon, etc.

French: Charbon symptomatique; Charbon bactérien.
German: Rauschbrand.

Definition.—It is an acute specific enzoötic and infectious disease of the ox, and occasionally of sheep, characterised by the appearance of a rapidly increasing emphysematous swelling, containing black extravasated blood and gases, in the region of the quarter, thigh, shoulder, neck, trunk, or elsewhere.

History.—The disease has been known since the dawn of veterinary science, and in the early days was thought to be a form of anhrtax. Charbert, in 1790, appears to have been the first to clinically distinguish the disease from anthrax. This opinion was strongly supported and amplified in later years by Wallraff and Pfisterer. In 1876 Bollinger, and subsequently Feser, revealed the presence of motile rods in the crepitant tumours, which they considered specific. They succeeded in transmitting the disease to cattle and sheep by inoculating them with virulent material from the lesions. In 1879 Arloing, Cornevin, and Thomas investigated the disease, carrying out experiments to distinguish it from anthrax, and describing the principal characters of its bacillus; they also devised a method of vaccination, which they published in 1884. More recently the important work of Kitasato, E. Roux, Duenschmann, and Kitt, has thrown a powerful light on the biological properties of the bacillus, and on the method of producing artificial immunity.

GEOGRAPHICAL DISTRIBUTION.

The disease occurs in every country, but it has a very uneven or regional distribution. In large areas it is quite unknown. In some districts it may be found on certain farms, and not on others; while on some farms it is well known that particular fields are notoriously infected.

It is especially prevalent in old permanent pastures and moorland, and tends to disappear with drainage and cultivation. It is reported to be the all-prevailing cattle disease in Porto Rico, a country where anthrax is unknown. It is scheduled as a contagious disease in Austria, Belgium, France, India, Italy, Norway, Sweden, and Switzerland. The statistics from these countries show that the disease has no distinct seasonal occurrence, the outbreaks being distributed in a fairly even manner all the year round.

ANIMALS SUSCEPTIBLE.

The vast majority of naturally occurring cases are seen in young cattle, ranging from three months to two years old, although cases are occasionally met with in cattle up to four years old. Cases of the disease in a three-day-old calf and a six-year-old cow have been recorded by Strebel and Foster respectively. The individual susceptibility of cattle seems to vary very much. Cases are occasionally met with in the sheep. It is, however, quite possible that a proportion of naturally occurring cases in this animal are overlooked or confounded with other diseases, owing to the absence of veterinary inquiry. As tested by experimental inoculation, the sheep is much more susceptible than the ox. The guinea-pig is susceptible by inoculation; very young guinea-pigs are, however, less susceptible than old ones. Cases have also been observed in the reindeer, camel, and young buffalo. The horse, dog, pig, cat, rabbit, and man, are regarded as virtually immune, although cases of the disease are recorded in the horse by Ganter, Liebold, Leonhardt, and others, and in the pig by Marek and Battistini (Friedberger and Fröhner).

PORTALS OF INFECTION.

Experimentally, endermic inoculation generally fails, but the subcutaneous and intramuscular methods of infection generally yield positive results, especially in the case of the subcutaneous method, if the part selected is rich in loose connective tissue. Experimental infection by the mouth is rarely, if ever, successful, and it is uncertain whether naturally occurring cases are infected by this channel or not. On the other hand, there is seldom, if ever, a cutaneous wound found associated with the lesion in natural cases. Notwithstanding the almost insuperable difficulty of infecting by the mouth, it is still possible that infection does naturally take place by that portal. The explanation is probably that naturally occurring cases follow upon some defective alteration of the defences of the body, or a quickening influence on the bacillus, which enables the latter to enter the tissue from the alimentary canal.

IMMUNITY.

One attack of the disease is said to produce immunity, but as black-quarter is one of the most fatal diseases of the bovine species, our experience in this matter is necessarily very limited. Old cattle hardly ever contract the disease, and seem to possess a natural immunity, which increases with age. Cases are not common in sucking calves, but they are not immune. Algerian cattle are said to possess a relatively high degree of resistance, and the Agro Romano cattle, although very susceptible to accidental infection, are reported to be refractory to experimental inoculation.

ETIOLOGY.

The essential cause of the disease is the invasion of the tissues of the body by the *Black-quarter bacillus*, which is also known as the *Bacillus anthracis emphysematosa*, *Bacillus Chauveii*, *Rauschbrand bacillus*, and the *Bacillus sarcophysematos bovis*.

BACTERIOLOGY.

The bacillus is a facultative parasite, living mainly a saprophytic existence in the soil, the vast number of outbreaks being of sporadic origin, due directly to a soil bacterium. The bacillus is motile and a strict anaërobe. It is chiefly a tissue parasite, confined to the lesions. In no animal is the disease of a septicæmic character, although in some cases in the ox it is possible to demonstrate bacilli in the blood at the time of death. In the sheep, however, they are as a rule present at the time of death, and can be easily detected. The bacilli are also found in any effusions which may exist in the serous cavities.

Sporulation does not take place in the living body, although the bacilli continue to multiply in the body for a considerable time after death, if the temperature is suitable, and the blood from a carcass may present a septicæmic picture some hours after death. The bacilli found in the blood and in very acute lesions at the time of death appear as slender rods, with rounded ends, measuring 3 to 6 microns in length, and about 0·5 to 1 micron in breadth. They stain uniformly throughout, and represent the actively multiplying form of the bacillus. In natural lesions, examined some time after death, bacilli of various shapes are found, and as a rule comparatively few of them are cylindrical or stain uniformly. The majority are spindle or cigar shaped, and stain irregularly; others, again, present the appearance of a tennis-racket, owing to the presence at one pole of the bacillus of a large oval spore. Another characteristic appearance is where two bacilli are joined together, and, sporulating at opposite poles, resemble the outline of a pair of spectacles. The bacilli do not form filaments, although two or three may be often found joined together.

They **stain** well with methylene blue and the other basic aniline dyes, and also by the method of Claudius; by Gram's method only when the process is greatly prolonged.

In **culture** they behave as the strictest of anaërobes, giving off a gas composed chiefly of hydrogen, CO_2, and nitrogen, and producing a sour smell resembling that of rancid butter, due to the formation of butyric acid. They grow at body temperature and in ordinary media, but Martin's broth appears to suit them best.

The bacillus is possessed of great vitality. It may live indefinitely in deep stiff clay soils, owing to the absence of oxygen and light. It is not injured by putrefaction, and intense cold does not affect it. Exposure to a dry heat of 100° C. for twenty minutes cannot be trusted to destroy it, but boiling for that length of time proves fatal to it. It is destroyed by such agents as mercuric chloride ($\frac{1}{10}$ per cent.), salicylic acid (2 per cent.), carbolic acid (2 per cent.), copper sulphate and boric acid (20 per cent.); while sulphuric acid, quicklime, chloride of zinc, alcoholic solution of carbolic acid, have given unsatisfactory results. The activity of the virus is increased by the addition of such agents as lactic acid, acetic acid, dilute alcohol. These, however, are supposed to act by preventing phagocytosis. The same effect can be produced by bruising the seat of inoculation before or after the operation.

PERIOD OF INCUBATION.

The period of incubation varies from one to five days, the average period being about two days, and, in exceptional cases, it may be reduced to a few hours.

MORBID ANATOMY.

There is usually an external emphysematous tumour seen in connection with some of the large muscular areas. In most cases it is located in a limb, but it may be on the trunk or the neck. Decomposition sets in rapidly, so that the whole carcass rapidly becomes emphysematous, and a blood-coloured frothy discharge appears at the natural orifices.

The skin over the tumour is sometimes dry and leathery, indicating a condition of dry gangrene; when cut into, the affected muscular tissue is found soft and friable, and of a dark brownish or almost black colour, and surrounded by a blood-tinged exudate, which shows little tendency to coagulate. The centre of the affected muscular mass is abnormally dry and somewhat emphysematous, showing hæmorrhages; outside this central area there is usually a hæmorrhagic zone saturated with blood-tinged exudate. The nearest lymphatic glands are enlarged and congested. Often there is a quantity of blood-tinged fluid in the peritoneal cavity, and occasionally also in the thoracic and pericardial cavities.

The thoracic and abdominal viscera usually show no lesions, although hæmorrhagic infiltration may be found in the heart, lungs, liver, kidneys, stomach, and bowels; the spleen is seldom affected, and never enlarged.

The muscular lesion gives off a rancid odour at first, which becomes highly offensive as putrefaction sets in. The extent to which the lesion becomes emphysematous is in proportion to the slowness of the disease, so that acute cases are œdematous, and not emphysematous.

In sheep the lesion is often obscured by the fleece, but it sometimes happens in this animal that prior to death the wool is shed over the affected part, and a blood-tinged exudate exudes from the denuded skin.

The blood shows no pathological changes, and forms a clot of normal firmness.

SYMPTOMS.

The disease is swiftly established and of brief duration, natural cases occupying a period ranging from less than twenty-four hours to three days. Not infrequently it happens that the first intimation of an outbreak, even in herds under daily supervision, is revealed in the carcass of a victim.

On the other hand, many cases present well-marked and characteristic symptoms; the animal is observed to be dull, refuses food, stands apart from its comrades, with slightly elevated back and head carried low, presenting an anxious and dejected appearance. Muscular tremors and spasmodic movements of the head are noticed, if carefully looked for. The pulse and respiration are accelerated. The temperature rises to 104° or 105° F., and in acute cases runs up to 107° F.

It should be noted, however, that from the observations of Schütt Warringsholz, and Steinbuck, it is possible that cases may occur without any increase of temperature.*

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* With reference to the question of the temperature in black-quarter, Baldrey (*Indian Veterinary Journal*, 1911) recorded his observations on twenty-one cases of the disease at Muktesar, and concluded "that in black-quarter there is usually at

If the animal is made to move, lameness or stiffness is evident; and on examination a swelling is noticed on the shoulder or hind-quarter, or some other part of the body. The swelling is quite commonly found on the neck, chest, back, and loins, where it may not cause lameness, but a general stiffness. Occasionally it may appear in the palate and back of the tongue. It is never found below the knee or hock, owing to the denseness of the subcutaneous tissue in these regions.

The swelling is at first somewhat œdematous, hot, and painful, and pits on pressure; but, increasing rapidly, it becomes emphysematous, crepitating on pressure, and resonant on percussion. The skin covering the part becomes cold and leathery, indicating necrosis and the advent of gangrene. When incised, no pain is evinced, and a dark frothy fluid escapes, giving off a sour smell. As the disease progresses the symptoms become aggravated, constipation is present, the pulse becomes weak and imperceptible; the temperature may fall or become subnormal before death. The victim refuses to move or rise, and dies in an almost comatose condition.

Brain symptoms are seldom or never noticed. Death usually results within twenty-four hours; but if the disease is generalised, it may take place in a few hours, while prolonged cases may linger for two or three days.

PROGNOSIS.

The disease is highly fatal, fully 95 per cent. of the victims succumb. Recoveries in vaccinated calves have recently been recorded by Hewetson and Roberts. Galtier states recoveries are frequent in Algeria, and rare in France. Tisserand puts the recoveries in the latter country at about 2 per cent.

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some period in the course of the disease a high temperature, but to the ordinary clinician the absence of this would not be remarkable." He pointed out that if the temperature be taken every hour it is very possible that every case would show a high temperature at some period in the course of the disease.

The temperature varies from hour to hour, as shown by three of the cases in which it was taken every two hours. It commences to fall very rapidly after reaching its highest point, and becomes subnormal just before death, in some cases descending to even 94° F.

In the very acute cases the marked rise in temperature may be overlooked, as collapse quickly occurs and the temperature rapidly runs down until death takes place. In the more chronic cases, extending over a space of from two to three days, a highly febrile period is always observed.

The rational deduction from the observations appears to be that black-quarter should be regarded as a febrile disease, but that the rise of temperature may be of such short duration that it is likely to be overlooked, and cannot be regarded as of any value in the diagnosis of the affection.—Ed.

DIFFERENTIAL DIAGNOSIS.

Anthrax and malignant œdema are the only diseases with which it is likely to be confounded.

From Anthrax.—Black-quarter is distinguished from anthrax—

1. *Clinically,* by the presence of the emphysematous crepitating tumour—a lesion which is never met with in anthrax.

2. On *post-mortem,* the presence of this tumour, with its characteristic conditions and peculiar sour odour, makes the diagnosis certain. Unlike anthrax, the spleen is never enlarged in black-quarter, and the blood appears unaltered to the naked eye, and forms a clot of normal firmness.

3. *Bacteriologically,* the two diseases are distinguished by the morphology of their causal organisms, the cultural behaviour of these organisms, and their effect upon the ordinary experimental animals, as set forth in the following table:

Black-Quarter.	Anthrax.
Short thick bacillus, with rounded ends, very motile, does not form filaments, often exhibiting a spore which gives it a club or spindle shaped appearance. It is an anaërobe, and mainly a tissue-parasite confined to the lesion.	A longer bacillus of a uniform thickness with square or cup-shaped ends, non-motile, forming filaments, and surrounded in fresh specimens by an envelope or capsule. It is an aërobe, and chiefly a blood-parasite invading the whole system.
In culture a strict anaërobe, producing gas and evolving a peculiar odour.	In culture an aërobe, does not produce gas or give off an offensive odour; produces characteristic growths in bouillon, gelatine, and agar.
It is not destroyed by putrefaction.	It is rapidly destroyed by putrefaction.
Rabbit immune; guinea-pig susceptible.	Both rabbit and guinea-pig very susceptible.
Impossible to infect by cutaneous inoculation.	Easy to infect by cutaneous inoculation.
Intravenous inoculation produces only slight disturbance with subsequent immunity.	Intravenous inoculation always fatal.
Subcutaneous inoculation causes extensive swelling at seat of inoculation.	Subcutaneous inoculation is followed by only a slight or negative reaction at seat of inoculation.

According to Hamilton, the bacillus of *Braxy* is distinguished from the black-quarter bacillus by its comparatively small size and delicacy of contour.

From Malignant Œdema.—The disease is distinguished from malignant œdema by the presence of the characteristic muscular lesion, with its accompanying sour odour. The black-quarter bacilli are actively motile, and occur singly or in very short filaments, while the malignant œdema bacilli are only slightly motile, and form characteristic long filaments. In culture the black-quarter bacilli give off a peculiar odour, while the malignant œdema bacilli produce a putrid odour. Young cattle are susceptible to black-quarter, but almost immune to malignant œdema. Horses, swine, dogs, rabbits, pigeons, and chickens, on the other hand, are susceptible to natural or experimental infection with the latter disease, but are practically immune to the former. Black-quarter is restricted to locality, while malignant œdema is universal.

A disease somewhat similar to black-quarter is described by Carl and Albrecht as occurring in aged cows two to four days after parturition. It is characterised by tumefaction of the external genitals, extending to the sacrum, thighs, and back; the swelling is emphysematous and crepitates. The mucous membrane of the uterus is also involved. This disease has been erroneously termed "parturient black-leg," but in reality, as demonstrated by Carl, it is caused by the bacillus of malignant œdema, and the diagnosis must be made by bacteriological investigation.

From Mechanical Emphysema.—Occasionally it may be found necessary to distinguish between black-quarter and mechanical emphysema of the skin arising from wounds in the region of the neck, sternum, and elbow, or injuries to the trachea or œsophagus. In this lesion, however, there is complete absence of inflammatory changes in the tumour, and puncture of it quickly reveals its true nature.

TREATMENT.

Black-quarter is such a fatal disease, and runs so very acute a course, that treatment is generally out of the question. If a case is discovered early, treatment may be attempted by freely incising the local lesion and injecting strong antiseptics into the subcutaneous tissue. Internal antiseptics might also be tried, but as a rule treatment is useless.

PROPHYLAXIS.

The agriculturist can do much to banish the disease by improved farming, draining, and liming of the infected land. Young susceptible animals should not be grazed on lands known to be badly infected, and young animals in poor condition should not be suddenly put on rich pasture. Affected animals should be immediately isolated and destroyed, the carcass being cremated, or buried deeply in lime. The carcass should be buried in its skin in some convenient place to which animals will not have access, and at a safe distance from any well or watercourse. The place where the carcass lay should be thoroughly disinfected. In the case of infected premises the buildings should be thoroughly cleansed and disinfected, all excreta and litter being burnt, or disinfected and buried.

In the past, various measures were practised as a means of preventing the disease—such as bleeding, purging, setoning, and the administration of drugs, such as carbolic acid; but their efficiency was doubtful, and they have been almost entirely superseded in recent years by various methods of vaccination, which have given much better results.

VACCINATION.

It is possible to confer a high degree of immunity on young cattle by several methods of protective inoculation, two of which, at least, have been practised largely—viz., (1) *Arloing's Method*; (2) *Kitt's Method*.

Arloing's Method.—In Arloing's method two vaccines of different virulence are used, and are prepared as follows: the affected muscular tissue from the tumour of an experimental case is cut out with sterile instruments immediately after death, dried, and powdered. The dry powder is then mixed with sterile water, to convert it into a uniform thick paste, and placed in an incubator for seven hours at a temperature of 100° C. for the first vaccine, and for the same time at a temperature of 90° C. for the second vaccine. After this treatment the material comes out of the incubator in the form of perfectly dry, brittle crusts, which, when pulverised, are fit for use. The dose of either vaccine for a yearling is 1 centigramme. The material is generally put up in vials containing 1 decigramme each, and at the time of inoculation the contents of each tube have to be ground up with 10 c.c. of sterile water. Each c.c. of the mixture will then contain one dose.

The ordinary seat of inoculation is the under-surface of the tail; the first vaccine being injected quite close to the tip, and the second a few inches higher up, after an interval of about ten days. The operation is attended with some difficulty, owing to the density of the subcutaneous tissue of the part. The operation is sometimes modified by omitting the first vaccine, and by operating behind the shoulder; but statistics tend to show that these modifications are attended with greater risk to the animal.

Kitt's Method.—In Kitt's method a single vaccine only is employed, and it is usually injected at the shoulder. The preparation of this vaccine differs from Arloing's in that the virulent muscle-powder is subjected to *moist heat* in a steam sterilizer for six hours, at a temperature of 97° to 100° C. The dose of this vaccine is a decigramme.

The only advantage of Kitt's method is that immunity is conferred at one operation. On the other hand, the double vaccine method, although more troublesome, is less risky for the animal, and invokes a more lasting immunity.

Both Arloing's and Kitt's vaccines are put up commercially in the form of strings, pellets, and powders, by veterinary laboratories and drug companies, and are used extensively throughout the world. Care should be taken when operating to prevent bruising or crushing of the tissues in the vicinity of the seat of inoculation. Immunity is said to follow the application of these vaccines in about a week's time, and last for about nine to twelve months. As to the proper age and time of year to vaccinate, much must depend on local circumstances. In this country calves are generally operated on in the spring or autumn, about the age of from four to seven months ; but the operation can be done at almost any age or season, and should be repeated annually on young bovines in districts where the disease is prevalent. On the other hand, it is, generally speaking, not advisable to carry out vaccination on farms where the disease is only an occasional visitor.

The losses attributed to the operation are very few, when calculated on the large number operated on, but are very unequally divided, amounting in some instances to 5 or 10 per cent., or more, in a small herd.

Serum Vaccine.—Kitt was the first to introduce this method. The serum is now got from horses which have been hyper-immunised by repeated injections of black-quarter cultures. A 10 to 20 c.c. dose gives a high immunity, which is only of a passive character, lasting about one week.

Serum and Virus Method (introduced in France by Leclainche and Vallée).—In this method the animal first receives 10 to 30 c.c. of a protective serum, and about one week afterwards 1 c.c. of a sporulating broth culture, which has been heated for three hours at 70° C. The object of the serum is to set up a passive immunity which will abolish the risk involved in applying the virus. This is said to be the safest of all methods, but is open to the risk that at the time the virus is injected the passive immunity is all-powerful, and therefore the chance of securing a lasting immunity is somewhat doubtful.

Immunity can also be established by the intravenous injection of a small quantity of virus, but the risk attending this method debars it from practical application.

PUBLIC HEALTH.

Although the disease is not transmissible to man, and the flesh not injurious to health, still, as it readily decomposes, and even when preserved develops a rancid odour, it should be invariably condemned as unfit for food.

BLACK-QUARTER IN SHEEP.

As already remarked, sheep are more susceptible to experimental inoculation of black-quarter than cattle, but outbreaks of the disease in these animals are not often recorded. It is, however, probable that the disease may be overlooked or mistaken for other affections of the sheep. McFadyean has met with several instances where on certain farms serious losses have occurred from black-quarter in sheep.* He points out that in sheep the susceptibility to the disease persists throughout life, thus differing from cattle, in which the affection is rather uncommon in animals over two years of age, and very rare in adult cattle.

According to Stockman, lesions of the skeletal muscles may be very slight or even absent in sheep dead of black-quarter.

Cave† investigated an outbreak of the disease that occurred most commonly on the Romney Marsh, and also in other marshlands of Kent, and occasionally in upland districts. The disease was locally known as "struck," and various theories were held by the flock-owners as to its true nature. Sheep of all ages were attacked, and, as in the case of cattle, it was generally found that the best animals in the flock were most susceptible. Several cases occurred in pregnant ewes—sixty of these dying in a few weeks out of a flock of 200; these fatalities occurring in a district at some elevation above sea-level, where the loss from the disease was said to be usually slight.

The lesions found were identical with those of black-quarter in cattle, and microscopical examinations of the exudate from the diseased muscles showed the presence of the black-quarter bacillus; this organism was also found in the blood and in the pleural and peritoneal fluids. The diagnosis was confirmed by McFadyean.

The course of the disease was very rapid, and the animal was generally found dead in the pasture, without any history of previous illness; in some instances the animals staggered, fell to the ground, and died in a few minutes. More rarely death was delayed for some hours; the animal stood with all four feet close together, its back arched, and its respirations greatly accelerated; a frothy, blood-stained discharge issued from the nostrils, and diarrhœa, with blood-stained fæces, was also present. The animal was made to move with difficulty, and showed considerable stiffness of the limbs. It soon fell to the ground, and quickly died. It was not decided whether any marked swellings were present during life, as, in consequence of the rapid onset of the disease, the suddenness of death, and the thickness of the wool, this point was difficult to determine.

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* *Vide Journal of Comparative Pathology and Therapeutics*, 1901.

† *The Journal of Agricultural Science*, vol. i., part ii., May, 1905.

Inoculation of a healthy sheep with the serum obtained from the diseased muscle of an affected animal, the fluid being injected deeply into the muscles of the thigh, caused death within twelve to fourteen hours after the inoculation. The inoculated animal presented all the appearances of an ordinary case of the disease, and microscopical examination of the exudate showed the presence of numerous black-quarter bacilli. Cave conducted numerous experiments with a view to immunising sheep against black-quarter, the vaccines used being those employed for a similar purpose in cattle. He arrived at the following conclusions:

"1. That the double vaccine *A* undoubtedly renders sheep strongly immune against black-quarter.

"2. That the double vaccine *A* is uncertain in its action, and under certain conditions may be highly dangerous when used in the field.

"3. That a single vaccine may be powerful enough to kill and may yet fail to give a lasting immunity to those animals which survive the inoculations.

"4. That none of the vaccines tried in these experiments have proved entirely satisfactory, although enough has been done to show that the loss from 'struck' sheep could be greatly diminished by protective inoculation, if a safe and efficient vaccine were available.

"It is possible that more favourable results will be obtained in the future by the use of vaccines specially prepared for sheep."

GLANDERS

By WILLIAM HUNTING, F.R.C.V.S.,
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Synonyms.—Equinia; Farcy.

Latin: Malleus.
French: Morve.
German: Rotz.

Definition.—Glanders is a specific disease due to the growth and development within the animal body of a micro-organism, the *Bacillus mallei*. The disease is characterised by morbid changes affecting the skin, mucous membranes, lungs, lymphatic glands, and other organs. These changes vary according to the activity and position of the causal organism, and are modified by the health, age, and condition of the infected animal.

HISTORY.

Glanders was recognised by Hippocrates about 450 B.C., and by Aristotle a century later. It was described by Apsyrtus in the fourth century of our era, and by Vegetius in the fifth. In 1618 the celebrated Italian anatomist Ruini described glanders, and declared it to be "a general disease and incurable."

No writer of the seventeenth century expressed any clear ideas about the nature of glanders, but they all seem to have observed that it was contagious, and it would have been well for horse-owners had this simple experience not been upset by theories of less acute observers.

In the eighteenth century some progress was made, and a French veterinarian, Gaspard de Saunier, 1734, recognised glanders as "highly contagious," and traced its transmission through harness, water-troughs, pails, and mangers. Unfortunately, in 1749, Lafosse, Senior Farrier to the King, communicated to the French Royal Academy of Medicine "A Memoir of the Glanders in Horses," in which he argued that the seat of the disease was in the nasal cavities, and he concluded "that glanders does not depend upon a general distemper of the blood, but is really and truly a simple and local malady." For half a century this non-contagion theory obtained general acceptance, although a few eminent veterinarians attempted to refute it. Lafosse's opinions gained weight from his prominent position, and their wide acceptance was disastrous to the adoption of any rational preventive measures.

Bourgelat, the founder of modern veterinary science, who established the schools at Lyons and Paris, was a believer in the contagious nature of the disease, but it was not till the nineteenth century had almost arrived that general acceptance was given to the view that glanders might be contagious; and even then another fifty years was to pass during which authorities held that, although contagion played a part, yet other causes might give origin to the disease. The Lyons School and its graduates supported a contagious origin, the Paris School took the opposite view, and so prevention was neglected.

In 1773, Vitet expressed the opinion that glanders consisted in a discharge from the nose of "a virulent and contagious humour," and he stated that "the chest and the head were the seat of the disease." In the same year, Volpi of Milan thought he noticed an analogy between syphilis and glanders, and suspected that glanders and farcy, from their frequent association, might be the same disease. He also asserted that "glanders is caused by contagion alone."

In 1797, Eric Viborg, a Danish veterinarian, published a work on glanders in which he showed that there was a specific virus in the purulent discharges of horses suffering from glanders and farcy; that farcy and glanders were one disease; that the virus could be destroyed by heat and desiccation; and that infection could be carried by harness or utensils, and more particularly by forage left in mangers from which diseased horses had fed. He also recognised a latent form of disease which might be infective, and warned against the danger from horses which had been apparently cured of farcy. It is sad to think of the losses which have since occurred, and which might largely have been prevented had the work of this excellent observer been accepted. Viborg, in 1797, knew practically all that was known about glanders up to the time of the discovery of the *Bacillus mallei.*

In the nineteenth century knowledge of glanders progressed slowly at first. Professor Coleman, who had succeeded St. Bel at the London Veterinary School, devoted much attention to glanders. He proved experimentally that farcy might be produced by inoculation of matter from acute glanders, and glanders by the inoculation of matter from farcy. He showed that the virus might exist in the blood by inducing glanders in a donkey through transfusion of blood from a glandered horse, and yet he held that the disease might originate without contagion.

Professor Sewell, in 1827, taught that the lungs were the original seat of the disease and the affection of the nose secondary. He believed that the miliary nodules in the lung constitute the original disease.

In 1840 a series of experiments were made by Renault and Bouley at Alfort, which established beyond a doubt that glanders was contagious; and since then very few men have ventured to deny that glanders may be transmitted by contagion. But many have believed that it might arise from other causes, such as the introduction into a horse's system of purulent fluids, or even by changes in the blood resulting from defective hygienic conditions.

In 1843-50 William Percival published his well-known work on "Hippopathology—a Systematic Treatise on the Disorders and Lameness of the Horse." In vol. iii. (revised edition, 1860) is an article on Glanders, extending to 200 pages, and embracing a most careful and extensive review of the work done on the disease by every writer from Solleysel downwards.

Percival himself says: "No doubt has ever been entertained by me of the spontaneous origin of glanders and farcy—of their origin apart from the influence of contagion." Further on we find: "Of the nature of the (so-called) virus of glanders we know no more than we do concerning the supposed viruses or poisons of syphilis, rabies, or variola. We have the same ground for arguing the existence of virus as there is for doing so in the diseases just named, and no more. . . . We do not imagine that simply an unhealthy or ill-conditioned state of the body can give rise to glanders or farcy. We believe that the specific virus must, in some form or another, somewhere or other exist."

For many years after Percival wrote, the views expressed by him were generally accepted, and few practitioners disbelieved in the occasional spontaneous origin of glanders. "Miasmas" or "mephitic vapours" arising from bad ventilation and overcrowding, tempered by climatic changes of an indefinite kind, were the assumed cause of outbreaks which could not be traced directly to contagion. Thus practical measures of prevention were neglected, or limited to the isolation of the most marked cases.

In 1882 the causal organism, *Bacillus mallei*, was isolated and cultivated in France by Bouchard, Capitan, and Charrin, and, about the same time, independently by Loeffler and Schütz in Germany.

In 1891 the Russian veterinary surgeons, Helman and Kalning, obtained from sterilised cultures of the bacillus a product called "mallein," which possesses the remarkable property of causing a specific reaction when injected into glandered horses, but which has no effect in horses free from glanders. Schütz, Roux, Nocard, McFadyean, Kitt, and Babes, have since added considerably to our knowledge, and Governments in various parts of the world have issued regulations for the control of glanders based upon the results of veterinary observation and scientific research. During the years 1891-92 no less than seven European scientists working at glanders lost their lives through accidental infection.

DISTRIBUTION AND PREVALENCE.

Glanders has been distributed by trade or war to every country in the world. In Europe it has been known for centuries. In India and China probably as long. All the great wars have increased its prevalence, and often carried it into districts where previously it was unknown. In the Crimea glanders prevailed widely. In the Italo-Austrian, in the Franco-Prussian, and in the Civil War of the United States, glanders prevailed not only during the wars, but even more actively after peace was made, by the movement and dispersal of superfluous horses.

Our minor wars in Egypt, Abyssinia, and Afghanistan were all attended by glanders to an extent which interfered with efficiency. The United States are said to have introduced glanders into Mexico and Cuba with their army horses. The South African War, where 240,000 horses perished, was not only accompanied by the prevalence of glanders, but left the whole country infected. The disease was carried to South Africa from home, from America and Australia, and possibly from Europe, by the remounts.

By trade we have received glanders from the United States, Canada, and Europe, and we have returned it. The trade in old horses to Belgium has introduced glanders to that country frequently. Probably no civilised country on the face of the earth is now free from the disease.

In Great Britain few counties remain free for any length of time, but the disease chiefly prevails in the large towns—in London and Glasgow especially.

ETIOLOGY.

Glanders arises from the development of a micro-organism, the *Bacillus mallei*, in the system of an animal.

In 1882, Loeffler and Schütz, in Germany, discovered the bacillus; so, too, about the same time, did Bouchard, Capitan, and Charrin, working independently in France. They isolated it, cultivated it, and produced glanders in animals by inoculation of the culture.

The bacilli are found in the discharges from glanders lesions, in unopened farcy buds, and in the nodules formed in internal organs. In the lungs the most recent nodules afford the larger number of active bacilli.

BACTERIOLOGY.

The microbes are rod-shaped organisms, about 3 to 5 microns in length by 1 in breadth. The extremities are rounded. The bacilli are non-motile, can be stained by suitable reagents, but are not very retentive of the stain.

They can be cultivated in suitable media in the presence of air and at temperatures varying from 75° to 103° F. They cease to grow at 112° and below 60° F. At 130° F. they are killed. Upon agar the colonies form a whitish layer. Upon potato there forms a yellowish, honey-like layer which becomes gradually darker, varying from light brown to chocolate. The formation of spores has not been demonstrated. These cultures, when inoculated into susceptible animals, are more virulent than any products of the natural glanders lesions. Accidentally men have been infected by needles and syringes which had been used for experimental inoculation of animals, and in every case death from acute glanders followed. "Horses, asses, mules, cats, goats, field-mice, and guinea-pigs, have all been infected. Rabbits, sheep, and dogs are slightly susceptible. Cattle, swine, and white mice have an immunity" (Crookshank).

By passing the bacilli through different animals their virulence may be decreased or increased, and it is said that the microbe becomes most virulent in man.

Subcultures rapidly lose their virulence. The bacillus can with difficulty be isolated from the blood. It probably does not retain its vitality long in the blood-stream; but that it exists there in acute cases is shown by the successful production of glanders by transfusion of blood from a diseased into a healthy animal.

The bacilli of glanders are killed by exposure to bright sunlight, by desiccation, and by a temperature of over 130° F. They remain active for about eighteen days in clean water, and cannot retain their vitality out of an animal body or a bacteriological laboratory for more than three months—seldom over six weeks. Most disinfectants destroy the bacillus rapidly. A 5 per cent. solution of carbolic acid or a solution of corrosive sublimate, 1 to 1,000, are quite reliable. Lysol, permanganate of potash, chinosol, and many others, may also be used.

INFLUENCES AFFECTING INFECTION.

As some species of animals are immune to glanders, and as some have a partial immunity, so some horses, if not altogether immune, show a strong resistance. When it was permissible to treat cases of glanders and farcy, there were instances of apparent recovery after the exhibition of clinical symptoms. Many horses made partial recoveries, and some recovered and worked for years without ever showing any further sign of disease. Horses have frequently been known to work with, and to stand in the stables alongside, visibly glandered horses, and yet never show a sign of infection. The most probable explanation is that they were infected, but did not develop the disease. I am inclined to doubt whether any horse was ever sufficiently resistive to be immune to a full dose of glanders virus such as exists in a pure culture, but my experience warrants me in saying that numbers of horses naturally infected have lived and worked for years without giving evidence of their infection.

Age has a decided influence on the susceptibility. Old horses withstand the disease to a marked extent. Young horses are easily infected, and the period is short before they give signs of their condition. Overworked or badly-fed horses are easily infected, and have no resistance to the rapid development of the bacilli. The most resistant horse is the animal in sound, hard condition.

Other influences which affect the development of glanders are certain drugs, debilitating diseases, chills, and injuries. So well known was the influence of excessive doses of physic that in suspected cases where no clinical symptoms were visible it used to be a common practice to administer a full dose of aloes. The superpurgation which followed was often accompanied by the signs of glanders.

Following upon influenza or acute chest affections, glanders commonly develops if the patient be suffering from the latent disease. Wounds and injuries are frequently the cause of latent glanders awakening into active disease. In a stud where latent glanders existed it was no uncommon event to have farcy in a leg following upon injury to the foot by a nail in shoeing. That the injured part should be the first to show symptoms of the disease is, I believe, explainable on the supposition that in chronic cases of glanders every now and then there is an escape from the lungs of stray bacilli, which get into the circulation, but soon perish if no suitable place for lodgment is found. A suitable place is found in injured tissues, and then the bacillus is detained and actively develops, giving rise to definite lesions.

That acute lung diseases may hasten the development of glanders when active nodules are in the lungs I think is certain, but it is also true that pulmonary glanders which has existed for months without any indication of its presence may give rise to pleurisy and pneumonia. At a postmortem examination, when acute pleurisy and old-standing glanders lesions are found, I know no way of distinguishing by naked-eye examination whether the pleurisy is specific or otherwise.

SUSCEPTIBILITY OF DIFFERENT ANIMALS.

Nearly all warm-blooded animals are susceptible to glanders infection except the ox, pig, and white mouse. The ass is particularly susceptible to infection, and generally succumbs rapidly to an acute attack of the disease. Chronic cases of glanders in the donkey are almost unknown. The mule is easily infected, and the resulting disease is usually rapid. It takes a sort of intermediate position between the horse and ass. The larger carnivora have suffered from glanders by the ingestion of uncooked flesh of glandered horses. The disease in these animals was probably produced by flesh from subacute cases of equine glanders, as the blood in chronic cases does not seem dangerous. Dogs are very resistant, no case having been reported of natural infection. By inoculation a local sore is produced, which heals spontaneously without producing a general infection.

Man suffers both from the chronic and acute form of glanders, and may be infected either by ingestion or inoculation.

PERIOD OF INCUBATION.

This expression is generally understood to mean the time which elapses between the date of infection and the appearance of the disease. When horses are infected by *direct inoculation of active virus* from a glandered animal, they give evidence of infection in from *three* to *five* days. The local sore becomes angry and ulcerous, whilst the lymphatic vessels are distended and the nearest lymphatic glands enlarged.

When horses are *naturally* infected, or when experimentally given glanders virus in their food or water, no evident symptoms of disease are noticed for some weeks, or it may be months. Recent experiments, done with more care than was taken a generation back, suggest that infection takes place and produces some constitutional disturbance, though slight and temporary, long before the visible appearance of external symptoms. The daily use of the clinical thermometer after infection frequently indicates a rise of temperature in a few days. A post-mortem examination discloses nodules in the lungs.

In *natural infection* the usual period of incubation—that is, the time between the reception of the virus and the appearance of clinical symptoms—is *from one to three months,* but in many cases much longer. The fact is that, although after infection no clinical symptoms may arise for an indefinite time, glanders lesions are being formed in the lung, and this latent pulmonary disease may, according to the state of the horse and the influences affecting him, develop slowly or rapidly, and in some cases cease to develop.

METHODS OF INFECTION.

As the only cause of glanders is the *Bacillus mallei*, no case can arise except by the passage of the bacillus from a diseased to a healthy animal. Of course, the passage need not be direct. The bacillus may leave its habitat, and remain on some place or material from which it may reach the system of the animal. So long as it retains its vitality out of the body, it may be infective. Thus it is that the exact way in which the disease is spread is often involved in doubt. A glandered horse may leave infective material on food or in water and pass on. The food or water may not be consumed by a healthy horse for some time after, and the connection between the infecting and infected horse is never known, but is none the less real.

Horses suffer from glanders lesions which may be open or closed. A farcy bud on the skin allows no escape of bacilli until it is opened. A glanders nodule in the lung is for a time closed, and only when some change occurs in it do bacilli escape—it may be into lymph vessels or it may be into bronchi. In the one case they may pass from the bronchi up the respiratory passages and set up ulcerations on the mucous membrane; in the other they escape into the lymph-stream, and if not soon detained to form fresh centres of infection in the lung, they reach the circulation, and may give rise to lesions in any part of the body.

The open lesions of glanders are those in the air-passages—trachea, larynx, and nose—and on the skin, "buds" or tumours which have broken and ulcerated. The discharges from these contain the virus, which spreads infection, either directly or indirectly, to healthy horses.

There are three ways by which the virus may enter the body: (1) Through the skin or mucous membrane; (2) through the air-passages by inhalation; and (3) by ingestion through the digestive tract.

Direct experiment has demonstrated that susceptible animals may be infected with certainty through the skin or mucous membrane, if these are injured. Inoculation introduces the material through the skin. An abraded surface permits infection to pass through, but it is very doubtful if a sound membrane or skin does allow the infection of glanders to pass. Successful infection by rubbing in virulent matter has been attained, but the rubbing may have destroyed the structure of the protecting surface. Abrasions and wounds have frequently afforded access to the poison of glanders. Splinters of wood from an old manger that has been used by a glandered horse might be infected, and then penetrate the skin of the lips of another horse. Sores might be inoculated by virus accidentally transferred on saddles and bridles. A glandered horse, by licking or biting, might infect a healthy horse. It has often happened that disease has been transmitted by cloths or sponges used on an unrecognised case of glanders, and then employed to clean or foment wounds on a healthy horse.

Direct experiment has also demonstrated the certainty with which glanders can be conveyed through food and water recently contaminated by virus from a diseased animal. Viborg, in 1793, recognised this fact by clinical observation. Simonds gave water containing glanders matter to healthy horses, and transmitted the disease. Renault and Bouley, in 1840, obtained the same results. All these early experimenters laboured under the possible fallacy of their experimental subjects being already infected before being made to swallow the virulent material. No such error could vitiate the experiments of Schütz, Nocard, and McFadyean, who all tested their subjects with mallein before proceeding. These experimenters used pure cultures, and passed them into the stomach with precautions against any accidental inoculation of the mouth, pharynx, or œsophagus. They successfully infected their subjects, and could always find glanders nodules in the lungs, though seldom any lesions in other organs.

From my own clinical observations, I long ago arrived at the conviction that not only is infection possible by ingestion, but that this mode of introducing the poison is by far the most common in practice. In a cab stable where two horses work a cab, but stand in different stables—a night stable and a day stable—the disease in one is usually followed by infection of the other, the most direct medium being the nosebag. In ordinary stables, a detected case of glanders is usually followed by the appearance of disease in the horses standing on either side of the diseased one. The transmission of glanders by a common water-trough, or by pails, in stables where glandered horses are kept and worked has been noticed and recorded frequently.

It is beyond doubt that glanders bacilli may pass through the stomach and reach the lungs, leaving no trace of their line of progression, but giving rise to unmistakable evidence of their arrival. The bacilli of glanders escape from the nose of an infected horse, with pus or mucus, in a wet condition, and are not long floating in the air. They fall upon mangers, walls, forage, water, and so are ready for ingestion. If they become dry, they must be detached from the surface on which they are dried before being capable of floating free. Desiccation is fatal to the bacillus. Even supposing a few chance bacilli retain their vitality, and, floating in the air, are drawn in with the breath into a horse's nose, how many are likely to escape detention by the moist passages of the nose, pharynx, larynx, and trachea, and to gain access to the lung? The analogy of the miner and the coal-dust is not applicable. The air which caused miner's lung was absolutely full of suspended particles, and improved ventilation in mines has rendered miner's lung a rare curiosity. How many glanders bacilli, dry but vital, could be found in a cubic yard of air in the most frightfully infected stable is a metaphysical puzzle incapable of solution.

It may be added that experimental attempts to produce glanders through aerial infection have never been successful.

METHODS OF SPREAD.

I have just attempted to show how the infective virus of glanders leaves a diseased horse, and how it enters the system of a healthy one. The fact that the virus remains active outside the body of an animal for at least some days renders it necessary to keep observation, not only upon the diseased horse, but upon the various ways in which infection may be carried. Most important of all is it to know that a horse, apparently quite healthy, may be a source of infection. The latent form of glanders is probably not infective until some open lesion occurs, but this may happen without warning. Ulcerative lesions may arise in the trachea, rendering the horse infective, and afford no clinical evidence until they have largely developed—a dangerous condition extending over some days, during which time the horse is excessively dangerous to all around him, but is unsuspected.

Then, again, visible disease may not be recognised. Although a very evident nasal discharge exists, it may be mistaken for catarrh, and so mangers and pails are infected, and other horses are allowed to use them. Every nasal discharge should be looked upon with suspicion until its nature is satisfactorily diagnosed. Sponges, leathers, combs and brushes, may carry the disease from one horse to another if they are used upon an infected animal. Of course, precautions would be taken if it were known that a diseased horse were on the premises, but latent disease is not recognisable, and it so suddenly develops in many cases that contagion is carried before it is known to be present.

There are in London many infected studs. Bankruptcy and death cause the sale of these, and the owners, without selling a single developed case of disease, often spread infection to many centres by distributing horses suffering from latent glanders.

In large studs having many different stables, glanders is spread to a great extent by the constant changes of horses from stable to stable to suit the exigencies of business. When once infection is introduced to such a stud, the changes of horses spread the disease to every stable unless very careful measures are taken to prevent it.

Newly-purchased horses are a frequent means by which glanders is brought into a stable. Purchasers who confine themselves to young horses from the country seldom introduce glanders, but men who buy aged town horses are never long before they have the misfortune to get an infected horse.

A horse put up at livery may eat out of a manger which has been used by a glandered horse. A healthy horse may stand next to a diseased one in a shoeing-forge, or in a railway horse-box. Drivers of horses may change nosebags, and one of the horses be glandered. Horses may be turned out to grass in the autumn, and there meet with an infected animal. I once saw five visibly glandered horses in a field with thirty others. Probably these animals belonged to ten different owners, and ten different stables would ultimately become infected.

In no small stud does glanders remain long, because it soon kills all the horses. The disseminating centres are the large studs, and the spread from them is by the cast horses with latent disease which are purchased by the poorer owners, and not suspected till too late.

Out-of-the-way places—villages and farms—have become mysteriously infected, and some such cases have been directly traced to travelling shows, circuses, and gipsies. Of course, all sales, fairs, and markets may disseminate infected horses. The way to prevent such an occurrence is not to close the yards and markets, but to look after the infected animals, and detect the latent cases.

One method of spread I must specially refer to—the public water-trough. It is quite possible that some cases of glanders have arisen as the direct effect of drinking at a public trough, but they are very few and far between. I have an intimate knowledge of the stables of three contractors who have had, during the last twenty years, four outbreaks of glanders in their studs. Each outbreak was clearly and directly traceable to the purchase of a horse from an infected stud, and was stamped out at once without spreading. Save these outbreaks, no glanders has troubled them, and yet their horses travel all over London, and drink at any water-trough they can reach. I feel convinced that infection from water-troughs is very rare, because in 90 per cent. of all outbreaks which I have personally investigated other methods of infection are traceable. Even if 5 per cent. of all outbreaks in London were traceable to the water-troughs, the gravity of the risk would be no argument in favour of closing the troughs, especially in summer. The harm resulting to horses from being denied water all day would cause a mortality greater than is caused by all the glanders in the Metropolis. The fact that an occasional case of glanders may be due to public water-troughs is an argument for the extermination of the disease, but not for closing the troughs.

MORBID ANATOMY.

There is only one post-mortem lesion that may be expected in every case of glanders—viz., nodules in the lungs. On two or three occasions I have failed to find pulmonary nodules in horses which showed during life distinct symptoms of farcy, and I believe these few cases were due to primary infection of the skin, which directly produced cutaneous lesions, and, with sufficient time, would have affected the lungs. The large majority of cases of farcy do not arise in this way. They are secondary signs of a long-established latent infection. This is demonstrated by the old lung lesions found on post-mortem examination.

Lung Lesions.—No matter in what way the causal organism enters the body, it will find its way to the lungs and produce nodules if the horse survives for a week or two after infection. The lung nodules persist, whilst the lesions caused at the point of entry may entirely disappear.

The *pulmonary nodule* is the most important of glanders lesions; it is also the most durable. Ulcers on the skin may heal and disappear; ulcers on the mucous membrane of the respiratory passages may heal, and leave only small cicatrices; but the lung nodule remains. Everywhere on the outside of the body the tendency of a glanders lesion is to suppurate and ulcerate. In the lungs the nodule containing active bacilli alters but slowly, and the changes taking place in it present little tendency to invade neighbouring tissues or to set up a violent necrotic action. Since horses have been slaughtered solely on the reactions to mallein, lung lesions have been examined in an earlier stage, and more or less free from the complications which accompany the generalisation of the disease. Even under these conditions, variations in the size, form, and quality of the nodules are noticeable, due principally to the age of the lesions, but also to the resistance offered by the pulmonary tissue of the individual horse.

I need hardly warn professional observers of the difference in all lungs brought about by the method of death, and by the time which elapses before a post-mortem is made. A horse that dies of glanders, and upon which a post-mortem is held some hours after, shows a deeper colour of all the organs than one which is killed, and probably bled, just before the examination. The side upon which an animal lies after death always shows a darker colour than that which is uppermost.

There is a great variation in the appearance of lungs taken from glandered horses. Many pulmonary changes are found which have resulted from inflammatory attacks having no connection with glanders, which occurred, perhaps, months previously, and which have subsided so far as to show no symptoms of the disease during life. These attacks leave portions of the lungs altered in colour and density. Parasitic invasions also leave their mark, sometimes in the form of large cysts, and sometimes as small, hard, calcareous particles. The small calcareous bodies which feel like shot under the pleura, when of parasitic origin, are generally more uniform and more widely spread throughout both lungs than the glanders nodules. They are all about the same age and density. They are more easily enucleated from their capsule, and the enveloping border is more definitely marked off from the surrounding tissue than in the case of the typical glanders nodule. Another cause of pathological variety is the specific pneumonia or pleurisy accompanying generalised glanders; also the pulmonary inflammations which may accidentally or coincidently arise in horses already suffering from latent glanders, and which superimpose their lesions upon those of the specific disease. In a case of gangrenous pneumonia, latent glanders in the lungs is generalised with startling rapidity, and the complication may be overlooked unless care is exercised.

The Typical Glanders Nodule is a firm, round body, varying in size from a pin's-head to a horse-bean. It may sometimes be seen as a little eminence under the pleura, but is best found by passing the fingers with gentle pressure over the wet surface of the lung. If the lungs have been removed for some hours from the carcass, and their surface allowed to become dry, nodules are not easily felt; they should be examined immediately after removal, or should be wetted before an attempt is made to detect nodules by touch. Nodules frequently exist and afford no evidence to the eye, whilst readily distinguished by touch.

The more recent nodules may often be recognised as dark circular spots visible through the pleura. When felt with the finger, they yield the impression of firm round objects embedded in the lung. Dark spots upon the pleura are not uncommon in horses quite free from glanders, but they are superficial, not nodular.

The term "miliary glanders" is sometimes used to describe the small nodules found in the lungs. It suggests at once a comparison with the miliary lesion formed in tuberculosis, in which thousands of small tubercles are distributed evenly all through the lungs. Such a condition is seldom found in glanders, although nodules may exist throughout one or both lungs.

The glanders nodules are found irregularly here and there, chiefly on the upper surface of the lungs—as a rule, more in one lung than the other. They often differ in age—some so old as to have become gritty, some caseous, and some merely dark hæmorrhagic centres.

Continental observers have described as the earliest stage of lung lesion due to the lodgment of the *Bacillus mallei* a yellow semi-transparent speck about the size of a pin's-head. I confess I have not yet recognised this condition. The earliest change I am conversant with is a dark hæmorrhagic sphere, not very clearly defined from the surrounding lung tissue at its circumference, especially when exposed by a deep cut into the lungs. How long this condition remains I do not know, but from a few cases in which the period of infection was known I estimate such nodules to have been in existence for about a month or six weeks. The next stage is for this dark spot, when incised, to present a centre of a yellowish-grey colour, which in course of time increases in size and becomes lighter in colour. As the light-coloured centre increases in size, the surrounding dark zone contracts and becomes more distinctly defined from the surrounding lung. This change goes on until the whole nodule consists of a caseous grey mass, varying in size from a pin's-head to a pea. The caseous nodules, if the horse lives, undergo a further development; they become gritty from calcareous alteration, and in some of the very oldest lesions we have a hard, solid, round body, not unlike a coated pill, which may be enucleated from its surrounding capsule. The fact of nodules of varying age existing together in a lung is not due to various dates of infection, but to the more or less constant escape of bacilli from the earlier nodules. These bacilli become lodged, and form fresh nodules, and so we have from one infection an increase of nodules which may continue for years. There are often very few nodules in a lung, but no one should say there are none until he has not only felt every part of the organ through the uncut pleura, but sliced the lungs and examined each section with fingers and eyes.

When making deep incisions into a lung in search of glanders lesions, it should be remembered that the round nodule may slip aside from the knife, and then does not disclose the characteristic light-coloured centre. It appears as a dark round prominence on the cut surface, and should be carefully divided so as to display its centre.

Every stage of the nodule may be found deep in the lung tissue, as well as just under the pleura; but they are less easily detected, owing to the colour of the surrounding tissues and to the hæmorrhage following the incision.

In some of the older cases very few dark-coloured nodules are found, but the incised surface shows numbers of small grey caseous masses, about the size of a small mustard-seed, without any hæmorrhagic zone surrounding them.

So far I have attempted to describe a typical glanders lesion, but there are others not so well defined. There is a nodule or small tumour, varying in size from a hazel-nut to a walnut, which is easily seen as a prominent object under the pleura, this membrane being opaque and thickened over it. When cut into, the nodule is of irregular form and partly caseous. The section is not like that of the typical nodule which has a well-defined centre. It appears to have penetrated into the lung substance—a sort of interstitial infiltration—so that portions of lung tissue intersect the nodule, especially at its circumference. Sometimes these lung nodules show, when cut into, three or four separate caseous specks, or each may be a distinct abscess.

Pneumonia is always present in acute cases, and the whole lung is dark and congested. In some places softening has occurred, and small abscesses are found; but cavities such as occur in tuberculosis are rare in glanders. Large dark-coloured masses, the size of an orange, are sometimes seen, which, when cut into, show one or more small abscesses. Similar lesions are seen in cases of pneumonia not accompanied by glanders, and therefore they cannot be accepted by themselves as evidence of the specific disease. When glanders nodules happen to be situated in a portion of inflamed lung, they very rapidly develop, forming small abscesses, containing pus of a firmer and more caseous quality than that due to other pulmonary inflammations.

Pleurisy is not uncommon, and may be specific or coincidental with latent glanders. A firm layer of tough adherent lymph covers the pleura, and effectually prevents the detection of nodules by the hand. Unless a lung is well filled with nodules, the changes resulting from acute pleurisy may render it very difficult to decide whether pulmonary glanders is present or not.

Fibroid Changes in the lungs as specific glanders lesions are not often seen. Occasionally a swelling the size of a small apple is met with, which, on being cut into, is found firm, hard, and light-coloured. Probably such morbid changes have no relation to the glanders lesions also present. The extensive fibroid change which is described as due to old-standing tuberculosis is very rare in glanders. Only once have I seen a lung the borders and apex of which were so firm and hard that it could hardly be cut with a knife. That case, I know, was infected for nine years, although it showed no clinical sign of disease, except for the first three months of that time.

The Spleen.—Next in frequency to the lungs, nodules are found in the spleen. They may be detected by the fingers as small, firm bodies under the capsule. They are generally rendered very noticeable by a red or a reddish stain of the capsule covering them. When cut into, they resemble the pulmonary nodule, but are seldom seen in the hæmorrhagic stage, if that is ever the primary state of a splenic nodule. The grey mass which they usually present when incised is firmer than the similar lesion of the lung.

The Liver is not often the seat of glanders nodules, but they are occasionally found in this organ. They present the form of a whity-grey spot on the capsule.

The Kidney is very rarely the site of glanders lesions, and, when they are found, their nature can only be determined by biological experiment.

The Intestines are said to disclose glanders ulceration to careful observation. I have never seen such lesions. They would be most likely to exist very shortly after ingestion of active bacilli, or in a very acute case of generalised glanders in which the virus from ulcerated respiratory passages passed into the pharynx and then through the stomach.

Lesions of the Respiratory Passages.—The lesions found on the mucous membrane lining the respiratory passages are *vesicles*, *nodules*, *ulcers*, *infiltration*, and *cicatrices*. These, except, perhaps, the infiltrations, are always secondary lesions, caused by the escape of bacilli from nodules in the lungs. I have never seen ulceration of the nasal or tracheal mucous membrane without finding nodules in the lungs of an age much greater than that of the ulcerations.

The Vesicle is a small circular blister, almost transparent. In a few hours the upper part of the raised membrane gives way, disclosing a red sore, which rapidly proceeds to ulcerate. These vesicles may be solitary, but more often are found in clusters. As each breaks down and forms a sore they coalesce, and thus we have the rapid appearance of extensive ulcerated patches so noticeable in the more acute cases of glanders.

Nodules may occur on the septum, larynx, and trachea, but are not common in these situations in the horse. They are small, white, firm bodies about the size of a small pea, or less. They are more persistent than the vesicle, but ultimately break down, forming a sore, which rapidly ulcerates.

Ulceration.—This is the most marked lesion of glanders. It is found in all clinically developed cases either on the skin or on the respiratory mucous membrane. As a rule, the ulcers are distinct and separate, but frequently we find them coalescing to form a large continuous ulcerated surface, and I have seen cases in which the whole of the nasal mucous membrane was affected, as well as most of that covering the larynx and trachea. The turbinated bones and the large sinuses of the head are often affected, in addition to the nasal cavity. Even the guttural pouches and Eustachian tubes have been found ulcerated.

Tracheal ulceration presents two very different forms, one—the more common—in which large solitary ulcers are found quite apart from each other. They are not below the surface of the surrounding tissue, but prominent and raised, dark in colour, and bathed in pus. The other form resembles a series of red streaks running always vertically to the direction of the trachea. They are not solitary, but are found in great numbers, often extending over the whole length of the mucous membrane. They may be well described as narrow slits through which protrudes a bright red granulation.

Laryngeal ulceration usually takes the form of isolated ulcers, varying in size up to that of a sixpence. They are found on the epiglottis, on the vocal cords, and on the border of the arytenoids, as well as on other parts of the laryngeal membrane. In a few cases the whole larynx presents one continuous ulcerated surface.

Nasal ulceration is usually found in long patches on the septum nasi, and a similar form is seen on the membrane covering the turbinated bones. In some cases it is located high up on the septum, in others we find only one, or perhaps two, small ulcers at the lower end of the cartilage, which are visible during life.

In some chronic cases, when nasal ulceration has been continued for a long time, we may have perforation right through the septum, and this condition is found more often at the lower extremity of the septum than at the upper.

The usual colour of the ulcerating surface on the membrane lining the respiratory passages is an angry red, covered by a blood-stained muco-purulent secretion. In a few cases we find a pale yellowish-grey surface covered with a viscid translucent mucus. This condition is the healing stage of the ulcerative process.

Cicatrices are found in the trachea, larynx, and nose. In the two first positions they are generally small, but on the nasal membrane covering the septum they may be extensive. Their usual size is about that of a shilling or less. They are irregularly stellate in form, smooth, and white. In conjunction with a recent cicatrix, it is not uncommon to find neighbouring lesions in a healing stage; instead of the angry coloured ulcerations, we see straw-coloured masses of granulation tissue.

Nowadays, when cases of glanders are closely looked after in London, and slaughtered as soon as detected, these cicatrices are becoming very rare. A stellate cicatrix on the septum of a horse so seldom results from anything but a healed glanders ulcer that its presence should suggest an immediate test with mallein.

Infiltration.—In glanders the nasal septum is often the seat of thickening and discoloration, with no ulceration. Sometimes the whole substance of the membrane is a bluey-black colour, raised and thickened by effusion beneath it. When the discoloration is only partial, the rest of the membrane is prominently marked by distended small veins. I have found portions of the membrane covering the septum almost black, and raised quite ½ inch by effusion under it. On cutting through the membrane a dark sanguineous mass was found, firmer than clotted blood, almost the consistency of a soft tumour. I ought to add that in some cases of farcy I have seen this infiltration accompanied by very few pulmonary nodules, and I am not sure that, like the ulcerations, it is always due to the escape of bacilli from the lungs. So frequently in cases of farcy do I find the membrane covering the septum infiltrated, but not ulcerated, that I have formed the opinion that the nasal membranes are specially selected parts for the attacks of glanders, just as the feet and buccal membranes are for foot-and-mouth disease in the ox. The frequency of nasal ulceration is easily accounted for by the passage upwards from the lungs of glanders bacilli. But nasal infiltration is found accompanying cutaneous glanders, even in cases where no pulmonary nodules can be detected.

Glandular Lesions.—Among the symptoms of glanders I shall mention enlarged glands. Post mortem they can be more thoroughly studied than during life, because available both before and after section. The submaxillary gland is often enlarged, and although it seldom forms an abscess such as occurs in strangles, we may, on section, find small collections of caseous pus. The more common changes in the gland are softening and distension of the lobules by effusion within them, a yellowish exudate in the connective tissue around them, and one or more grey spots in the lobule when incised. Lymphatic lobules vary in colour in healthy horses. They are yellowish-white, reddish, or sometimes black; but whatever the colour, it is uniform throughout. Whity-grey centres in the lobules are the lesions of glanders. The bronchial and thoracic lymphatic glands are often the seat of lesions in glanders, but not so often as when the lungs are affected by tuberculosis. The mesenteric glands are seldom the seat of any specific lesion in cases of natural infection. Glanders, experimentally produced by ingestion of pure cultures, has caused mesenteric lesions, but it must be remembered that the culture used contained probably a hundred times as many active bacilli as would be found in a similar quantity of the most virulent natural infective material.

Muscular Lesions are found in man in the form of large intramuscular abscesses. I have never seen such changes in the horse, nor do I recognise any other muscular lesion in this animal.

Joint Lesions.—Not often does glanders in horses cause any change in joints which can be detected after death. I have seen hocks and knees during life show synovial distension without any signs of acute pain. In man, joints frequently are enlarged and painful in acute glanders, and are sometimes the seat of purulent synovitis. Such a condition in the horse, I believe, is unknown.

Cutaneous Lesions are tumours, abscesses, ulcers, and inflamed lymphatic vessels. The larger tumours I shall describe in the section on symptoms. The smaller, known as farcy buds, occur singly or in chains. Appearing first as small, round, hard tumours, they soon soften, the top gives way, allowing the escape of a dirty blood-stained pus or of an oily fluid, and the base remains, showing an angry red surface, with little tendency to heal. The lymphatic vessels around this " bud " become inflamed and swollen, along their course other buds form, and so we have the familiar chain of buds connected by enlarged lymph vessels—the so-called " cords." When the cutaneous form of glanders is permitted to exist, a very large area may soon be covered with " buds," ulcers, and " cords." In some chronic cases a bud or two make their appearance, enlarge slowly, and then as slowly decrease and disappear. When the skin is removed from a part showing cutaneous lesions, a condition is often revealed quite disproportionate to the mild changes seen on the surface during life. The lymphatic vessels have become purulent sinuses; subcutaneous abscesses, sometimes the size of a man's hand, are found, and a large area may be infiltrated with straw-coloured effusion.

All these different lesions may be found in one case, but not often. Sometimes we find only one or two, sometimes a number; but the association of lesions apparently follows no rule, and the results of a post-mortem examination can never be anticipated with any certainty.

The development of glanders lesions is most irregular in cases of natural infection. Some horses that we know have been long infected show very few lesions on a post-mortem examination; others with a short period of infection are found after death to present extensive morbid changes. Doubtless one cause of this variation is the dose of bacilli received at the time of infection. One horse may be infected by one dose of virus contained in one feed or one pail of contaminated water; another may for many days be stabled alongside an infected animal, and thus ingest a number of small but repeated doses. Then there is the state of the horse to remember—his individual resisting power or his susceptibility. One may be infected and live a year or more without a clinical sign of infection showing; another dies of generalised lesions in a few weeks.

It is, therefore, almost impossible to form an exact opinion, even after a post-mortem examination, on the question of the *duration* of the disease. In only a few cases where an autopsy is possible do we know the exact date of infection; and unless this is known, it is impossible to arrive at a sound basis for any definite conclusions. Without sufficient data, but with a number of cases in view in which a post-mortem has been obtained, and in which the time of infection has been fixed to a week or two, I have ventured to form some opinions. When the lung lesions are only hæmorrhagic nodules, they are not more than a month old; when nodules are caseous, they may be six months; and when calcareous, probably twelve months old. When nodules of all stages exist, I fix the duration by the state of the oldest. In cases where acute glanders has existed for some days, or in which infected horses have been continuously overworked or underfed, I think it probable that my estimates of duration would be useless, and that the changes in the lungs would be much more rapid up to the caseous stage. A similar difficulty occurs in fixing the age of ulcerating lesions. In some horses extensive changes occur in a day or two; in others ulceration goes on for a week or two, and then is limited to patches.

In the majority of cases it is very rash to express an opinion as to the age of glanders lesions.

Very little assistance is given the clinical observer by the experiments of pathologists, who use pure cultures as infecting media. I suppose that a fluid drachm of a pure culture, or a scraping from a growth on potato, might contain as many active bacilli as ½ pint of any natural infective discharge. The result is that these artificial infections act much more rapidly and with tenfold more violence than any natural infective material.

Nocard, Schütz, and McFadyean have experimentally produced glanders by causing horses to swallow pure cultures of the bacillus. In some of Nocard's cases clinical symptoms were evident on the eighth day, and mallein gave a reaction on the seventh. Horses killed fifteen days after infection were found by Nocard to have their lungs full of nodules in all stages. Horses killed three months after infection had their lungs literally crammed with nodules—fibrous, calcareous, and caseous.

Schütz made post-mortem examinations on the fourteenth, fifteenth, and twenty-fifth days after infection, and found nodules in the lungs. He also found what Nocard did not describe—enlarged mesenteric glands, nodules in the spleen and intestines, enlarged abdominal lymphatic glands, and distended chyle vessels. Nocard found calcareous nodules in a case only three months after infection. These findings are certainly not in accordance with the lesions seen in natural infections. I think I may say positively that I have never seen calcareous changes in lesions less than six months old.

McFadyean infected four ponies by administering scrapings from potato cultures in such a manner as to avoid any method of infection save that of ingestion. In each case glanders nodules were found in the lungs, but only in one were distinct lesions found in the mesenteric glands.

These experiments afford positive evidence that infection by ingestion of glanderous material may produce nodules in the lungs in from eight to twenty days, and cause no macroscopic lesion in the abdominal organs, through which the bacilli must have passed. In one case mesenteric and intestinal lesions were caused, and probably were noticeable owing to the short time which elapsed between infection and the post-mortem examination.

Incidentally, another fact was disclosed in these experiments—that each case, except the first, reacted to an injection of mallein on the thirteenth day after infection.

SYMPTOMS.

Although there are cases in which the symptoms of glanders may well be described as acute, and others in which they may be called chronic, the difference is only one of degree, dependent upon the activity of the bacilli or the resisting power of the horse. If we divided the disease into acute and chronic, we should have to add another division—subacute—which would probably include more of the cases seen in practice than the other two forms added together. I do not, therefore, propose to give separate descriptions of the two conditions. Again, glanders may appear in the cutaneous form or in the nasal form, but it is superfluous and erroneous to divide a description of symptoms into those of glanders and of farcy. In many cases the symptoms are mixed, but the cutaneous form seldom or never kills without the advent of other lesions.

Loss of energy may appear without any apparent cause, and without loss of appetite. It may be temporary, but recurrent. Probably it is only noticed by the driver, who complains that a previously energetic horse has now to be encouraged with the whip.

Cough, short, hollow, and provoked by exertion, should suggest inquiry for some definite cause.

Grunting, when the horse is turned round or when threatened, is usually a symptom of "roaring," but it is exhibited in most chest affections of a painful nature. When detected in a horse which is not feverish and not a "roarer," and when accompanied by signs of pain on pressure in the intercostal spaces, it is suspicious.

Polyuria—excessive secretion of urine—is another symptom seen frequently in advance of the generalisation of glanders. It is also sometimes noticed in tuberculosis. When only one horse in a stud shows this symptom, it may be looked upon as suspicious.

The Temperature, as indicated by a clinical thermometer, is very indicative. Whenever a horse with apparently no definite disease shows a fluctuating temperature, rising to 102·5° F., and falling a degree or two, only to rise again, glanders or tuberculosis may be suspected. In some cases the temperature remains pretty constantly at 102° F., with occasional rises to 103° F. Such horses may work, and give little other indication of anything abnormal, but they should be put under close observation and rested. We now test such horses with mallein, but in the days when that agent was unknown a constant use of the clinical thermometer in an infected stud was a useful but tedious method of picking out and isolating horses, which were then carefully watched. Many cases in time showed development of other symptoms, which proved the value of the thermometric index.

In clinically visible glanders there is always a rise of temperature, and in acute cases it may reach 107° F., when, of course, constitutional disturbance is also well marked. In chronic cases the temperature varies a great deal, and may not rise over 102° F., but as a rule it is higher. The temperature does not seem to be proportionate either to the extent of glanderous lesions in the lungs or to the amount of ulcerative change in the air-passages, although, of course, when this is excessive, a rise of temperature always exists. I incline to think that the fever in glanders depends upon the escape from open lesions of the products of the causal organism, and that when the bacilli infect the blood-stream the temperature rises; when the lesions are closed, no escape of bacilli or their products takes place, and the temperature is not affected.

Coming to the more definite indications of glanders, we have a long list of separate symptoms, which may appear singly or in numbers. No one symptom can be said to be always the pioneer. Sometimes one, sometimes another, ushers in the disease, and therefore the order in which they are described is merely a matter of convenience.

Constitutional Disturbance, as evidenced by fever, loss of appetite, and some quickening of circulatory and respiratory movements, usually attends the onset of glanders. It may be temporary and be overlooked, and it may not appear till the arrival of other symptoms. This is the stage at which errors are often made, and a diagnosis of common cold or catarrh may distract attention from the real mischief. Such an error is easily made, and when no more definite symptom is visible we cannot blame the practitioner. Any slight constitutional disturbance accompanied by a nasal discharge should be suspected, unless some definite disease is recognised. When in doubt, the mallein test should be applied.

Glandular Enlargements.—The most frequent glandular enlargement is that of the submaxillary lymphatic glands. As a rule, the gland on only one side is affected, but in a few cases both are enlarged. A very slight thickening is the first stage, and in some chronic cases nothing more is noticeable for weeks. The gland may reach the size of half an apple. It is hard, not painful, and very seldom suppurates. Usually it is adherent to the jaw-bone, and accompanied by little or no surrounding œdema. The enlargement of the gland may be the only symptom of glanders, and may remain without complication for a long time. It may be accompanied by an intermittent nasal discharge, which is noticeable during work, but ceases shortly after the horse has returned to his stable. The enlarged maxillary gland is very persistent, but it may entirely subside. In cases where a gland has once been enlarged and resumed its normal aspect, it may become a very prominent hard swelling in the short space of a day, or at most two, but is then always accompanied by some marked development of the disease—by acute generalisation of an old latent infection.

In Great Britain we have very few cases of glanders in entire horses. In countries where castration is not so general the testicles are often affected, showing swelling and pain on pressure. It should be remembered that tuberculosis may cause a similar condition. An undiagnosable lameness has occasionally been the first symptom noticed in a horse that later has developed glanders, and a post-mortem examination has disclosed specific disease in the inguinal or in the brachial glands. Such cases are rare. When glanders was not so specially cared for as now, in old cases which had been temporarily cured (?) and had again developed, enlargement of the prepectoral glands was occasionally met with. They could be felt in front of the border of the scapula. In cases of farcy with enormous swelling of a hind-leg the inguinal glands are usually affected.

Nasal Discharge is a very frequent symptom of glanders. In nearly all but the acute cases the discharge is from one nostril. In acute cases the discharge may be copious and distinctly purulent in appearance, but more often it is muco-purulent, of a dirty grey colour and a viscid nature. It may be streaked with blood. In chronic cases with a one-sided discharge the matter is thinner, sticky, and less in quantity. It adheres to the edges of the nasal opening, and is often accompanied by some swelling and hardening of the skin covering the nares. The nasal discharge in glanders is not proportionate to the amount of ulceration of the nasal membrane, sometimes being slight when deep ulceration exists, and at other times copious, with very few ulcerating lesions. The discharge may be intermittent, and only seen after exercise or exertion. As a rule, no offensive smell accompanies the nasal discharge in glanders.

Nasal Nodules and Ulceration.—Very seldom is nasal ulceration seen in any other disease of horses but glanders. It may occur. When it does, the ulcerating surface is raised and prominent, accompanied by very little discharge, and no constitutional symptoms are noticeable. Of course, all such cases should be tested with mallein.

Glanderous ulceration may exist high up on the septum, or on the turbinated bones, and cannot be seen during life. When the ulceration is visible, we usually find it lower down on the septum, or within the fold of the mucous membrane which covers the anterior portion of the cartilage forming the alæ nasi. White firm nodules are sometimes found on the nasal membrane, but they are not common. They differ in size from that of a pin's-head to a small pea. The most common lesion forming the first stage of ulceration is a small white vesicle, which does not persist, but in about twenty-four hours breaks, leaving a red sore, which forms a rapidly progressing ulcer. This ulcer is round, with dark red circumference, a depressed centre, and clean-cut raised border. When near the entrance of the nose, the discharge from the ulcer often forms a soft scab over it, which is easily detached. As a rule, more than one ulcerating spot makes its appearance, and when two or more are near they soon coalesce, forming an angry-looking ulcerating patch. In the worst cases the whole mucous membrane of the nasal passages may be one continuous ulcerating surface, and perforation of the septum has frequently been seen.

Although the early ulcerations visible on the nasal membrane are rightly described as depressed ulcers with raised borders, those higher up the cavity, or which have existed for some days, are frequently prominent and almost fungous-looking objects.

Nasal Infiltration.—In some cases of glanders—frequently in the so-called "farcy" cases—we find no ulceration in the nasal cavity, but a swollen and discoloured membrane of a bluey-black colour. The veins and lymphatics are distended and prominent, but the mucous membrane is unbroken.

This blue-black condition of the membrane is also seen in hard-worked old horses, but to a less degree, and therefore is not to be accepted as an indication of infection, without other symptoms. It is, however, sufficiently suggestive to warrant a mallein test.

Snuffling and Snoring.—As a result of nasal ulceration and œdema of the mucous membrane of the nose, the passage of air in respiration is obstructed, and we have snuffling and snoring as well-marked symptoms of glanders. These conditions exist sometimes when very little can be detected by an examination of the nose in a living horse. The thickening and ulceration of membrane are too high up in the head to be visible, but their effects are most noticeable. Usually ulceration of the septum can be seen when these sounds are emitted by a glandered animal.

Roaring.—When a horse has recently suffered from strangles or influenza, no surprise is felt at the emission of a "roaring" sound; but when a horse in regular work commences to "roar" suddenly, we are apt to suppose some traumatic cause to be in operation. This may be the very first noticeable symptom of the development of glanders. In every case of sudden roaring, so far as my experience goes, the horse has been the victim of latent glanders, and the roaring arises from ulceration of the larynx. Ulceration of the trachea may exist without a symptom to suggest its presence. Even ulceration of some parts of the larynx may cause no roaring, but when the lesions are on the anterior portions—on the vocal cords, the free border of the arytenoids, or on the epiglottis—roaring results. Such a symptom occurring in an infected stud is most suggestive, and wherever it happens without an apparent cause the mallein test should be resorted to. An error in diagnosis may induce a practitioner to resort to tracheotomy; this is attended by some danger to the operator.

Œdema of Limbs.—In those cases of glanders which are first made evident by the appearance of lesions on the skin, swelling of the limbs is a common symptom. We may have a localised swelling on the arm or thigh, resembling a bruise. There is no history of injury and no sign of abrasion. The swelling is firm, irregular in shape, and slowly spreads.

After a day or two, one or more small tumours or buds appear, and these rapidly suppurate.

A general œdema of the limbs often accompanies the development of glanders, as it does other debilitating conditions. "His legs are filled," say the stablemen. This slight dropsical effusion is most noticeable below the knees and hocks, and may not be accompanied by any more specific symptom. In many cases, however, small spots become prominent and break, discharging a viscid fluid, which mats together the hair, and, running down the limb, leaves a track of dried brownish material, not unlike varnish, firmly adherent to the hair.

In other cases œdema of the limbs is much more marked. As a rule only one leg is affected, and the swelling is extensive, rapid, and painful. We have all the appearances of an acute lymphangitis, extending high up into the arm or thigh. Not until some other lesions appear can we recognise this œdema as due to glanders. As a rule, we soon have development of "buds" on the inside of the limbs, which give way on the surface and allow of a discharge. One bud is followed by another along the course of the lymphatic vessels, so that in time one finds a series of lesions of different ages—some unbroken, some suppurating, some deep ulcers, and some healed or partially healed sores, with a few cicatrices.

The mallein test enables us to diagnose these cases in the earlier stages, and the various developments of the "farcy buds" are themselves quite indicative. It is necessary to remember that lymphangitis commences in the same manner, that epizoötic lymphangitis may closely resemble the state I have tried to describe, and that another disease—ulcerative lymphangitis—is sometimes only to be differentiated by failure to react to mallein.

Tumours.—There are two kinds of swellings seen in cutaneous glanders—the well-recognised farcy bud and a larger tumour. These larger tumours vary in size from 1½ to 3 inches in diameter; they show no tendency to suppurate, and are very persistent. I have seen them remain for weeks, and, if the horse be permitted to live, they gradually decrease and finally disappear. If opened, their contents are found semi-gelatinous, and of a straw colour, rarely purulent. Their most common site is on the back and sides, less frequently over the gluteal region. They are round, and as prominent as would be the third of an orange laid on a flat surface. We seldom find more than one or two at a time on any horse, and they are usually widely separated from each other. It is worthy of note that these large tumours are never accompanied by any enlargement of the lymphatic vessels in their neighbourhood, whereas the ordinary " bud " is seldom seen without its accompanying " cord."

The ordinary tumours—viz., the farcy buds—seldom exceed an inch in diameter. They may appear anywhere, but are most frequently seen on the face, neck, sides, and down the inner side of the limbs. The appearance of one is usually rapidly followed by others, arranged in a row, and connected by a linear enlargement, due to distension of lymphatic vessels, known as a " cord." These " buds " are first hard and prominent; they rapidly soften, the skin gives way, suppuration takes place, and an angry ulcerating surface with a depressed centre and thickened margin is left. The discharge is almost oily in character, and dries into adherent masses on the hair below the sore.

In a chain of farcy buds every stage of the lesion may be seen—a partially healed bud marking the place first affected, then one just broken, then one softening, and two or three more, diminishing in size, which are still hard. On the fine skin of the face they seldom exceed ½ inch in diameter, and when the ulceration stage is reached, they show very clearly the deep sunken condition, with thickened margins, which is most characteristic of sores in glanders.

Hard Swellings, irregular in shape and varying in size, are not uncommon in the early stage of cutaneous glanders. They appear on the limbs above the hocks and knees, on the sides, and on the sternum and abdomen. They vary in size, often reaching a measurement of 8 or 10 inches in length, and resemble a bruise rather than the enlargements of purpura.

Swelling of Joints.—It is not common to find swollen joints in horses suffering from glanders, but in a few cases we have observed the rapid development of enlargements of knees and hocks, the latter joint presenting a well-marked bog-spavin. Specific inflammation of bursæ is also sometimes seen. I remember one case, in the days when farcy was not doomed to slaughter, in which bog-spavins and wind-galls were very prominent, and caused some stiffness for a week or two. The horse afterwards worked for a year, and these lesions entirely disappeared, but glanderous pleurisy ultimately killed him.

Subacute Glanders is the most common form of the disease seen in London. The temperature ranges from 102° to 104° F. The appetite is not lost, and the horse may remain at work without showing ill-effects other than fatigue and bodily wasting. These symptoms are often accompanied by an enlarged submaxillary gland, and later by a nasal discharge and visible ulceration.

Acute Glanders is the term applied to cases in which the disease is generalised, and the blood-stream invaded by the bacilli. The temperature is 105° F., or higher; appetite is lost; respiration is rapid and accompanied by snuffling or roaring; the pulse is much accelerated; a nasal discharge is seen from both nostrils, often tinged with blood; and a cutaneous eruption or swelled legs may also appear. The horse dies in a few days from septicæmia and pneumonia.

Chronic Glanders is the form in which some symptoms, such as an enlarged gland and slight nasal discharge, exist without much rise of temperature. These cases are now rare, but were common when compulsory slaughter was not enforced. They might develop gradually from a latent case, but more often followed a subacute attack, which had subsided after rest and treatment, in a horse of extra strong constitution. Symptoms abated for a time, but few horses survived many months, and any violent upset to the general system, such as a dose of physic, excessive work, or the supervention of any acute disease, ended in rapid generalisation and death. These horses in olden times were separated from the stud and worked together. In double harness the pole of the vehicle was painted black, so that those who knew might be warned off. White states that he has known chronic glandered horses to work for two years before being killed.

Chronic cases of the cutaneous form were formerly not infrequent. A horse with a thick leg and a few old scars resulting from an acute attack of farcy might work for weeks or months without much alteration. In cases favourably treated all the symptoms might disappear, but the rule was for the submaxillary gland to remain enlarged and hard. In time the marked limb suffered again from an acute lymphangitis. Finally, the temperature rose, nasal symptoms appeared, and death followed.

Well-marked cases of farcy sometimes show no pulmonary nodules at the autopsy, and frequently only two or three recent typical hæmorrhagic centres. The greater number, however, are found to have nodules in the lungs of a form and structure which demonstrate that they have been in existence for months anterior to the cutaneous symptoms.

Farcy lesions are nearly always secondary to disease in the lungs, and result from the escape of bacilli from pulmonary nodules, and their passage through lymph and bloodvessels to the skin.

DIAGNOSIS.

When a horse presents a nasal discharge, nasal ulceration, and an enlarged submaxillary gland, diagnosis is easy. A swollen leg and a typical chain of farcy buds is another form of the disease about which no doubt need exist. But there are many cases of glanders in which the symptoms are not well marked, and there are some other diseases which at times closely resemble glanders. There are also cases in which the symptoms of glanders may be masked by the morbid changes due to other maladies.

An Enlarged Submaxillary Gland may exist for some time before any other sign is apparent. This symptom may also arise from disease of teeth or sinuses, but usually is then accompanied by a unilateral nasal discharge with an offensive smell. An enlarged gland may be due to melanosis, which only occurs in grey horses, and is attended by no nasal discharge, but often by tumours in other parts of the body. Another rare cause of enlarged submaxillary gland is tuberculosis. In some unusual cases of glanders the submaxillary gland suppurates, and the condition may be mistaken for strangles, but the slow development of these cases should cause their true nature to be suspected. An injection of mallein assists diagnosis, and should be resorted to at once on the appearance of glandular enlargement.

Strangles, Catarrh, and Influenza very often show a purulent nasal discharge, which has been confidently credited as peculiar to those diseases. It is not sufficient to observe the character of the discharge. Many cases of glanders commence with a purulent or semi-purulent discharge not distinguishable from that of other diseases. Some cases of latent glanders are wakened into activity by an attack of catarrhal fever. These are the cases which led to the error of supposing that "influenza turned to glanders." In young horses a nasal discharge may usually be accepted as innocent, but from an old horse should be treated with care. Newly-purchased horses from a town stud should always be suspected if they exhibit a nasal discharge, and the mallein test should be applied.

Nasal Ulceration is almost diagnostic of glanders, but slaughter should not be adopted until mallein has been used, unless the character of the ulceration or the presence of other symptoms are such as to leave no doubt.

Nettle-Rash has been mistaken for cutaneous glanders. Although the swellings at first somewhat resemble farcy buds, their sudden appearance, their general prevalence over the body, and their tendency to coalesce, should be sufficient to found a diagnosis upon. The nettle-rash lumps disappear as rapidly as they make their appearance. Only once have I seen a case of acute glanders with the skin covered with lumps about the size of walnuts; they were all about one size, and discrete. The horse was killed on other symptoms, and I considered the case one in which the two diseases coexisted.

Purpura Hæmorrhagica has often been mistaken for glanders. When the head is badly affected, we find swollen nostrils, a blood-stained septum, and a sanguineous discharge from the nose. These conditions may mask a case of latent glanders which has been generalised by the purpura. It is extremely difficult to open the swollen nostril in these horses sufficiently to see the septum. The swelling of the limbs may be mistaken for farcy of the legs, but as a rule farcy appears only in one leg, whereas the swellings of the purpura are in two, or in all four, and in size and position bilaterally symmetrical. At the commencement of a case of purpura dark spots upon the septum nasi may suggest the use of mallein. The temperature is always raised—often high—so is useless as a reaction symptom. In almost every case of purpura, swelling at the point of injection is considerable, but as it rises in twenty-four hours, so it disappears in the next twenty-four, and its rapid subsidence indicates freedom from glanders.

Laryngeal Disease may cause roaring in horses, and occasionally this symptom is the first to show itself in glanders. When roaring comes on suddenly, suspicion should be aroused, and the fact of its disappearance and recurrence should not permit a diagnosis of non-specific disease. The sudden access of roaring without any clear cause should be followed by an injection of mallein, especially in town horses or those newly purchased.

Ulcerative Lymphangitis.—There is a form of lymphangitis which may be mistaken for farcy. In addition to the general dropsical enlargement of the limb, we have a subcutaneous collection of pus, causing sloughing of a comparatively large portion of skin inside the thigh. Then the lymphatic vessels become enlarged and suppurate, and smaller abscesses form around and below the original lesion. These small abscesses are often very similar in appearance to farcy buds, but the larger sloughs almost differentiate the two diseases. An injection of mallein affords the most reliable aid to diagnosis.

Horse-Pox.—Some cases of horse-pox very closely resemble glanders on a superficial examination. They may have as symptoms a nasal discharge, enlarged maxillary glands, and small ulcers on the fine skin of the nose and lips. But there are other lesions not found in glanders—viz., vesicles, which soon form ulcers, on the mucous membrane of the lips. These ulcers are not like the malignant ulcers of glanders; they are not so deep, show no tendency to spread, and soon heal.

What is called **Stomatitis Pustulosa Contagiosa** is, I believe, only horse-pox (equine variola) under another name. When stomatitis happens to accompany lesions in the heel and back of the fetlock, no one disputes that the case is one of variola, especially if depilation follows, and the legs lose most of their hair. Stomatitis confined to the lips is communicable to man, and in two or three cases I have seen, the human lesion from accidental inoculation was typically variolous.

Botryomycosis.—Not often can this disease be mistaken for glanders. In one case in which some similarity existed there were two tumours on the inside of the thigh of the horse—one the size of a walnut, the other as large as a Tangerine orange. There was also an irregular, hard swelling on the antero-inferior aspect of the sternum. The tumours had been present for some weeks, and had suppurated. The swelling on the breast had just appeared, and this led to doubt of the nature of the case. Mallein was injected, and gave no reaction. Afterwards Sir John McFadyean cleared up the mystery by discovering the botryomyces.

Epizoötic Lymphangitis is now a non-existent disease in Great Britain, but in some countries it is prevalent. There are cases of the disease which only the most experienced practitioners can distinguish from the cutaneous forms of glanders. The mallein test should be resorted to at once, and the negative results should be followed by a microscopic examination of the pus from the sores, so as to obtain a positive diagnosis. This is reached by the discovery of the lemon-shaped causal organisms of epizoötic lymphangitis.

Aids to Diagnosis.

There are many methods suggested for verifying a diagnosis of glanders. The microscope may be used to show the bacilli in the pus or juices of a suspected lesion. If a man is expert in using the microscope and in staining preparations, he may identify the organism, but in the majority of cases a practitioner would be well advised to leave this method to the trained bacteriologist.

The agglutination test is another means of assisting diagnosis which is only useful in the hands of an expert.

Inoculation of other animals, such as horses, asses, or guinea-pigs, may also be adopted, but it is expensive, and requires time for development. Inoculation into a susceptible animal gives positive demonstration if active bacilli are present in the material used for injection. The discharges from any visible lesion are, however, mostly contaminated by mixed organisms, and sometimes wanting in active glanders bacilli. Inoculation of pus from the nose into the larger animals has often failed to give a definite result; and even separate nodules from one pair of glandered lungs which appeared morphologically alike have given some negative and some positive results when singly inoculated into guinea-pigs

Cultivations of the bacilli are not easily carried out unless a tolerably pure material can be obtained. The discharge from the nose, or from an ulcer on the skin, contains so many other organisms that it is useless for culture purposes. When an unopened farcy bud exists, material can be obtained by incision which, in skilled hands, may afford good results from cultivation on sterilised potato.

All these aids to diagnosis are of use in their degree, but they can only be carried out by a bacteriologist in a laboratory. The general practitioner will find injection of mallein quite as trustworthy, much simpler, and more prompt in its indications.

Mallein was discovered in 1891 by Kalning and Helman, two Russian veterinarians. It is obtained by growing the *Bacillus mallei* for some weeks in a suitable medium and at a definite temperature. The resulting virulent culture is then heated in an autoclave, to kill all the organisms, and filtered through a porcelain filter to remove the dead bacilli. To the material thus obtained is added glycerine and a solution of carbolic acid.

Mallein so prepared is a transparent fluid of a pale yellowish-brown colour. If injected under the skin of a healthy horse in small doses, it produces no symptoms, save, perhaps, a swelling the size of half a walnut at the spot of injection. This swelling, which soon disappears, may be due to a dirty skin or a dirty needle, to mallein which has been allowed to become dirty, or even to too large and blunt a needle. When injected into a horse suffering from glanders, two effects result: *a rise of the general temperature* and a *swelling at the seat of injection*, which together constitute a specific reaction. This double reaction renders mallein more useful than tuberculin as a diagnostic agent, because in many cases of suspected glanders and tuberculosis an abnormally high temperature is present. The elevated temperature in the tuberculosis case renders tuberculin useless, but, with a high temperature in glanders, we may still obtain some indication from the presence or absence of a local swelling. It is inadvisable to use either agent where the temperature is abnormal; but we may have to wait days for the temperature to reach the normal, and when diagnosis is urgent, mallein may be employed with hope of some guidance.

Mallein is injected under the skin in doses of 20 drops or less, by means of a hypodermic syringe. Any part of the skin may be selected, but the best is the side of the neck, about halfway from the ear to the shoulder Injection over the shoulder or ribs results in a needlessly painful swelling. Injection into the skin of a leg is followed by great lameness, if a reaction takes place; and injection into the loose skin in front of the stifle causes a swelling, easily felt, but not very visible. The neck as the site for injection has other points in its favour—the skin is easily lifted into a fold, the operator is in a good position if the horse is bad-tempered, and the swelling of a reaction is well thrown up by the firm tissues underneath.

Most of the needles supplied with veterinary hypodermic syringes are too long and too thick. The finer the needle, the more easily is it inserted, and the less the horse resents it. There are two forms of junction between needle and syringe—the screw and the plug. The screw is objectionable, as the needle can only be used with the syringe attached, and when the horse is fidgety or spiteful the operator may have his choice between leaving the syringe hanging from the neck by the needle, or keeping his hold and being injured. The detached needle is easily inserted, and remains in place if it has to be left; whilst the syringe with the plug junction is used with the greatest facility.

There are certain precautions which should be observed in the application of a mallein test. The first is to take the temperature of the animal before an injection is made. In a working stud of fifty horses it is quite usual to find a horse, apparently well, with a temperature of 104° F., and as this may be the commencement of an illness which would lead to even a higher temperature, that animal is left over for a further test. When the injection is made without taking the temperatures, such cases are only discovered when the reaction is looked for, and the error is unavoidable of considering the rise a sign of general reaction, whereas it has nothing to do with the mallein.

Of course, the syringe used should be surgically clean, and the mallein should be clear and without sediment. How long mallein will keep good and active is not, perhaps, known; kept under proper conditions, certainly six months, and probably a year. But it is best to obtain fresh supplies, and these can always be in amount just sufficient to cover probable demands. Directions to wash and disinfect the skin of the neck before injecting are usually given and obeyed in practice. I consider this a waste of time, and my experience is that if the skin be dry the results are invariably good.

When the temperature of a horse is normal, and a subcutaneous injection of mallein is made, we have a double reaction as an indication that he is infected with glanders. We have a rise of temperature of 2° or 3°, or more, and we have a local swelling at the site of injection 4 inches or more in diameter.

The rise of temperature commences about seven hours after the injection is made, and continues to increase up to about the sixteenth hour. If the horse be infected, the temperature may remain high for some hours longer, and in a few cases for some days.

The following is a typical reaction :

		Temperature.	Diameter of Local Swelling.
At	8 p.m. (time of injection)	100° F.	
,,	8 a.m. (12 hours after injection)	104° ,,	3 inches.
,,	12 noon (16 ,, ,,)	105° ,,	4 ,,
,,	4 p.m. (20 ,, ,,)	105° ,,	5 ,,
,,	8 p.m. (24 ,, ,,)	104° ,,	6 ,,

The local swelling develops gradually, and, when specific, increases in size for over the twenty-four hours following injection. Very little value attaches to the vertical measurement of a swelling, which results from the effusion gravitating. The typical swelling is round, raised well above the surrounding skin, painful on pressure, but, above all, one which increases in diameter after the twenty-fourth hour.

Although I accept a mallein reaction as specific of glanders, I must acknowledge that an ideal double reaction is not always given. Sometimes the rise of temperature is less than we consider indicative, whilst the swelling is typical. At others the swelling is too little to offer any guidance, whilst the temperature is typically high.

A temperature rising from normal to 105° F., with a swelling 6 inches in diameter, is an indication of glanders upon which slaughter may be safely advised. In an infected stud I should also accept a temperature of 104° F. with a 4-inch swelling which increased or remained after the twenty-fourth hour, as signs of infection ; so should I regard a temperature of 103° F. with a 6-inch swelling, or a temperature of 105° F. with a 3-inch painful swelling.

There are imperfect reactions which render it difficult to draw a hard-and-fast line as to when a reaction may be positively accepted as indicative of glanders, and these have a very practical bearing now that the law recognises reaction to mallein as equivalent to "diseased."

As the exact time after injection at which the double reaction reaches its maximum is not definite, it becomes important to know when the horse should be examined, his temperature taken, and the swelling measured. I do not think any useful information is missed by not visiting the horse after injection until sixteen hours have elapsed. In a few cases the temperature may rise to 105° F. by the sixteenth hour, and fall to 104° F. by the twenty-fourth, but that register is quite sufficient if the swelling is indicative ; and as the swelling in an affected animal goes on increasing after the twenty-fourth hour, it is advisable not to make the concluding visit too early. There are very few definite reactions which need for their recognition more than one visit after injection, and that should be made on the twenty-fourth hour. To read the double reaction of mallein most accurately—to recognise the maximum temperature and see the full quantity and quality of the swelling—we must take the temperature at the *time of injection*, again at the *sixteenth* hour, and again when we examine the swelling at or just after the *twenty-fourth* hour. The really indefinite reactions—temperature under 103° F., and swelling less than 4 inches—must be tested again, and this may best be done after a period of two or three weeks. I fix this time as the best, but I have seen perfect reactions when the injection has been repeated in a week. In cases which have reacted to mallein, a second test often shows a typical swelling, but a reduced rise in temperature. Here the swelling may be accepted as indicating disease, even if the temperature is only 103° F., or even a degree less. There are a few exceptional cases in which a rise of temperature occurs within twenty-four hours, with no swelling, but in the next twenty-four hours a typical swelling develops. These cases are infected, and the possibility of this delayed local reaction should make us careful not to pronounce a horse free from glanders, if a rise of temperature has occurred, before the lapse of forty-eight hours.

It is inadvisable to make a mallein test of a horse with a temperature over 102·5° F. In cases where diagnosis is urgent, and we test an animal with a temperature of 103° F. or higher, a *fall* of 2° in twenty-four hours is a suspicious sign which may be considered in conjunction with the local swelling.

I have seen some reactions to mallein which were apparently misleading. I have heard of others from good observers. These results force one to acknowledge that the use of mallein is only an aid to diagnosis. There are cases in which mallein has failed to reveal any specific lesions. There are also cases in which mallein has failed to give a reaction, whilst a post-mortem examination has disclosed definite glanders lesions. These misleading results are few and far between.

When a stud of horses that has been infected for some time is tested with mallein, we expect to have a number of animals give a reaction, and a few to show an indefinite reaction. These latter are retested in ten to fourteen days, and those that react are killed. After this it is supposed that the stud is clear, but it is not uncommon to find another case or two to develop clinically in the course of two or three months.

The probable explanation of this is that the disease must reach a certain stage before it is indicated by mallein. Nocard infected a horse by ingestion of glanders bacilli, and injected mallein on the seventh, fourteenth, and twenty-first days after. No reaction was given until the third test was made. If this experiment may be accepted as generally applicable, horses infected seven days before being injected with mallein would give no indication of their infection. In some horses experimentally infected by Sir J. McFadyean, a reaction to mallein was given thirteen days afterwards. We should probably be certain to detect all infected by repeating the test in any stud after the lapse of fourteen days. In a stud from which a number of clinical cases has recently been removed, the possibility of these undiscovered infections warns us that a retest of the whole should be adopted if we desire to be quite certain that no disease remains.

A safe rule to adopt is: When in doubt, repeat the test. No one asserts that mallein is infallible. It is to be accepted as an aid to diagnosis, and its reactions considered with the history and surroundings of the horse. But it is an aid far more reliable than any other, more trustworthy than any expert opinion, and indicative when no trace or sign of disease can be detected by the most careful clinical examination.

PREVENTION.

The prevention of glanders requires intelligent action upon the part of horse-owners with reference to their premises and their studs. It also requires well-considered regulations to be enforced by all local authorities, and supervision over the whole by a central body, such as the Board of Agriculture.

In Great Britain the Diseases of Animals Acts are administered by a central authority—the Board of Agriculture—assisted by local authorities. The owners of stock and their private veterinary advisers also play an important part in co-operating, or otherwise, with the authorities.

The discovery of disease is a matter antecedent to all preventive action, and the owners or their veterinary surgeons must be trusted to make this discovery first, and then to notify their suspicions. The prevention of all the scheduled diseases, as well as those not yet scheduled, must commence in the stable, field, or shed.

It is apparent, then, that, so long as glanders prevails around us the most careful owner of horses may be unable to protect himself against infection. He may introduce the disease by a new purchase suffering from the latent stage of glanders, or he may have one of his old horses infected through the mangers or pails used in another stable by a diseased horse.

The owner who has a large stud, and has to make frequent purchases to maintain it in working order, may guard himself against the introduction of disease by the new horse suffering from latent infection, by testing with mallein every new purchase soon after its arrival. No one can absolutely provide against contagion if his horses cohabit with others that are infected, and therefore it is the interest of all horse-owners to assist in stamping out the disease throughout the kingdom.

The man who buys cast horses from large studs, or who buys at sales, should always test every new purchase within twenty-four hours of receiving it into his stable. If it reacts, he can demand the return of his money when a warranty of soundness has been given, but in no other case. At the worst, he protects the rest of his horses from infection, and probably may be able to come to some compromise with the vendor. If it became a general practice for horse-buyers to test their new purchases, the custom would have a most beneficial effect in preventing the spread of disease, because owners would then hesitate to sell any horse which would, if diseased, be detected at once by the purchaser.

When a stud is already infected—and many in London have been so for fifty years—the most essential preventive action is to find out which horses are diseased. This can be done by applying the mallein test to the whole. To make doubly sure that no infected horse has escaped detection, the test should be repeated in six or eight weeks. All that react must be separated from the healthy, and slaughtered.

With a large stud stabled in widely distant depôts, the full value of testing with mallein may be lost through carelessness in changing horses from depôt to depôt. A horse moved from one stable to another without having been first tested, may carry disease and spread it widely before being detected. An oversight of this kind has been the means of reinfecting more than one stable.

Turning horses out to grass—at any rate, on farms round London—has been a fruitful source of infection. Horses from different stables run together, and there may be as many as twenty or thirty horses in one set of meadows. I knew a case in which eighteen horses were attacked with glanders within four months of being at grass. The infection was traced to one old horse not worth £10. The owners of pastures round London should admit no horses to their fields unless from a known clean stable, and after a test with mallein has been made. London horse-owners would be well advised not to turn out their horses, except with a guarantee from the farmer that no strange horses are allowed to run with them.

It is evident that whenever horses are collected together in numbers from various places, there is a chance that an animal suffering from latent glanders may be among them. The accommodation at large fairs and race-meetings favours infection spreading if a diseased horse should happen to be present. Military manœuvres have often helped to spread glanders, not by infection from the well-looked-after army horses, but by a diseased animal introduced with the mixed lot supplied by contractors.

War always spreads glanders among the troop and transport horses, but the spread does not cease with the war. Economy suggests the sale of superfluous horses, and thousands of infected animals are in this way scattered over the whole country in which the war took place. Since mallein became available for testing horses, the dissemination of glanders by sale of cast horses is no longer excusable, and I am glad to say that in South Africa very great care was taken to guard against the sale of infected animals, thousands of doses of mallein having been used to protect stock-owners who bought the superfluous horses.

One of the most important preventive measures to be taken by authorities engaged in stamping out glanders is to trace back the history of infected horses, and attempt to discover the source of their infection. The relation of a concrete case or two will make this point clear.

A glandered horse is found in a cab-yard, and the owner states that he has never before had any cases of the disease. The history of the horse is that he was purchased at a sale ten days before he was noticed to be ill. At the sale-yard the name and address of the vendor are obtained, and his stud is inspected. It consists of seventeen horses. One showing slightly suspicious symptoms is tested with mallein, reacts, and is killed. The post-mortem reveals old glanders lesions. Then the remainder of the stud is tested, and five horses react, which are found glandered on post-mortem. From the first case we were able to find six dangerous animals, and put a stop to further infection.

A dead horse is found in a knacker's yard with glanders nodules in the lungs. On going to the stable from which he came, there are seen eight horses, none of which show any clinical symptoms of glanders, but all are rather poor in condition and aged. The lot were tested, and every one reacted, their infective condition being proved by post-mortem examination.

A horse in a well-managed stud was found affected with farcy. The animal had been there two years, and no case of glanders had been seen in the stud. The history of the horse included two months at grass for lameness, and he had only resumed work about a month. The field was in another authority's district, so application was made to the veterinary inspector of the district, and he replied that he had seen four cases of glanders on the farm at which the horse had been grazing.

When the spread of infection can be traced to its source, an unnotified outbreak is often discovered; it may or may not be one known to the owner.

One of the cases I have referred to was discovered in a knacker's yard, and if these places were inspected by the veterinary surgeons of all local authorities, as they are in London, many cases of glanders would be found that now escape detection.

It has been suggested that public sales and auctions should be inspected by the official veterinary inspector. If clinically diseased horses were ever sold there, inspection would be valuable; but they are not. The horses entering every horse repository in London are examined by the auctioneers' private veterinary surgeon, and very strictly examined. The horses coming through repositories that help to spread glanders are those in which the disease is latent, and may remain latent for a long time. To inspect these and pass them would inspire many buyers with a sense of security that was more apparent than real, and when the latent disease developed shortly after purchase, the same class of buyer would feel great contempt for the inspector who had not discovered that the horse was infected.

Disinfection and cleanliness are enforced by law in all stables which have been the scene of a glanders outbreak. This application must not be confined to the mangers, walls, and floors. Nosebags, pails, and stable utensils must all be disinfected, if not destroyed. Rugs and halters, if they are worth saving, may be soaked in a solution of carbolic acid and hung to dry in the sun for a day or two. Before any disinfection is done, the place should be made as clean as possible. Mangers must be emptied and, with the stalls, walls, and bales, soaked with a few pailfuls of water overnight; then hot water and soda applied with a brush until the grain of the wood is visible. After a day or two to dry, the walls, etc., may be dressed with hot limewash. The floors and manure are best treated with dry quicklime.

Of course, no prevention of glanders is real which does not aim at stamping the disease out of the country. This means the slaughter of every case as soon as it is diagnosed. Before mallein was discovered, it would have been a long and expensive task to have cleared the country of glanders. By the use of mallein we may now clear all infected studs, but it must be remembered that the most careful use of this agent will not always indicate every infected horse by one testing.

Professor Bang long since pointed out, in the case of tuberculosis, how herds which had been tested with tuberculin, and from which all reactors had been removed, when retested a few months later disclosed a small percentage of infection which had escaped the first test. A similar experience is found with glanders, and is likely to be even more noticeable in stables from which animals that give an indefinite reaction are not removed. It is well to remember this, because the expectation of an easy and prompt clearance of disease from a large stud may lead to disappointment, and both owners and officials become disheartened and sceptical.

The stamping out of any disease is a laborious process, and mistakes are certain to occur. Every mistake may give rise to a fresh outbreak.

The Order of 1907 will, I believe, prove successful, but local authorities would do well to arrange with the owners of large studs which are supposed to have been freed from disease by mallein-testing and slaughter, for a second test of all horses a month or two after the slaughter of the last reactor.

Importation.—Considering the trouble and expense devoted to clearing our home stock, it is right that provision should be made against the introduction of glanders from other countries. The new Order imposes a very mild regulation upon horses, asses, and mules—they shall not be landed unless accompanied by a veterinary certificate to the effect that they show no symptom of disease. Experience proves that not much risk is run by importing a good class of young horse, but a few infected horses have arrived, and others may arrive. When we have cleared our own studs, the Board of Agriculture will probably enforce the use of the mallein test upon imported horses.

State legislation for the control or suppression of glanders is necessary, because the efforts of individuals can only apply to their own studs. After prolonged and expensive action, a horse-owner may be successful in stamping out the disease in his stables; but when glanders is allowed to prevail around him he is never safe, and sooner or later is almost certain to again find his stud infected.

Legislation should not be all penal. With penalties for offences it should combine rewards for assistance. It is a misfortune, not a crime, for a man to have his animals affected with glanders. To get rid of the disease, legislation must interfere with his use of horses which may be of great intrinsic value to him and of no injury to his neighbour. It must destroy horses which present no sign of disease, and which, though infected, may never become infective. To meet these cases compulsory slaughter of stock should be accompanied by monetary compensation. The argument for compensation in cases where legislation is directed, with probable success, to eradicating a disease from the country is not on the same level as a claim for trade losses due to unpreventable accidents. These accidents will occur again, no matter what care and cost is expended on them. But success in stamping out disease means the permanent freedom from the loss caused by such disease, and therefore a national loss is saved which far exceeds the national contribution in the form of compensation. Legislation must not be in advance of intelligent public opinion. If it is, there is resistance, active or passive, which renders the regulations a failure. When owners of stock acknowledge the value of legislation nearly everyone interested co-operates, and success is assured. There are, of course, always some owners who will attempt evasion, and a few who wilfully spread disease by moving or selling diseased animals. For such penal clauses are requisite. Legislation fails in its object when weak half-measures are adopted. Their want of success, with the accompaniment of hardship to owners from rigid restrictions, brings the whole of the legislative measures into disrepute.

In 1899 a Departmental Committee was appointed by the Board of Agriculture, with Lord Stanley as Chairman. It took evidence and issued recommendations. Still legislation remained the same, and it was not till 1907 that a new Order, based on the 1899 Committee's suggestions, and in accordance with modern veterinary science, came into force. The recommendations of the Committee were as follows:

1. That the Board of Agriculture should exercise a more extended supervision of the working of the Glanders or Farcy Order.
2. That the Glanders or Farcy Order should be amended to permit of notification of disease being made either to a constable or a veterinary inspector.
3. That where practicable the local veterinary inspector should not engage in private practice.
4. That it should be made obligatory for veterinary surgeons to notify cases of glanders of which they become aware.
5. That occupiers or owners of knackers' yards should notify any case of glanders found in animals taken to their yards for slaughter.
6. That horses that react to the mallein test should be considered as possible sources of infection.
7. That horses that the Veterinary Inspector may consider to have been exposed to contagion should be dealt with in the same manner as suspected horses, but with certain reservations.
8. That the slaughter of all animals showing "clinical" symptoms of glanders should be made compulsory.
9. That compensation for horses slaughtered solely on account of reaction to the mallein test should be on a higher scale than that for a "clinically" diseased horse, with a limit of £25. But in the event of no glanders lesions being found post mortem, the compensation should be the full value, with a limit of £50.

10. That the Board of Agriculture should conduct experiments with regard to the use and influence of mallein.

By 1905 the Board of Agriculture had really grasped the facts of the case, and tentatively drew a new Order. Then the Board endeavoured to obtain some financial assistance from the National Treasury, knowing that any earnest attempt to stamp out glanders would be attended by expense—greater expense than could honestly be inflicted upon local rates. The Treasury declined to assist, and in 1907 the Board issued a new Order, leaving local authorities to do the work and pay for it.

GLANDERS IN MAN.

In man glanders appears in two forms—acute and chronic—but either may be a sequel of the other. When acute glanders does not cause death in three or four weeks, the patient may linger on with the chronic form, showing itself by more or less active lesions, which sometimes heal, but more often reappear in different parts of the body. A very few cases seem to recover.

The period of incubation in man is usually short—from two to six days. There are recorded cases in which symptoms have not appeared until months after the patient has ceased to have any connection with horses, and these are probably cases in which the disease has run a latent course.

The symptoms of an acute case are high fever, headache, and pains in the limbs. These may be the only manifestations of disease for a week or more, and then a cutaneous rash appears in the form of nodules, which rapidly break down into ulcers. Frequently this rash is not visible till a day or two days before death, and the patient dies of acute septicæmia, with few or no local lesions.

I quote some reported cases showing how protean are the forms of human glanders.

Two cases were reported in the *Lancet* by Dr. Goodall:

A man, aged fifty-five, a printer, was admitted into the Eastern Fever Hospital on October 28, 1903. He had been seized with shivering and pain in the limbs on the 9th. On the 11th he was worse, and was medically attended at home till the 20th. On that date he went as out-patient to a general hospital whence he was sent as a case of typhoid fever to the Eastern Hospital. On admission he was semi-comatose. On the skin were a number of small superficial pustules, most abundant on the chest, right leg, and right forearm. There were a few on the left cheek, the back, left thigh, and left upper arm. Besides these pustules, there were several swellings of varying size in different places—one on the right frontal eminence, two on the right forearm, two on the left forearm, one on the right leg, and one on the left. The largest swelling fluctuated, the smaller did not. Some appeared to be in the subcutaneous tissue, others were distinctly in muscles. There was no discharge from the nose. On the morning of the 30th the contents of a swelling on the right leg were drawn off and microscopically examined. A bacillus resembling that of glanders was at once detected. The patient died on the 31st.

The second case was that of a stableman, who was taken ill with "pains all over him" ten days before admission. He was sent to a general hospital as a case of blood-poisoning, and then to the Eastern Hospital as one of typhoid fever. In one of the muscles of the left forearm was a nodular swelling. The left knee-joint was much swollen, very painful, and distended with fluid. The patient said that the lump in the arm had been present since February 18, and the knee-joint had been affected since the 16th. Next day an examination of fluid from the knee and arm was made, but no organisms were found. Cultivations in agar were made, and on the second day what appeared to be the *Bacillus mallei* was detected. A day or two later Drs. Lewis and Wood obtained the bacillus from the fluid of the knee-joint. The patient died delirious on March 11.

An inquest was held at Westminster on August 15 on Albert Allen, a horse-keeper in an omnibus-yard. Dr. Cope saw the patient first on June 12, when he showed symptoms of an ordinary cold, with aching of the limbs. He was treated for a rheumatic attack. On the 19th there was a nodule on the leg, and some inflammation of the limb. On July 12, other nodules appeared on the right arm, and on the 13th the man was taken to Westminster Hospital. Mr. Frank Mott saw him on the 14th, and as there was a suspicion of glanders, some pus was examined and the *Bacillus mallei* discovered. The man died August 11. An autopsy revealed eleven abscesses scattered over the arms and legs. There was pus in the knee-joint. Nothing was found in the nose, throat, larynx, or trachea; but in the lungs some small nodules about the size of a pea were discovered.

In a note published by Drs. Bullock and Twort, of the London Hospital, the first sentence is: "Glanders in man is generally believed to be uncommon, although, no doubt, the belief is partially due to the protean characters which the disease manifests clinically, and which render it difficult of diagnosis." They then give details of six cases which had come under their notice at the hospital, and of a seventh case which was of doubtful character. Four of these cases were chronic, had been treated as syphilis, and were characterised by recurring abscesses and throat ulcerations which lasted for months.

Inquest on Charles Nicholls, a horse-keeper, at Westminster, May 19, 1905: On May 1 the man complained of illness, and saw a doctor, who said he had pleurisy. Two or three days later he was taken to St. George's Hospital, where he died on the 16th. Dr. Hunt said he had diagnosed the case as rheumatic fever. Dr. Etherington, House-Physician, said the deceased on admission seemed to be suffering from rheumatic fever. The day before death it was thought to be glanders, and a post-mortem examination verified the diagnosis.

From the *Lancet* of April 1, I take the following from a report by Mr. M. J. Cromie, House-Surgeon, Westminster Hospital: A horse-keeper, aged twenty-five, was admitted on November 12, 1904. A consultation between Dr. Hebb and Mr. Spencer led to a surmise that the patient was suffering from glanders. On the following day Dr. Hebb found the *Bacillus mallei*. The patient died thirty-three hours after admission. "A full consideration of the case exposes possibilities of infection, and of such cases being overlooked," is Mr. Cromie's comment, and the history more than bears this out. On November 3 he had pain and swelling of the left side of the face, and was advised by a dentist to bathe his face in hot water, as an abscess was forming. He next saw two other medical men, who told him that he had cellulitis and erysipelas. On the 9th small hard lumps began to appear in the neighbourhood of the joints, with pain in the joints, especially the knees. On this day the patient went to a hospital, and on his out-patient paper was entered "Cellulitis of the face." An incision was advised, but as he could not be admitted, and felt very ill, he declined it. On the 10th the nodular swellings became red and inflamed, and pustules appeared on the swollen face, the lips, and the buccal aspects of the lips and cheek. The lips became black, nasal discharge commenced, and dyspnœa supervened. He was therefore taken to another hospital, when he was given a paper saying he was suffering from cellulitis and cephalitis (*sic*). Finally, he was taken in an ambulance to the Westminster Hospital.

The post-mortem examination showed abscesses in the muscles all over the body. The mucosa of the nose and sinuses was much swollen, but there was no ulceration of the nasal membrane. The lungs were engorged and the seat of numerous abscesses.

An inquest was held at Hammersmith in November, 1893, on William Wallace, a horse-keeper. The widow stated that towards the end of September the deceased told her he had been bitten by a horse, and about a week later he complained of severe headache. Following this came pain in the arms and legs, and on October 7 he went to a dispensary. The pains increased, and on October 14 he went to the West London Hospital, when he was told that he was suffering from rheumatic fever. He was admitted into the hospital on the 18th, and died on the 24th. Dr. Wilks, the House-Physician, said the case was of a very doubtful character, and it was not till the 23rd that a diagnosis of glanders was made.

I think it may be concluded that two symptoms which have been trusted to are not often prominent—a recognised spot at which inoculation has taken place, and the nasal ulceration and discharge. The symptoms which are most suggestive are the combination of high fever, a cutaneous rash, and pains in the muscles and joints.

It is remarkable how seldom any direct evidence of inoculation through the skin is given in recorded cases of human glanders, although a wound is always expected and looked for. I venture to suggest that too much credence has been given to the idea that man is generally infected by inoculation.

Horses are almost always infected by ingestion of contaminated food, and there seems no reason to believe that a similar method of infection may not take place in man. The habits of some stablemen are careless in the extreme, and I have frequently had to tell men who had handled the pus-covered nostril of a glandered horse to go and wash their hands. They were quite satisfied to wipe the contaminated hand on the seat of their trousers, and then resume their meal of bread and beef, or bread and cheese.

Experimental inoculation of glanders through the skin and accidental infection of a wound always cause local primary sores, and usually some lesions of the nearest lymphatic vessels and glands. We might expect that inoculation of the face and hands of man, if it occurred, would give rise to local lesions, and we know in cases where the wound and its infection are evident that marked local lesions do occur. The eruption that appears on man, usually a day or two before death, soon forms sores or ulcers; but these lesions are secondary, and their occurrence on the face does not prove that they indicate the site of inoculation. The ten days' illness which often precedes any local lesion must have been due to glanders infection, and any inoculated spot would have given evidence of morbid change at least as early as the access of the febrile symptoms. The fever, high temperature, and muscular pains, which exist for days without a skin lesion, are most suggestive of infection by ingestion.

Glanders in man is such a loathsome and fatal disease as to deserve more attention than it receives. Probably, if all cases were recognised, the mortality would be equally heavy, but prevention would be more actively assisted. If the medical profession called for the suppression of glanders as loudly as they did for the extermination of rabies, prevention in all animals would be accelerated.

Glanders has recently been scheduled as an industrial disease, and payment of compensation to infected employés by the owner of diseased horses follows. This ought to be more widely known, and the fact would lead to greater care.

The Glanders Order of 1907 required notification by the local inspectors to the Medical Officer of Health of the district in which the disease is found. This regulation is of little value, as any infection of man has taken place before the notification is served. If the Medical Officer of Health forwarded his information to the medical practitioners in his district, it might do some good by directing their attention to possible infections, and thus, perhaps, lead to an early diagnosis of other cases in man.

Finally, I would suggest that glanders should be included in the list of human diseases which are subject to compulsory notification.

TUBERCULOSIS

By E. WALLIS HOARE, F.R.C.V.S., and J. S. LLOYD, F.R.C.V.S., D.V.S.M.

Synonyms.—Consumption ; Scrofula ; Pining ; Grapes ; Pearl disease ; Phthisis pulmonalis ; Tabes mesenterica.

French : Tuberculose ; Pommelière ; Phtisie calcaire ou pulmonaire.

German : Tuberkulose ; Tuberculose ; Perlsucht.

Definition.—A contagious and inoculable disease affecting all species of animals, and also man, and caused by the *Bacillus tuberculosis.* The lesions are characterised by the presence of small tubercles in various organs of the body, or of diffuse infiltrations of tuberculous tissue, all of which tend to undergo certain degenerative changes, such as caseation, calcification, ulceration, and sometimes fibroid degeneration.

GENERAL REMARKS.

Tuberculosis is a disease of the greatest importance, not only from the fact that it causes serious losses to owners of cattle, but also, owing to its marked prevalence amongst the bovine species, there is a grave risk of the affection being communicated to mankind through the ingestion of the milk and meat of infected animals.

The relation between animal and human tuberculosis has been studied for many years. Although Koch believed that infection of human beings from the above source was of rare occurrence, the consensus of opinion amongst medical authorities is that bovine tuberculosis is communicable to man ; hence active steps should be taken to eradicate as far as possible the disease from cattle, and to secure efficient inspection of the meat and milk supply.

Tuberculosis in animals has been extensively investigated in many countries, and is still engaging the attention of scientists. These researches have resulted in not only a clearer perception of the etiology and pathology of the disease, but also in the adoption of preventive measures which are likely to prove of far-reaching importance.

The discovery of the tubercle bacillus and of tuberculin must be regarded as amongst the most important achievements in the history of the disease.

History.—Tuberculosis appears to have been recognised from the earliest times. According to Kitt, the communicability of pulmonary consumption was suspected even by physicians of antiquity, including Hippocrates and Isocrates, and also by some of the laity. According to Nocard, human phthisis is said to have been declared a contagious disease in one of the Gothic Laws.

The risk of ingesting the flesh of tuberculous animals was evidently recognised at a very early period, as rules against using the flesh of animals suffering from "wen or scurvy" occurred in the Mosaic Laws, also in the Talmud, and especially in the Mischnal (third century), while in the Gemara (fifth century) are found enactments of a similar nature, the terms "kandi" and "timari" probably referring to tubercle. (Friedberger and Fröhner). In the ninth century in Germany the Franks had ecclesiastical laws forbidding the use of the flesh of cattle and pigs affected with tuberculosis of the serous membranes. Laws were also enforced in various parts of Germany on the same subject between 1370 and 1582; but in 1783 the Berlin Board of Health declared that the flesh of tuberculous animals was fit for human food.

In 1816, however, rules for the inspection of meat were laid down by Tscheulin, in which only under certain conditions was the flesh to be condemned, and a similar procedure was adopted in several other countries on the Continent.*

In 1702, tuberculosis in cattle was erroneously identified with syphilis in man, and this view was adopted up to 1783, the term "French disease" being given to the affection, and compulsory slaughter of infected cattle was enforced in Prussia and Germany. When the duality of these diseases was accepted, the restrictions on meat inspection, as already mentioned, were withdrawn by the Berlin Board of Health.

In 1774, Ruhling of Göttingen regarded tuberculosis as a contagious disease in cattle, and Huzard and other observers in France, about 1790, believed in the identity of human phthisis and bovine tuberculosis.

In 1831, Gürlt pointed out the similarity of the lesions of tuberculosis in man and in cattle, while Spinola, in 1858, believed the two affections to be identical.

Other observers, however, held the view that the disease in cattle was distinct from human phthisis, and even doubted the contagious nature of the affection.

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* *Op. cit.*

The first important confirmation of the contagious character of the disease resulted from the inoculation experiments of Villemin in 1865. By means of inoculating human tubercular matter into animals he demonstrated that tuberculosis was an inoculable infective disease, and stated that the affection in man and in cattle was identical.

St. Cyr, Chauveau, Gerlach, Klebs, and others, conducted extensive feeding experiments from the year 1868, and demonstrated the transmissibility of tuberculosis by means of infected food.

Koch, in 1882, discovered the specific bacillus of the disease—the *Bacillus tuberculosis*—and considered this micro-organism to be identical in man, cattle, and fowl. During the same year Ehrlich directed attention to the special staining properties of the bacillus. In 1884, after fresh studies, Koch reaffirmed his former conclusions, and found the specific micro-organism in tuberculosis of the ox, horse, pig, goat, monkey, rabbit, guinea-pig, dog, cat, fowl, rat, and mouse.

Rivolta, in 1889, pointed out that marked differences existed between the bacillus of mammalian origin and that of birds. Later researches by Nocard and others showed that in reality these differences were very slight, and that by a series of passages through certain animals the bacillus of the mammalian type was identical with that of the avian variety.

In 1901 Koch retracted his earlier opinion, and stated that, as the result of his later experiments, he could only conclude that human and bovine tuberculosis were distinct affections, and consequently the infection of human beings from the ingestion of meat and milk derived from tuberculous cows was of very rare occurrence; also, special preventive measures in this direction were not necessary.

Koch's views, however, were only upheld by a few authorities, and the great majority of observers in the present day believe in the unity of human and bovine tuberculosis.

Nocard, Arloing, and others, believed that, although there are three types of tubercle bacilli—viz., the human, the bovine, and the avian—they are in reality only modifications of the same organism produced by environment.

In British veterinary literature of an early period the disease did not appear to attract much attention, if we are to judge from the articles on the subject and the cases recorded in the volumes of the *Veterinarian*.

From 1828 to 1842 we find no mention of the disease, and in 1843 there occurs a translation of a paper read by M. Rayer before the Academy of Science on "The Comparative Frequency of Phthisis in Man and Animals." In this paper tubercular phthisis is stated to be, of all chronic diseases, the most common both in man and animals; and wit reference to the latter the points of distinction between "tuberculou granulations," verminous and glanderous lesions, are given, attentio being also directed to the disease in the dog. From this period to 187 cases of the disease are occasionally reported, and it is surprising t note that the majority of these are cases of equine tuberculosis; but th clinical histories and the description of the pulmonary lesions given read remarkably like those of glanders.

In 1854 an article on "Tubercular Disease in Animals in India" appears. From the year 1875 onwards the disease attracted more attention, and in both the *Veterinarian* and *Veterinary Journal* numerous editorials, articles, and clinical records appeared. Amongst those who devoted much labour to the subject we may mention Fleming and Walley. Since the discovery of the tubercle bacillus the disease has received an enormous amount of attention from both veterinary pathologists and clinicians, and the literature on the subject is very extensive. The discovery of tuberculin by Koch in 1890 has proved of inestimable importance with reference to the diagnosis and prophylaxis of tuberculosis.

Geographical Distribution.—The disease is found in most parts of the world, and its prevalence seems to be unaffected by climatic influences.

It is said to be rarely met with in Iceland, Newfoundland, Arctic North America, the Gulf States, the northern parts of Norway, Sweden, Lapland, and Finland.

Jersey cattle are stated to be free from the disease when in their native country, but when housed in infected British cowsheds they frequently become affected.

In Japan the indigenous breeds of cattle are said to be unaffected, and the same is said of the Argentine native cattle; but when the latter are crossed with English breeds, the offspring are subject to the disease, the explanation being that infection occurs from the imported parents, when tuberculous.

Occurrence.—Tuberculosis may be justly described as the most widespread of diseases in animals, but it is unevenly distributed in the various species. Cattle are remarkably subject to the malady, and statistics show a very high percentage in dairy cows. In the Berlin dairies 75 per cent. were found to be affected, in London 26 per cent., in Edinburgh 20 per cent., and in Dublin about 12 to 15 per cent. Next to cattle, swine show the largest number of cases.

The horse is not specially liable to the disease, but since 1888 a number of cases have been recorded, and it is probable that with more exact methods of diagnosis it will be found more common than was formerly imagined. In sheep the disease is rarely met with, the percentage of affected animals being about 1 in 5,000. It can, however, be readily conveyed to them by experimental inoculation.

Goats are very rarely the subjects of tuberculosis in Great Britain, but the fact that these animals only form a small part of the animal population, and are usually kept singly, must be taken into consideration. On the Continent, however, where goats are much more numerous, and the conditions under which they are kept favour the spread of infection, caprine tuberculosis is more commonly met with. According to Ostertag, goats kept in stalls become tuberculous to the same extent as cattle.

The dog and cat were formerly regarded as not being very susceptible to the disease under ordinary conditions, but of late years, as more attention is being paid to the subject, and more exact methods of diagnosis adopted, the number of cases recorded has largely increased. In several instances infection from man to these animals has been traced.

Birds are very susceptible to the disease, and it is common in farmyard poultry, in pheasants kept in aviaries, and in cage-birds kept in zoological gardens. According to Fröhner, about 25 per cent. of parrots brought to the Berlin Veterinary School for treatment are tubercular. The disease has rarely been observed in wild animals in a state of nature, but it occurs when they are kept in confinement, as in zoological gardens. Monkeys are very susceptible, and reptiles and fishes may also be affected, also guinea-pigs and rodents.

With reference to the susceptibility of the different species of animals to tuberculosis, McFadyean pointed out that it was fallacious to calculate this from the frequency with which the disease attacks the individuals of each species. Account should be taken of the fact that the exposure to risks of infection may not be uniform in all species. "There is every reason to suppose that the main cause of the excessive prevalence of tuberculosis in cattle is not their great natural susceptibility to infection, but their indoor existence, which multiplies the chances of infection. Conversely, the escape of sheep is not due to natural resistance to infection, but to the fact that their outdoor life minimises the risk of infection. Tuberculosis is a disease of animals that have to spend the whole, or a great part, of their existence in houses."* We may, however, remark that at the Tuberculosis Congress of 1901. Reeves the Agent General of New Zealand, stated that tuberculosis was frequently detected in cattle that have never been housed.

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* *Journal of Comparative Pathology and Therapeutics*, vol. v.

ETIOLOGY.

The essential cause of tuberculosis is the *Bacillus tuberculosis*, which, gaining an entrance to the tissues of the animal body, multiplies therein, and induces the characteristic lesions of the disease.

Hereditary transmission at one time was regarded as an important etiological factor, but, owing to the researches of Bang, it is now clearly demonstrated that tuberculosis is not an hereditary disease. *Hereditary predisposition*, however, undoubtedly plays an important part in its genesis. According to Moussu, the offspring of tuberculous parents have not the same vital power to resist infection as those born of healthy parents, and a greater tendency exists in them to contract the disease. He regards this aptitude, or predisposition, as one of the essential factors in the development of the malady, as the tubercle bacilli exert their effect more readily under such conditions.

BACTERIOLOGY.—The tubercle bacillus is a thin rod-shaped organism, its average length being from 2 to 4 μ, and its breadth about $\frac{1}{3}$ to $\frac{1}{2}$ μ. Occasionally bacilli twice this length may be found. They may be straight, but are often slightly bent or curved; they have rounded ends, and are aërobic and non-motile. The longer rods may not stain uniformly, and thus may show alternating stained and unstained portions, a beaded surface composed of a series of round or oval granules being produced. The unstained portions, oval in shape, are sometimes regarded as spores, but it is doubtful whether this view is correct.

The bacilli may be solitary, but both in the lesions and in artificial cultures they tend to group themselves together in a manner resembling bundles of faggots. In the tubercles they are often found to arrange themselves in a radiate manner at the periphery of the giant cells.

Variations.—Small branching forms may be found, and complex structures forming club-shaped groups resembling actinomyces are described.

Habit.—The tubercle bacillus is regarded by most authorities as a strictly obligatory parasite that cannot multiply as a saprophyte in temperate climates, as its growth is slow even at temperatures slightly below that of the body. Some investigators, however, including Nocard, have shown that the bacillus of avian tuberculosis can be made to live in suitable dead mixtures of organic matter.

The bulk of evidence goes to show that the bacillus does not grow free in Nature, and that it occurs only when it is deposited with tuberculous discharges from the body.

Staining Reactions.—There are two important peculiarities in connection with the staining reactions of the tubercle bacillus: (*a*) It offers marked resistance to staining with the ordinary basic aniline dyes. This is believed to be due to the presence of fatty or waxy substances in the outer part of the bacillus. (*b*) It retains the stain after treatment with aqueous solutions of mineral acids, and hence is termed "acid-fast."

Other pathogenic bacilli are now known to be acid-fast — viz., the bacillus of Jöhne's disease and the bacillus of leprosy; the latter disease, however, does not occur in animals. Other bacterial species also share this characteristic—viz., the *Bacillus smegmatis*, the Timothy grass (*Phleum pratense*) bacillus, the dung bacillus of Moëller and the butter bacillus of Rabinowitsch.

Occurrence in the Body.—The tubercle bacillus is a tissue parasite, and, although it may be carried by the blood-stream, there is no direct evidence to show that it ever multiplies therein; moreover, the bacilli are never found in large numbers in the blood. In post-mortem examinations the bacilli are found almost entirely confined to the lesions. In tuberculous poultry they are said to occur in the liver without any lesions being observed in this organ.

The bacilli occur in large numbers in actively growing tubercles, but are scanty in the chronic processes of lymph glands and of joints.

If a tuberculous focus communicates with a vein or with lymphatic vessels, the bacilli may be spread throughout the body. According to von Behring, Calmette, and Guèrin, the bacilli may be located in the tissues, especially the glandular—*e.g.*, the mesenteric glands—without any macroscopic lesions being observed. In old lesions their presence may not be detected microscopically, and cultures and inoculations may be necessary to demonstrate the true nature of the specimens.

Resistance and Vitality.—Tubercle bacilli possess a moderately strong resistance to high temperatures. According to Bang, a temperature of 85° C. was sufficient to render them harmless. Jensen demonstrated that when the bacilli were uniformly distributed, exposure for ten minutes to a temperature of 75° C. killed them. The higher the temperature, the shorter is the period of exposure necessary to render them innocuous.

Exposure to sunlight is said to destroy them in from two to four hours, but some observers state that several hours' exposure is necessary for this purpose.

As regards the effect of *chemical agents* on the bacilli, solutions of $\frac{1}{10}$ to $\frac{1}{50}$ per cent. of perchloride of mercury render human tubercular sputum sterile in from twenty to twenty-four hours. In bouillon culture the bacilli are killed in thirty seconds by a 5 per cent. solution of carbolic acid, by a 1 per cent. solution of the same acid in one minute, and by a 1 to 1,000 per cent. solution of perchloride of mercury in ten minutes.

According to Cadéac, the bacilli remain virulent in ordinary water for 120 days. Human tubercular sputum, when dried, is said to remain virulent for a year. Exposure to alcohol, putrefaction, desiccation, or freezing as low as—8° C., is said not to render it sterile.

Of 118 dust samples from hospital wards or the rooms of human phthisical patients, forty were infective and produced tuberculosis by inoculation into susceptible animals; virulent bacilli were obtained from the dust of the walls of fifteen out of twenty-one medical wards; but in two wards with many phthisical patients the results were negative, indicating that the dust in such regions is not necessarily infective (Osler).

According to Moussu, contagion only occurs in cattle-byres as the result of the presence of animals with open tuberculous lesions, such as caverns in the lung, tuberculous bronchitis with ulceration of the mucous membrane, tubercular enteritis, etc. The bacilli are expelled in the saliva, nasal discharge, fæces, urine, etc., and contaminate forage, litter, manure, drinking-water, etc. After desiccation, the bacilli may be spread by currents of air. Mangers, hay-racks, drinking utensils, etc., may also become contaminated.

According to Forster, when salt was sprinkled on pure cultures of the bacilli, they retained their vitality for two months. Portions of tubercular organs, finely minced and laid in salt brine for eighteen days, still remained virulent. Tubercle bacilli may remain for six hours in gastric juice without their virulency becoming impaired, and twenty-four hours' exposure is necessary in order to destroy them.

According to the feeding experiments of Cadéac, conducted on guinea-pigs, alimentary tuberculosis was not produced with certainty except the animals received at least 1 gramme of tubercular material moderately rich in bacilli. In smaller amounts the results were uncertain, varying, and limited. Wesner's experiments showed similar results (Ostertag).

Types of Tubercle Bacilli.—For descriptive purposes, three types of tubercle bacilli were distinguished by the Royal Commission on Tuberculosis in the final report (1911)—viz., the bovine, the human, and the avian type—and from this report the following notes and extracts are taken:

The Bovine Type.—In natural cases of tuberculosis in cattle, this is said to be the only type of bacillus present. It is distinguished by its cultural characters and by the characteristic effects produced when it is inoculated in certain doses into calves and rabbits; also it grows solely on serum. In arriving at a conclusion as to the type of the bacillus, these features must be taken into consideration. The bovine type of tubercle bacillus has been termed "dysgonic," because of its slow growth on serum. At the end of two or three weeks it forms a thin, greyish, uniform growth, which is neither wrinkled nor pigmented. These properties, as we shall see, are in contrast with those shown by the human type of tubercle bacillus.

When 50 milligrammes of a culture of bovine tubercle bacilli are administered to a calf by subcutaneous injection under the skin of the neck, severe generalised tuberculosis results, which starts from the point of inoculation, and death generally ensues within eight weeks. This marked virulence is in contrast with the effects produced by the other types of tubercle bacilli.

In rabbits, 0·01 or 0·1 milligramme of a culture of bovine tubercle bacilli administered by intravenous injection produced generalised tuberculosis, terminating in death within five weeks; with doses of 0·1 to 1 milligramme given by intraperitoneal injection, extensive tuberculosis of the peritoneum and abdominal lymphatic glands, extending afterwards to the internal organs, was produced; while subcutaneous inoculation with doses of 1 milligramme to 10 milligrammes caused a local lesion, with tuberculosis of the neighbouring lymphatic glands, also of the lymphatic glands of the body and of the internal organs, especially the lungs and kidneys. Death occurred in from 29 to 165 days and 28 to 101 days, according to the doses employed.

In the goat, pig, and cat, subcutaneous inoculation of the bovine tubercle bacillus produces generalised tuberculosis, and, provided the dose be adequate, the results are of equal importance in distinguishing this bacillus from the other types, as those obtained by inoculating the calf and rabbit.

In the chimpanzee, monkey, and guinea-pig, very small doses of the bovine tubercle bacillus administered by subcutaneous inoculation produce acute tuberculosis.

The dog shows marked resistance to subcutaneous inoculation of the bovine tubercle bacillus, but large doses inoculated intravenously or intraperitoneally may cause general tuberculosis and death.

The rat and mouse also prove highly resistant to the subcutaneous inoculation of the bovine tubercle bacillus; but if administered by intraperitoneal inoculation, the bacillus tends to multiply in the body, and can be demonstrated in large numbers in the organs, and even in the blood; but no tuberculous lesions are formed, such as occur in animals susceptible to this type of bacillus.

In the horse moderate doses of the bovine tubercle bacillus, administered by subcutaneous inoculation or by feeding experiments, do not produce progressive tuberculosis; if, however, a dose of 10 milligrammes be given intravenously, death from acute tuberculosis occurs in twenty days.

In the fowl even large doses of bovine tubercle bacilli, administered by intraperitoneal or intramuscular injection, produce only a local lesion. If, however, the bacillus be administered by intravenous injection, death occurs in about half the number of cases, being usually preceded by emaciation, the post-mortem generally showing pulmonary œdema and pallor of the liver, while in some instances definite tubercles are found in the lungs, and necrotic areas in the liver. It was found that dead tubercle bacilli of the bovine type injected intravenously produced similar effects to those of the live bacilli; hence the fatal results were believed to be due to the toxic effect of these micro-organisms.

The Human Type.—This is the type of bacillus found in the majority of cases of human tuberculosis. In consequence of its more rapid growth on serum as compared with that of the bovine tubercle bacillus, it is described as "eugonic." The growth tends to assume a wrinkled appearance on glycerinated media, and becomes more or less pigmented on all media.

In calves it was found that a subcutaneous inoculation of 50 milligrammes of a culture of the human bacillus under three weeks old did not produce progressive tuberculosis or cause death. In most cases a local lesion resulted, consisting of a mass which was either large or small, and might become converted into a cyst surrounded by a fibrous capsule. When internal lesions resulted, they generally consisted of caseous or calcareous nodules in the lymphatic glands nearest the seat of inoculation, or of a few calcareous tubercles in various internal organs. The results of the experiments carried out were, that in about half the number the lesions did not extend beyond the glands nearest to the seat of inoculation, and the conclusion drawn was that the human tubercle bacillus possesses a much lower virulence for the calf than the bovine tubercle bacillus.

In the rabbit subcutaneous inoculation of the human bacillus gave, as a rule, similar results to the above. When, however, intravenous inoculations are carried out in this animal, a slowly progressive tuberculosis with limited lesions is induced in the majority of cases, which is in marked contrast with the acute generalised form of the disease, terminating in death within five weeks, which occurs when similar doses of the bovine type of bacilli are employed. In some cases, however, the rabbit shows extensive and fatal tuberculosis from inoculation with the human type of bacillus.

In the goat and pig inoculation with the human bacillus was found to produce only a slight retrogressive tuberculosis, but pigs were slightly more susceptible than calves.

In the chimpanzee, monkey, and guinea-pig, inoculation with the human tubercle bacillus produced acute tuberculosis, the effect in the two former animals being similar to that following inoculation with similar doses of the bovine bacillus; but in the guinea-pig the duration of life was longer when the human bacillus was employed.

In the dog the effects are similar to those produced by the bovine type of bacillus.

In the cat resistance is shown to the human tubercle bacillus when administered by all methods, including feeding experiments. Intraperitoneal inoculation produced a few tuberculous lesions which were not widely distributed, and did not affect the health of the animal.

In the rat and mouse intraperitoneal injections of large doses of human tubercle bacilli resulted in dissemination of the bacilli over the body, and their multiplication in the tissues; but death occurred without the production of characteristic tuberculous lesions.

In the fowl the effects produced were similar to those induced by the bovine tubercle bacillus.

In the horse one experiment was carried out, and a subcutaneous injection of 50 milligrammes of the human bacillus produced only a local lesion.

It is of interest to note that in the case of a gnu and of an antelope that suffered from generalised tuberculosis, naturally acquired, the human type of tubercle bacillus was found in the lesions; and in the case of a monkey and a chimpanzee, under similar conditions, the presence of the human bacillus was also demonstrated.

In a case of naturally acquired tuberculosis in a cat, a culture isolated directly from an affected mesenteric gland grew like the bovine tubercle bacillus, and, like the latter, it produced fatal generalised tuberculosis in rabbits. " These cases show that the tuberculosis developed by mammals kept in a state of captivity may be due either to the human tubercle bacillus or to the bovine type. Owing to the small number of cases investigated, no conclusions can be drawn as to the relative frequency of infection of such animals by the one or the other type of bacillus."

Another point of interest is with reference to the transmutation of the bovine bacillus into the human bacillus, and *vice versâ.*

Although most of the attempts in this direction failed, and a few only were equivocal, the Commissioners stated: " Thus we are inclined to regard transmutation of bacillary type as exceedingly difficult, if not impracticable, of accomplishment by laboratory procedure, though, in view of certain instances in which we obtained from one and the same human body both types of bacillus, we are not prepared to deny that the transmutation of one type into another may occur in Nature."

Certain bacilli which were isolated from cases of lupus in man and tuberculosis in the horse possessed exceptional characters. "In some of these cases the bacilli had the ordinary cultural characters of the bovine type, associated with a degree of virulence for the calf and rabbit no greater than is usually exhibited by the human type."

These might be regarded as distinct fixed types, or as either modified human or modified bovine bacilli.

"On the former assumption they had acquired the character of growth ordinarily exhibited by the bovine type, and on the latter assumption they had lost the higher degree of virulence for certain animals which is characteristic of the bovine type. The latter appears to be the more probable explanation, and if that view be accepted, the discovery of these exceptional bacilli makes it impossible to regard difference of virulence for the calf and rabbit as sufficient to establish the non-identity of the human and the bovine types."

"There would, therefore, remain only slight cultural differences on which to found the conclusion that the human and the bovine types represent two distinct organisms. We prefer to regard these two types as varieties of the same bacillus, and the lesions which they produce, whether in man or in other mammals, as manifestations of the same disease."

The Avian Type is the one present in cases of natural tuberculosis occurring in poultry, and shows certain peculiarities with reference to its growth; thus it forms a slimy, whitish growth which is readily emulsified, and it grows especially well on glycerinated media.

The fowl is very susceptible to the avian bacillus by intravenous, subcutaneous, and intramuscular inoculation, also when feeding experiments are carried out.

The lesions produced by inoculation occur in the spleen and liver, and frequently in the lungs, cervical glands, muscles, and bones. Similar effects are produced by feeding experiments, but in addition lesions of the intestinal mucosa are observed.

In the parrot similar lesions are produced, but when feeding experiments are carried out, intestinal lesions are not so constantly observed as in the fowl. The parrot is susceptible to the action of both the bovine and human tubercle bacillus, and when either of these is administered by inoculation or by feeding, the lesions are similar to those produced by the avian bacillus.

It is found that the bovine bacillus is apparently more virulent for the parrot than either the human or the avian bacillus.

In the rabbit and mouse the avian bacillus causes progressive tuberculosis, these being the only two mammals in which this form of the disease is produced.

The rabbit shows certain peculiarities in the results produced by the avian type of tubercle bacillus. When moderately large doses are given by inoculation, they prove fatal, but the results show that the avian type of bacillus is less virulent for this animal than the bovine type, but more virulent than the human type.

Subcutaneous inoculation of the avian bacillus in doses ranging from 50 milligrammes to a fraction of a milligramme produce in the rabbit a very chronic form of tuberculosis, in which the distribution of the lesions differs to a marked extent from that caused by the bovine and human tubercle bacillus, and this peculiarity is practically unaffected by the size of the dose administered. Similar results follow intravenous injection of a very small dose (0·001 milligramme). In addition to local lesions, the nearest lymphatic glands are involved, and also the kidneys to a variable extent. Slight œdema of the lungs, with a few tubercles on the surface of these organs, is observed, also caseating tubercles in the areolar tissues, in a few of the lymphatic glands, and in the testes; while the liver and spleen are but rarely affected.

The most characteristic and important lesion, however, is a chronic tuberculosis of the joints of the limbs. Tuberculosis of the joints occasionally occurs after intravenous inoculation with the human tubercle bacillus, but not after subcutaneous inoculation, and similar lesions may be observed after subcutaneous inoculation with the bovine bacillus, provided the animal survives for a sufficiently long period.

In the mouse the avian bacillus produces generalised tuberculosis, whether administered by subcutaneous or intraperitoneal inoculation, or by feeding.

In the calf, pig, horse, cat, rat, monkey, and guinea-pig, progressive tuberculosis is never produced by the avian bacillus, although in some instances it may multiply in the body and become disseminated in the tissues; but if a large dose be administered by intravenous injection a fatal result may ensue.

In the dog the intravenous injection of large doses produces no effects.

In one experiment carried out on a chimpanzee, 50 milligrammes injected subcutaneously produced no effect, and on post-mortem examination of the animal three years afterwards no tuberculous lesions were discovered.

MODES OF INFECTION.

1. Hereditary Transmission.—Congenital tuberculosis is very rarely met with. According to Moussu, the passage of tubercle bacilli through the placenta only occurs under exceptional conditions, such as when the bloodvessels are affected.

Other authorities hold that the "almost constant method of transmission in congenital tuberculosis is through the blood-current, the bacilli penetrating by way of the placenta. Certain authors hold that in these cases the placenta is invariably the seat of tuberculosis, and tubercles, indeed, have been demonstrated in several cases; but there are undoubted instances in which, with an apparently sound placenta, both the placental blood and the fœtal organs contained tubercle bacilli, notwithstanding the fact that the organs also appeared normal."*

Tuberculous lesions of the maternal genital organs may lead to congenital infection of the fœtus. Moussu points out, however, that tubercular disease of the ovaries, Fallopian tubes, or uterus, generally prevents pregnancy and causes sterility. Direct paternal infection, according to the same author, is only possible when ulcerating tubercular lesions of the testicle, prostate gland, or vesiculæ seminales, are present, and it is very rare.

The experimental work of Gärtner and others shows that healthy female rabbits impregnated by tuberculous males do not produce tuberculous offspring, although the females may contract the disease.

The consensus of opinion in the present day is that infection usually takes place after birth, and that the disease is rarely present in the newly-born, even when both parents are tuberculous.

The proportion of tuberculous cows that produce calves congenitally affected is said by some observers to be about 1 in 300, but this estimate seems far too high. According to McFadyean, congenital tuberculosis in the calf is very rare; he recorded the first case in Great Britain in 1891. Up to 1891 only six cases were recorded on the Continent, in which the presence of the disease was proved by the demonstration of the tubercle bacillus. The proportion of cases in calves under one month old is said to be about 1 in 70,000.

2. Infection by Inhalation.—This is recognised by some authorities as the most probable mode of infection in cattle. As compared with the mode of infection by ingestion, it may be described as the older theory. It is believed to take place in confined, ill-ventilated buildings, the chief source of the infection being the expectorate coughed up by a

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* Osler's "Principles and Practice of Medicine."

tuberculous animal. This infective material may be in the form of a fine spray, which contaminates the surrounding atmosphere, and, being inhaled by the other occupants, may be the means of introducing the bacilli into their lungs. Or the expectorate may be of a coarser nature, and, falling on surrounding surfaces or objects, it becomes desiccated; it is readily disturbed, and, in the form of dust particles, contaminates the atmosphere, and may thus prove a source of infection by inhalation.

Direct infection of the lungs may be produced by the above means.

This view is contested by many authorities, who hold that infection by ingestion is by far the more common method.

Lately McFadyean has stated that primary tuberculosis of the lungs is probably always caused by inhalation of infective material, and that when the lungs are affected from the alimentary tract, the pulmonary lesions are secondary to diseased conditions elsewhere in connection with the digestive system. In a paper entitled "What is the Common Method of Infection in Tuberculosis?"* he weighs the evidence for and against the inhalation theory of infection in a remarkably clear and lucid manner, and arrives at the following conclusions:

"(1) The inhalation of tubercle bacilli suspended in the atmosphere is a very certain method of infection in susceptible animals, even when small doses of bacilli are employed.

"(2) Experimental infection with tubercle bacilli by way of the alimentary canal is comparatively difficult to realise even in highly susceptible animals, and success is certain only when very large doses of bacilli are administered.

"(3) With few exceptions, in animals experimentally infected with tuberculosis by way of the intestine, the primary lesions are intra-abdominal, and the intrathoracic lesions, when present, are secondary.

"(4) Inhalation is probably the commonest natural method of infection in those species (man and cattle) in which the primary lesions of tuberculosis are usually intrathoracic.

"(5) Naturally contracted cases of tuberculosis in man and other mammals can be ascribed to infection by ingestion only when the lesions revealed at the post-mortem examination are confined to the abdomen, or when the existing abdominal lesions are recognisably older than those present elsewhere in the body."

3. Infection by Ingestion.—Chauveau (1868), Gerlach (1869), and others, held, as the result of their experiments, that infection by ingestion was the commonest method. Calmette, Guèrin, and Whitla advanced a

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* *Journal of Comparative Pathology and Therapeutics*, September and December, 1910.

similar view, and claimed that ingestion of tubercular material can produce pulmonary lesions without causing any visible lesions in the intestinal mucous membrane. They found that in young animals the bacilli induced lesions in the mesenteric glands, but in older animals the bacilli more generally entered the lymph stream, and, reaching the pulmonary artery, passed to the lungs. These observers also held that the bronchial and mediastinal lymphatic glands may become infected as the result of ingestion of tubercular material without any lesions being found in the intestinal mucosa or in the mesenteric glands.

Further information on this subject, and an analysis of the views of various observers, will be found in the paper by McFadyean already referred to.

Infection by ingestion may occur from feeding on fodder, etc., contaminated by tubercle bacilli. When a number of cattle are fed together, a tubercular subject amongst these may contaminate the fodder during the act of coughing by depositing thereon the infective expectorate. Straw, litter, etc., may be contaminated by the fæces of animals suffering from intestinal lesions, and so become infective if eaten by cattle or horses; also, as tuberculous animals swallow a large proportion of the expectorate that is coughed up in the pharynx, the fæces may be rendered infective by this means. The pharynx and pharyngeal glands may become infected during the swallowing of food containing tubercle bacilli; some observers, however, believe that this region is a seat of predilection for the disease, and that the bacilli are conveyed thereto by the blood. Dogs and cats may become infected by consuming food contaminated with expectorate from tuberculous human beings, or from ingesting meat or milk containing tubercle bacilli. Pigs are infected in a similar manner; probably tonsillar infection may occur in this species as in man. Birds contract the disease from the ingestion of grain, etc., contaminated with the fæces of birds suffering from tuberculosis, or by the sputum of infected human beings. It is probable that ingestion plays a very prominent part in the etiology of the disease in young animals when these are fed on milk obtained from animals suffering from tuberculosis of the udder. Foals fed on such milk may become infected; also calves, pigs, and probably dogs.

According to Friedberger and Fröhner, the milk of tuberculous cows may contain tubercle bacilli in the absence of mammary tuberculosis. Whey, cheese, and, butter prepared from the milk of tuberculous cows, nay transmit the disease even after a lapse of from one to four weeks, according to the investigation of Bang, Roth, and others. Bang has also demonstrated by inoculation experiments that skimmed milk and cream prepared by centrifugal methods from tubercular milk are infective.

4. BY INOCULATION.—This mode of infection is rare in animals. It may occur if tubercle bacilli become implanted deeper than the epidermis, local tubercle of the skin resulting. The lesion occurs in the form of raw warty projections, which contain tubercle bacilli and show caseating centres at certain points.

A similar form of the disease may occur in man as the result of local infection from handling organs or parts affected with tuberculosis when abrasions are present on the skin of the hands, most of the cases recorded being in demonstrators of pathology, butchers, handlers of hides, etc. The dorsal surfaces of the hands or fingers are usually affected in the form of a reddened mass of granulation tissue known as the *Verruca necrogenica* of Wilks. It usually remains local, but a case is recorded in which, after excision of the primary lesion, the lymph glands of the axilla became infected, and, when removed, showed tubercular changes and the presence of tubercle bacilli.

It is said that some cases of mammary tuberculosis in cows may be due to local infection through a wound or abrasion of the skin of the udder. According to Bang, it is possible that primary tuberculosis of the udder may occur by tubercle bacilli penetrating through the canal of the teat.

Tuberculosis can be readily transmitted to animals by experimental inoculation, a fact which is extensively taken advantage of in testing milk or other material suspected of being infected by the disease. Usually two forms of inoculation are made use of—viz., the subcutaneous and the intraperitoneal. If the suspected material contains living tubercle bacilli, tuberculosis results, at first as a localised infection, which usually tends to become generalised afterwards, and to prove fatal.

RARE MODES OF INFECTION.—Amongst these may be mentioned tuberculosis of the vagina in the cow, due to transference of bacilli by the penis of a bull that has recently served a cow suffering from this disease in the vagina. On the other hand, the bull may contract tuberculosis of the penis from a similar source, and convey the disease to the vagina of a healthy cow by coition.

LATENT INFECTION.—Some authorities, including von Behring and Baumgarten, are of opinion that it is possible for tubercle bacilli to remain latent in the tissues of young animals, and to subsequently develop in adult life, and give rise to the disease. The reasons for this phenomenon are not understood, and this form of infection cannot at present be regarded as otherwise than a theoretical one.

CONDITIONS INFLUENCING INFECTION.—Dark, damp, ill-ventilated, badly-drained buildings, favour infection. The bacilli are less likely to be inhibited or destroyed under such conditions. Overcrowding offers facilities for the conveyance of the disease from the affected to the healthy animals. According to Friedberger and Fröhner, the vitality of the respiratory organs becomes weakened in cattle kept in ill-ventilated sheds; hence they are predisposed to the entrance and development of the tubercle bacilli in their systems. The same authors state that catarrhal affections of the respiratory mucous membrane, which are often caused or favoured by the air in ill-ventilated sheds, favour the penetration of the bacilli; also that the abundant production of milk and the strain of yearly breeding tend to weaken the system and impair the natural powers of resistance to infection. Continuous inbreeding is regarded by the same authors as a predisposing factor.

With regard to age, the disease is most prevalent in old milch cows, especially those kept in town dairies.

Infection in cattle is usually the result of cohabitation with affected animals. Contact between diseased and healthy animals for a period of some days, or even weeks, does not seem sufficient to transmit the disease. Nocard regards a mean period of five to six months as necessary, and Moussu has arrived at a similar conclusion as the result of observations made by placing tuberculous cattle and healthy cattle together in byres reserved for such researches. Of course, the differences in individual susceptibility must be taken into consideration when dealing with such experiments.

MORBID ANATOMY.

THE TUBERCLE is the primary lesion produced by the *Bacillus tuberculosis.*

Nodules, identical in structure to tubercles, are found in other diseases, and in the present day the term "nodule" is applied to lesions which are not tuberculous, while the term "tubercle" is restricted to the primary lesion caused by the tubercle bacillus.

The tubercle is very small at the commencement of its growth, and increases to the size of a pin's head or larger. A number of these small tubercles may coalesce and form masses of various sizes.

The essential cause of the formation of these tubercles in any organ or tissue is the presence of tubercle bacilli, which multiply and cause irritation. The effect of this irritation is to cause first a proliferation of the fixed cells and the production of epithelioid and giant cells. An inflammatory reaction next occurs, associated with exudation of leucocytes.

According to Baumgarten, three varieties of cells are found in a typical fully-formed tubercle.

1. *Epithelioid Cells*.—These are the first cells formed, and their origin is believed to be the proliferation of the fixed cells of the part, especially those of connective tissue, endothelium of the blood and lymph capillaries, and sometimes from epithelial cells. The epithelioid cells are the most numerous, and very young tubercles are formed altogether of them. In shape they are generally angular; they present a single vesicular nucleus and a large cell-body, and some may contain tubercle bacilli.

2. *Round Cells*.—These are leucocytes which have migrated from the capillaries of the infected focus. In the fully-developed tubercle they are not numerous, and are found chiefly at its periphery, and during the development of the tubercle many of these cells undergo rapid destruction; they are polynuclear, but as growth proceeds in the lesion, they become chiefly mononuclear, and do not undergo marked degeneration.

3. *Giant Cells*.—These are large in size and multi-nucleated. They are probably formed by fusion of the epithelioid cells around tubercle bacilli. According to certain observers, in some, but not all, tubercles giant cells are formed by an increase in the protoplasm and in the nuclei of an individual cell, or possibly by fusion of several cells. As compared with the other cells in a tubercle, they are in a minority. In man the giant cells seem to be in inverse ratio to the number and virulence of the bacilli (Osler).

In developed tubercles, giant cells are constantly present in the ox and horse; in the latter they generally occur in considerable numbers. They are usually present in tubercles of swine, and are often found in those of the dog and cat. In the horse the bacilli in a group often occupy the body of the cell, while in the ox and other animals they are often radially arranged towards the periphery of the cell between the nuclei. According to Ostertag, the formation of giant cells only fails to occur in cases of extensive penetration of tubercle bacilli, or when the latter are very virulent. In some instances no bacilli are present in the giant cells.

Giant cells are not peculiar to tuberculous lesions, but their presence is of diagnostic value, especially when they are found in lesions showing evidences of necrosis and caseation. They bear a close resemblance in shape to the giant cells of bone-marrow, and, also in common with the latter, the multiple nuclei are generally massed together towards the periphery of the cell. As regards situation, giant cells are found scattered in an irregular manner throughout the tubercle. The presence of these cells in a tubercle is, according to McFadyean, an indication that it has been forming for at least two or three weeks. Some authorities regard giant cells as a special variety of phagocytes.

In addition to the structures mentioned, there is also found in a tubercle a reticulum of fibres formed by the fibrillation and rarefaction of the connective-tissue matrix, usually most apparent at the margin of the growth.

A tubercle is usually composed of the three varieties of cells mentioned above and tubercle bacilli, while in some instances traces of the original histological structure of the part can be recognised. Generally these disappear, together with the capillaries, and, as no new vessels are formed, the tubercle is non-vascular. Exceptions to this rule as regards vascularity are met with in the tuberculous lesions of the pleuræ and peritoneum of the ox, as the tubercles in these locations contain bloodvessels.

Degenerative Changes in the Tubercle—1. *Caseation.*—After four weeks, or sooner, a process of coagulation necrosis occurs in the cells at the central part of the tubercle. These lose their outline, become irregular, no longer take the usual stains, and finally are converted into a homogeneous substance devoid of structure. Caseation quickly follows on necrosis, the process extending from the centre outwards. Prior to the occurrence of degenerative changes, the tubercle is grey in colour and somewhat translucent in appearance; after necrosis occurs, the centre becomes opaque, and with the advent of caseation it becomes of a yellow colour, and the tubercle is gradually converted into a yellowish-grey body, in which tubercle bacilli are still present, but bloodvessels are absent.

These changes are due to the direct action of the bacilli or of their products. According to Kitt, caseation is to be ascribed to a toxic material which is given off by the bacilli.

Caseation is constant in tubercular lesions of the ox, horse, pig, and fowl, but there is less tendency to this change in those of the dog and cat.

Although caseation is pathognomonic of tubercle, it is not peculiar to this disease, as this degenerative change is also met with in the pulmonary lesions of glanders and in other specific inflammations.

2. *Calcification.*—In this, a deposition of irregular granules of carbonate and phosphate of lime occurs in the caseous part of the tubercle. This change is most marked in the ox and pig, and occurs with comparative frequency in the horse, while it is uncommon in the dog and cat. The tubercle is hard and cretaceous both in appearance and on palpa-

tion, and grates under the knife. Calcification is regarded as evidence of chronicity in tubercular lesions.

3. *Softening.*—In the ox the caseous material in the tubercle may become converted into a yellow custard-like material, while in the horse it resembles thick pus.

4. *Fibroid Degeneration.*—This is also known as *fibrosis* and *sclerosis.* According to McFadyean, it is a very uncommon alteration in the tuberculous lesions of animals. In the tissue of the tubercle a productive fibroid inflammation occurs, which may convert the lesion into a firm, hard structure. Or the change may be of a fibro-caseous nature, the tubercle showing a caseous centre, while the zone nearest to the normal tissue shows fibroid development. The process is regarded as a conservative and healing one. When present, it is chiefly found affecting the lesions in the peritoneum, and very few bacilli can be discovered in them.

Rôle played by Tubercle Bacilli.—When the bacilli gain access to the tissues, provided they find a suitable soil, they multiply and form toxic substances or toxins, which exert an injurious influence on the tissue cells. The presence of the bacilli and of their toxins causes a reaction on the part of the tissues, as the result of which the tuberculous lesion is formed. If the soil is not suitable, and the animal not very susceptible, or if the bacilli are not very virulent, the latter may remain enclosed in the primary lesion, or they may perish therein, and the animal recovers completely.

More commonly, however, when the soil is suitable, the lesion becomes developed, and the effect produced is that the course of the invasion is delayed. The site of formation of the tubercle is in the connective tissue of the affected part, or in the walls of the bloodvessels and in the lymphatics of the part.

Secondary Inflammatory Processes.—Extensive inflammation may be produced by the irritation resulting from the presence of the bacilli and their toxins. Thus in the lungs pneumonia may occur, with proliferation of the connective-tissue elements in the septa, infiltration of the latter with round cells, and alterations in the bloodvessels and lymph vessels. Or the development and spread of the tubercles may be limited by the production of a cicatricial connective tissue, resulting from inflammation of a slow reactive type.

SUPPURATION.—This process may occur in the tuberculous lesions of various organs, and also in the form of the so-called cold tuberculous abscess.

In the latter the pus is described as "sterile"—*i.e.*, it does not contain the usual pus organisms—and some authorities state that the material in the abscess is not histologically pus, but consists of broken-down cells and cheesy material. Such pus, however, is not sterile in the strict sense of the word, as it contains tubercle bacilli, which may be detected by staining and microscopical examination, or, if not, by guinea-pig inoculations. In some instances the number of tubercle bacilli in such pus may be small, and may thus be overlooked by microscopical examination, or they may have died out. Such pus would be more correctly described as "sterile on ordinary media," as tubercle bacilli will not grow on these.

Ordinary pus, however, occurs in tuberculous lesions of joints and bones, and is said to be due to the products of tubercle bacilli or to a mixed infection. It may also occur in pulmonary tuberculous lesions, and is said to be largely the result of an infection with pus organisms.

Anatomical Forms of Tuberculosis.—These vary according to the predisposition of the tissues in different animals, and also according to the extent and duration of the disease.

The principal forms met with are as follows:

1. *The Miliary Tubercle.*—The structure of this has already been described (see p. 123). It is the earliest manifestation of the tuberculous lesion. Miliary tubercles take about two weeks after infection has occurred to develop to macroscopic size, and after four weeks they become as large as ordinary pin-heads. The microscopic stages at the commencement may be observed in from five to ten days after infection. In colour these tubercles are of a grey or yellowish-red; they may be single or multiple, and may extend widely over a large surface or become disseminated through the tissues of the organs affected, the pleuræ and lungs being most commonly involved.

Acute Miliary Tuberculosis.—In this condition the organ affected is the seat of miliary tubercles which are of uniform size and age.

Chronic Miliary Tuberculosis is characterised by a local multiplication of the tubercles; they enlarge in size, and the larger forms arise from either fusion or conglomeration of the smaller tubercles. When these tubercular masses occur on serous surfaces, they form what is sometimes termed "pearl disease" or "grapes."

2. *Diffuse Form.*—In this form no well-defined tubercles are present. It is found especially on serous surfaces in the form of a soft grey or greyish-red layer; new bloodvessels are formed in the process, the lesion has a granular appearance, caseation only occurs to a slight extent, and not until a considerable time has elapsed. This type of lesion is believed to depend on the presence of large numbers of bacilli through-

out the tissue, instead of being in separate groups, such as occurs in the ordinary form of the disease. When occurring in serous membranes, the lesion may cause actual exudation on the surfaces of the affected parts.

3. *Tuberculous Fungoid Granuloma.*—This form of tuberculous lesion is chiefly met with in the pleuræ and peritoneum, and occasionally in the intestinal mucous membrane. It results from the formation and profuse growth of cellular tubercles on a free surface. A vascular connective-tissue growth develops simultaneously with the tubercles, the result being the production of tumour-like masses of various sizes. These, by encroaching on each other, may form excrescences of large size and weight, covering extensive surface areas. Degenerative changes occur, such as caseation and partial calcification.

4. *Tuberculous Cheesy Infiltration, or Infiltrated Tubercle.*—The lungs and lymph glands are the organs chiefly affected by this form of tuberculous lesion. It results from the fusion of several small foci of infection; the latter may not be visible to the naked eye. Microscopical examination of these small foci in the lungs shows that they are composed of scattered centres surrounded by areas in which the alveoli are filled with exudative products and proliferation of the alveolar epithelium. Caseation occurs, due to the action of the bacilli; generally only small groups of lobules undergo this change, but occasionally an entire lobe, or the greater part of the lung, may be so affected. Ultimately the affected area is converted into a dense caseated mass of a yellowish-white or a deep yellow colour. When the lymph glands are affected by tuberculous infiltration, they may form very extensive masses.

5. *Tuberculous Ulcers* occur especially in the intestine, larynx, and bronchi. They result from the disintegration of parts which have undergone caseation, and also from purulent softening of tubercles on the mucous membrane. In the intestine the ulcers show crater-like surfaces. On the base and margins of the ulcers minute tubercles are found. These lesions are common in the dog and cat, and occur especially on the skin in these animals.

6. *Tuberculous Cavities, or Vomicæ,* are met with in the lungs, when a large extent of the pulmonary tissue is destroyed and converted into caseous masses; the latter undergo central softening, and may be coughed up by the animal, an irregular cavity resulting. Vomicæ are not so frequently met with in animal tuberculosis as in the human form of the disease, the reason probably being that animals are not permitted to live sufficiently long for the formation of this lesion.

We may remark that the anatomical forms of tuberculosis which we have mentioned may be combined in a variety of ways, and special modifications may occur depending on peculiarities of the tissues involved in the different animals.

We shall again refer to these points when considering the morbid anatomy of the individual organs affected.

Modes of Dissemination of Tubercle Bacilli in the Body.—A knowledge of this subject is essential in order to understand the manner in which the generalised form of the disease occurs. The primary lesion is the process which arises immediately at the point of entrance of the infection and in the vicinity of this point, without the co-operation of the circulation. From this primary lesion bacilli usually escape, most frequently by the lymphatics at its periphery. These bacilli are probably carried by the lymph-stream to the nearest lymphatic gland; here they are arrested, and produce a new lesion and focus of infection. Bacilli from this new lesion may, after a time, escape, and, reaching the next lymphatic gland, become arrested. When bacilli have reached the last of the glands on the path of invasion, they may be carried into the thoracic duct or into the right lymphatic duct, and so gain entrance to the venous system and to the general circulation. In the blood-stream they usually become arrested in the capillaries of various organs, but chiefly in the lungs.

Generalised tuberculosis occurs in this manner. It is evidenced by the development of a large number of miliary tubercles in the lungs, and a smaller number in the liver, spleen, and kidneys. An important point to remember is that generalisation only occurs in a minority of cases, also that the commonest method by which bacilli spread throughout the body is by way of the lymphatics. It may be regarded as a definite rule, with few exceptions, that when an organ is affected with tuberculosis, the neighbouring lymphatic glands become involved.

There are other modes of dissemination of the tubercle bacilli in the system. For example, in the lungs the bacilli escaping from an existing focus into a bronchial tube may, by the act of coughing, reach a larger bronchial tube, and then, by aspiration, be drawn into another ramification of the bronchi. Or the bacilli may reach the trachea, and be drawn back into the opposite lung.

Again, the pleuræ may be infected from the pulmonary surface by the bacilli spreading therefrom during the respiratory movements. The peritoneum may become infected by extension of the disease from the intestines, during peristaltic movements of the latter. In the alimentary canal infection may take place by means of expectorate containing bacilli being swallowed.

Primary and Secondary Foci.—*Primary Foci* include the lesions which arise at the point of entrance of the bacilli from outside the body, and also those in the vicinity of the infected part, the bacilli being carried to the latter by means of the lymphatics, and not by the circulation.

To primary foci belong all primary lesions in mucous membranes and tubercles in their vicinity, also affections of the corresponding lymph glands and foci in serous membranes arising by extension of the original lesion.

Tubercle bacilli, according to some authorities, do not regularly produce tubercular lesions at their point of entrance into the body, as they are capable of passing through the epithelium of mucous membranes and causing lesions first in the neighbouring lymph glands. The point of entrance of the bacilli into a tissue is said to usually occur in regions where lymphatic glands are more or less exposed, such as the pharynx and intestine, and the point of infection may escape being involved if the bacilli are carried onward in a rapid manner by the lymph, the lesions appearing at first in the lymph glands.

According to some authorities, feeding experiments carried out in pigs with tuberculous material show that the primary lesions occur in the mesenteric lymph glands, while the intestinal mucous membrane may escape infection.

Authorities who believe that the commonest mode of infection in tuberculosis is by ingestion attempt to substantiate this view by the results of feeding experiments, and state that in animals fed on tuberculous material the lungs may become the seat of primary tuberculosis, the bacilli being carried thereto by means of the chyle vessels, thoracic duct, and circulation. McFadyean, however, has shown* that the conclusions of Calmette, Guèrin, and Whitla are unsupported by the experiments they themselves carried out, and has also quoted numbers of feeding experiments which have given directly opposite results to those of the authorities named, and demonstrated that primary pulmonary tuberculosis can with certainty be determined by the inhalation of tubercle bacilli (see p. 119).

Secondary Foci.—In these, infection occurs through the agency of the blood-stream. By some writers they are regarded as hæmatogenous embolic foci, and they are found in internal organs at points where arteries branch into capillaries in the interstitial connective tissue. Tubercles of large size may result, or conglomerations of smaller growths; they are sometimes termed "embolic tubercles." In the lungs both primary and embolic tubercles may occur.

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* See *Journal of Comparative Pathology and Therapeutics*, September and December, 1910.

Local and Generalised Tuberculosis.—*Local Tuberculosis*, in a broad sense, indicates that the extension or distribution of the disease takes place through the lymphatics, and that the general circulation is not concerned in the dissemination of the bacilli. In cattle, tuberculosis has a pronounced tendency to localisation; this is attributed by some to a prompt filtering action of the lymphatic glands, and also to the fact that the tuberculous products in these animals are, as a rule, poor in bacilli.

Generalised Tuberculosis.—In this form the general circulation is said by some authorities to become the carrier of the bacilli. Penetration of bacilli into the systemic circulation is said to occur by means of veins becoming affected by tubercular processes, or the bacilli from tuberculous lymph glands may reach the venous system by way of the thoracic duct or right lymphatic duct, as already mentioned.

According to Weigert, when large numbers of bacilli enter the blood, a tuberculous condition of the walls of a bloodvessel or of the thoracic duct is present, and accounts for this phenomenon.

Two principal forms of generalised tuberculosis are recognised by some authorities:

1. A slight infection of the blood, leading to the formation of isolated tubercles in various organs. These small tubercles usually grow to a larger size, and caseous foci appear, the condition being one known as *chronic generalised tuberculosis*.

2. In this form there is an extensive infection, which leads to the occurrence of innumerable tubercles in the majority of organs. This gives rise to the condition known as *acute miliary tuberculosis*.

McFadyean, however, points out, as the result of numerous experiments, that even when large numbers of tubercle bacilli are thrown into the blood-stream embolic tubercles of the lymphatic glands are not set up, and that it is hardly likely in natural cases of the disease for much larger numbers of bacilli than those introduced experimentally to find their way into the blood-stream; otherwise the resulting pulmonary tubercles would be so numerous as to cause death in a short time, and it would be impossible to explain the presence of macroscopic lesions in the lymphatic glands. He points out also that in generalised tuberculosis the kidneys, liver, and spleen, or all of these, may be free from macroscopic lesions, although the lungs contain myriads of embolic tubercles visible to the naked eye. The macroscopic lesions in the organs mentioned, and also in the lymphatic glands, are generally due to lymphatic infection, and give no evidences of generalisation by way of the blood-stream. In some parts of the body at least the tubercle bacilli can be readily carried by the lymphatic vessels in a centrifugal direction—*i.e.*, in a direction contrary to what is believed to be the normal flow of the lymph-stream. These remarks do not apply to the congenital form of the disease, as the circulation differs in the fœtus from that of the adult. The evidence of generalisation is the presence of tubercles of approximately equal size scattered throughout the entire spongy lung tissue. If the lungs are removed from the carcass prior to inspection, reliable evidence of generalisation cannot be obtained.*

The various organs do not show a uniform predisposition to become infected by the bacilli. Certain organs appear to offer favourable conditions for the growth of the bacilli, and are frequently infected; others are seldom attacked. Thus the lungs, lymph glands, and general lymphatic tissues, are the favourite seats of the disease. The lungs are especially exposed to infection by the inhalation of tubercle bacilli, and in a secondary manner by bacilli entering the venous system from the thoracic duct, and becoming lodged in the pulmonary capillaries. The liver is liable to infection from the entrance of bacilli by way of the lymphatics, and also by the portal vein. According to Kitt, the voluntary muscles show the greatest degree of resistance to the disease, and it is generally only the intermuscular lymph glands that are affected.

Sequence of Organs affected in Generalised Tuberculosis.—In generalised tuberculosis in cattle a certain sequence of the various organs involved is observed. Thus the bronchial and mediastinal lymph glands, the lungs and liver, are generally those first involved, next the spleen and kidneys, followed by the prescapular and inguinal lymph glands, and the udder, bones and joints.

In females, when the posterior region of the peritoneum is affected, the uterus is nearly always involved. In generalised tuberculosis in young cattle up to about four years of age the spleen is generally affected, while the kidneys are free from the disease. On the other hand, in older animals the kidneys are more often affected, while the spleen escapes.

In pigs the lungs, liver, spleen, and kidneys generally become affected simultaneously, and lesions of bones, especially the vertebral column, are more frequently met with than in cattle.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xii.

I. TUBERCULOSIS IN CATTLE.

Lesions of Special Organs.

Lymphatic Glands.—The most frequently affected are the bronchi and the mediastinal glands, and these are regarded as the commone seats of tuberculosis in cattle. They are affected not only in conjuncti with pulmonary tuberculosis, but also when no macroscopic lesions ca be detected in the lungs.

The pharyngeal and mesenteric glands are also frequently affecte while other groups suffer less severely. The lesions consist at first congestion, with more or less infiltration and swelling, followed by tube cular induration and enlargement. By aggregation growths of large si may be produced. On section a tuberculous lymphatic gland may sho all the various stages of tubercle degeneration. The lymph glands i connection with the lungs and intestines may show evidences of tuberc losis, although no macroscopic lesions can be detected in the orga mentioned. It is believed that the bacilli may pass through these orga and reach the glands without establishing centres of disease in the forme

Amongst other lymph glands that may become affected in additio to those mentioned are the hepatic, sublumbar, inguinal, subdorsa phrenic, intercostal (especially the first and second), parotid, submaxillary, prescapular, precrural, prepectoral, axillary, and popliteal, the las two being very rarely involved.

The infection of lymphatic glands usually takes place by mean of bacilli entering them by way of their afferent vessels. In some case the bacilli enter in the opposite direction; thus the precrural and supra mammary glands, and even the udder itself, may become infected fro the peritoneum by this means.

Lungs.—Next to the bronchial and mediastinal lymph glands, the lung are the commonest seats of tuberculosis. The usual form of pulmonar lesion is that of a chronic tubercular broncho-pneumonia, which is characterised by a tendency to early caseation. By the aggregation and confluence of miliary tubercles, caseous masses of various sizes are formed and a large extent of pulmonary tissue may be thus destroyed. Sometimes central softening occurs in these masses, and a communicatio may be established between them and a bronchial tube; in such instance portions of the masses may be coughed up by the animal, and irregula cavities, termed "vomicæ," are formed in the lungs.

Chronic indurating inflammatory processes may occur in the pulmonary interstitial tissue, especially in the vicinity of the caseous foci. The caseous nucleus may become calcified in its outer portion and invested by a firm fibrous envelope, forming what is known as a "closed lesion"; such a one may not extend further in the pulmonary tissue.

Extensive abscesses may be found, containing thick caseo-purulent material, which is odourless; when, however, the abscess gains entrance to a bronchial tube, a mixed infection results, and the pus assumes an offensive character.

Another form of pulmonary lesion not so frequently met with consists of the presence of large numbers of miliary tubercles diffused over a considerable portion of one or more lobes. This lesion is termed "diffuse miliary tuberculosis," and is acute in character. The tubercles occur both on the surface of the lung and also on the surface of the section. Later on degenerative changes occur, and several of these miliary tubercles may by aggregation form large tubercular growths, which in advanced cases also undergo caseation and calcification.

The bronchial mucous membrane shows chronic catarrhal changes, and dilatation of the bronchi is not uncommon. Ulcers and miliary tubercles may occur in the bronchial mucosa. A characteristic feature in pulmonary tuberculosis is the presence of lesions of different ages, from the initial miliary tubercle to the caseous or calcified mass. In some cases the recent lesions may be due to inhalation of the infective material from an old lesion into the deeper parts of the lung by way of a bronchial tube that communicates with the original lesion.

Pleuræ.—This structure is involved in a large number of cases, infection taking place either from the pulmonary tissue or from an affected thoracic gland. But the pleura may be affected primarily, through infection by the blood, or by extension of the disease from the peritoneum through the lymphatic vessels of the diaphragm. The tendency of the disease to involve the pleuræ and peritoneum is one of the marked characteristics of bovine tuberculosis.

According to Friedberger and Fröhner, the lungs and the serous membranes are simultaneously affected in about half of all the cases of tuberculosis, the lungs alone in about one-third, and the serous membranes only in about one-fifth of the cases.

The essential lesions in the pleuræ consist in a chronic dry pleurisy, leading to the development of excrescences on the membrane, which are popularly known as "grapes." These are at first connective-tissue outgrowths of a rather vascular nature, growing over the parietal and visceral layers of the pleuræ. Later on cloudy points appear in them and caseous foci are formed. These tubercles are also characterised by the presence of a well-delimited connective tissue. In size they are very minute at the commencement; in colour a light grey, and translucent in appearance; and they induce a granular condition of the surface of the pleuræ. A number of these tubercles become confluent and form growths of various sizes both on the visceral and parietal membranes, at first being of a soft consistence, but later on becoming firmer and presenting an uneven surface. Caseation or calcification may occur in their centres, and they become a bluish-grey colour. By grouping together they assume various forms, some presenting the appearance of warty or cauliflower growths, others being of a pedunculated or polypoid shape. They may attain a very large size, and cover the entire pleural surface. The term *perlsucht* (pearl disease) is applied in Germany to the tubercular lesions of the pleuræ and peritoneum.

Tongue.—This is occasionally affected, the lesion consisting of an irregular swelling due to the formation of tubercles in the organ. Caseation may occur in the centre of the swelling, and ulceration has also been observed.

Larynx.—The lesions may consist of miliary tubercles and tubercular ulcers on the laryngeal mucous membrane. In some cases the lesion may be in the form of a tumour in the larynx, or the submucous tissue may show infiltration or abscess formation.

Pharynx.—The pharyngeal mucous membrane may show infiltration, swelling, and sometimes ulceration, and the pharyngeal glands are frequently affected. The retropharyngeal glands are a favourite seat of the disease, the lesions consisting of induration, and in some cases large abscesses are formed, the pus having the appearance of custard and the abscess cavity having firm walls.

Occasionally the lateral pharyngeal, the intraparotideal, and the submaxillary lymph glands, are involved.

Alimentary Canal and Associated Lymph Glands.—In this region the usual seats of the disease are the small intestine and the mesenteric glands.

The lesions may be primary, and due to the ingestion of food containing tubercle bacilli; or they may be secondary, and result from the animal swallowing the expectorate derived from his own tubercular lungs. As already pointed out, the mesenteric glands may be involved without any lesion being discovered in the intestine. The intestinal lesions usually consist of ulcers, which may extend along the lines of the lymph glands.

According to some observers, the large intestine may also be involved, but not so frequently as the small bowel. Others state that the large intestine is more commonly affected.

Lesions of the interior of the stomach are rare, but are of comparatively frequent occurrence on the peritoneal surface of the first three divisions of this organ. They consist of rounded tubercles of various sizes.

Peritoneum, Mesentery, and Omentum.—The lesions resemble those described as occurring in the pleuræ. When the pleura is first involved, the disease may extend to the peritoneum through the lymph vessels of the diaphragm.

Liver.—This organ is a comparatively common seat of the disease, especially in advanced or in generalised cases. The tubercles are often numerous and of large size, so that the organ is greatly increased in bulk and weight. By direct infection from the peritoneum, tubercles are formed on Glisson's capsule, and the lymph glands in the vicinity of the viscus are also involved, while abscesses may form in some instances.

The comparative frequency of tuberculous lesions in the liver is said to be from 28 to 54 per cent.

Spleen.—In adult cattle lesions in the spleen substance are comparatively rarely met with ; the peritoneal covering, however, is often involved. In younger animals the lesions consist of a uniform swelling of the Malpighian corpuscles and the development of tubercles in the lymph follicles and in the perivascular connective tissue (Friedberger and Fröhner). The comparative frequency of splenic lesions is stated to be about 19 per cent., and of the spleen capsule about 21 per cent.

Kidneys.—Miliary tubercles may appear in these organs, not only in cases of generalised tuberculosis, but also when there are extensive lesions in the abdominal cavity. In the latter instance infection occurs by way of the lymphatics. The organs are enlarged, the tubercles vary in size, undergo caseous degeneration in their centres, and become surrounded by a capsule of connective tissue. The tubercles are said to develop in the renal connective tissue, and the glandular structure of the organ may be destroyed by the encroachment of the lesions. Evidences of parenchymatous or hæmorrhagic nephritis may be observed. In some cases an abscess may be present. The capsule of the kidney may be involved in some instances.

The comparative frequency of renal tuberculous lesions is said to be from 0·07 to 5 per cent.

Ovaries.—These organs, when affected, are greatly increased in size ; they show rounded projections, become indurated, and on section display the usual forms of degeneration.

The Fallopian tubes may be obstructed by tubercular proliferations.

Uterus.—Uterine tuberculosis is said to occur in about 3 per cent. of advanced cases of the disease. The tubercles may be situated on the serous covering, in the subserous intermuscular connective tissue, or in the submucous connective tissue, also in the broad ligaments. The uterine mucosa may show the presence of ulcers, and sometimes degeneration of the cotyledons may be observed, these structures having a caseous appearance and being very friable. Occasionally tubercular degeneration of the placenta may be present. A muco-purulent fluid, rich in tubercle bacilli, may be found in the interior of the organ.

The bladder and vagina may be involved in some cases. The vulva may show tubercles and ulcers.

Testicles.—These organs are occasionally affected. The lesions consist of tubercles of various sizes; caseous foci, swelling, and induration, may also be observed.

The tunica vaginalis may be involved, with hydrocele as a complication; the epididymis, vas deferens, and prostate gland, may also be implicated.

Penis.—This organ is rarely affected. A case of primary tuberculosis of the penis is recorded by Eber in 1891. The connective tissue surrounding the corpus cavernosum was involved, also the prepuce.

Udder.—The lesion is in the form of a diffuse interstitial inflammation of the gland substance. Well-defined tubercles are only met with in very advanced cases. The disease usually commences in one of the posterior quarters of the gland, and generally at the upper part of the quarter.

As a rule, it commences in an insidious manner, and progresses slowly; the affected quarter or quarters become enlarged and indurated, and in far-advanced cases this enlargement may be very marked. Occasionally the disease assumes more acute characters; then the hypertrophy is more rapid and more marked. In the early stages, and even after the disease has made considerable progress, the secretion of milk is not interfered with; but after a variable period the milk is lessened in quantity and inferior in quality. Later on it becomes scanty in amount, resembles whey in appearance, and contains flocculi and stringy materials.

At the stages during which the milk may appear normal in appearance, tubercle bacilli may be discovered in it, while in the later stages it contains these bacilli in abundance. On section, the affected quarter is found to be firm to the touch and to the knife; the cut surface is rough on palpation, and presents the appearance of coarse lobulation.

On microscopic examination, the interacinous spaces are found to be increased in breadth by the newly-formed cells, which are chiefly epithelioid in character. Later on the acini are compressed by the new material formed; hence they become atrophied, and are ultimately destroyed. As the process proceeds, giant cells in varying numbers are formed in addition to the epithelioid cells, and degenerative changes such as necrosis and caseation occur. In the newly-formed interstitial tissue and in the epithelium of the acini and ducts tubercle bacilli are present, and may also be found in a free state in the lumen of the ducts. In some cases tubercular ulcers may be present in the larger ducts.

The supramammary lymph glands are usually involved, and in some instances they may be affected prior to the occurrence of lesions in the udder.

The udder becomes involved (*a*) as the result of a generalised form of the disease; (*b*) by way of the lymphatics from an affected peritoneum to the supramammary lymph glands, and thence to the udder; (*c*) occasionally as the result of a local infection, the bacilli gaining entrance through the teat or through an abrasion of the skin of the udder.

Tuberculosis of the udder is more common in the cow than in any other species. The affection has also been met with in the sow.

The frequency of tuberculosis of the udder in tuberculous cows is stated by some observers to be from 2 to 3 per cent.; in the Dublin dairies it is computed as being about 1 per cent. amongst tuberculous cows; in Germany it is also stated to be 1 per cent. In Denmark 0·5 per 1,000 of all cows are, according to Bang, affected.

Pericardium and Heart.—The pericardium may be involved in far-advanced cases of the disease, by extension of the process from the pleura or from the bronchial glands. The frequency of the lesion in tuberculous cows is said to be about 5 per cent. According to some observers, this structure may be independently affected.

The myocardium may be involved by extension of the disease from the pericardium; the lesions of the heart wall are generally diffuse, distinct tubercles being rarely found in this situation in adult animals. Some observers state that the heart may be the seat of primary tuberculosis in some instances.

Brain and Spinal Cord.—The lesions usually take the form of tuberculous meningitis, and may be acute or chronic. This form occurs especially in young cattle; tubercles of various sizes and ages appear on the meninges, and set up inflammation in these structures. The pia mater at the base of the brain is chiefly affected. In the acute form there is usually a turbid fibrino-purulent exudate, and the presence of small whitish tubercles on the membranes. In some instances the tubercles may be difficult to discover, and the amount of exudate does not bear a definite relation to the number of tubercles present. The cerebral bloodvessels may be involved, especially the smaller arteries, the lesions appearing as nodular enlargements ; and the cerebral substance may be more or less œdematous and infiltrated with leucocytes. The lateral ventricles may be dilated and contain a turbid fluid. The cerebellum is involved in some cases.

In a typical case of tubercular meningitis in a heifer, recorded by Ainsworth Wilson,* the lesions occurred in connection with those of generalised tuberculosis. " Miliary tubercles were scattered over a considerable area of the pia mater, especially on the corpus callosum between the right and left cerebral hemispheres, and on the cerebellum. The crura cerebri were studded all over with greyish firm tubercles, varying in size from a millet-seed to a hemp-seed. The tubercles were most marked, however, on the surface of the cerebellum ; the latter, on section, showed intense congestion in the neighbourhood of the fourth ventricle. . . . No fluid was present in the cranial cavity or in the meninges." According to Friedberger and Fröhner, the tubercles in the pia mater at the base of the brain may remain for a considerable time without producing any further inflammatory changes ; also the tubercles may occasionally unite to form large masses.

In the spinal cord tubercles may be found in the dura mater and pia mater, and these membranes may be adherent to each other ; the lumbar enlargement of the cord is said to be the commonest seat of the lesions. The comparative frequency of lesions of the nervous system in tuberculous cattle, according to German observers, is, for the brain, 0·04 per cent., and for the spinal cord 0·03 per cent.

Eyes.—Occasionally tubercular lesions occur in the cornea, sclerotic, iris, choroid, and retina. Degenerative changes, such as necrosis and caseation, are induced, and the entire organ may ultimately be converted into a caseated or granulating mass.

Muscles.—Even in very advanced or in generalised cases lesions are very rarely found in the voluntary muscles. When they do occur, the disease probably extends from lesions of bones or joints to the muscles. The tubercles are of various sizes, and may occur in or between the muscular masses ; in some instances they show caseous centres. More commonly the lesions are found in the intermuscular lymph glands, and not in the muscles themselves. The muscles chiefly affected are those of the croup, abdomen, and thorax.

Bones and Joints.—In cattle, tuberculous lesions of bones are only met with in a small porportion of cases—about 0·4 per cent., according to German authorities ; other observers state that the proportion is 1 per cent. The bones of the vertebral column are those generally attacked ;

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* *Veterinary Record*, April 29, 1911.

occasionally the ribs, sternum, petrous temporal, frontal, and occipital bones, may be affected. The disease usually starts from the medulla of the bone close to the articular extremities, or in the vicinity of the lines between the epiphysis and diaphysis; a considerable exudate is thrown out, and thickening of the bone, complicated with arthritis, may result.

Caries with central caseation and the formation of cavities may occur in the bone, and detachment of epiphysis and apophysis has been observed.

Lesions of the joints are not common; tubercular processes may occur in the cartilages and ligaments; softening, caseation, or fibroid changes, are induced, and synovitis results. The knee and hock joints are said to be the more common seats of the disease; the tendon sheaths of the extensor muscles of the forearm may be involved. Occasionally tubercular lesions are met with in the nasal septum and in the cartilages of the ear (Friedberger and Fröhner).

Skin.—Small tubercles may occur in the substance of the skin or in the subcutaneous tissue, their location being usually on the side of the abdomen. In other instances the lesions consist of enlarged masses, forming raw, warty projections with caseated centres; or such projections may occur in clusters in the form of grapes.

According to Moussu, tuberculous lesions of the skin are very rare; they consist of small, hard swellings beneath the skin, varying in size from a hazel-nut to a walnut, and containing caseous or calcareous material; they may be found grouped together within certain areas, or irregularly distributed over the entire body, but especially towards the base of the tail. They have no connection with the superficial lymphatic glands.

Lesions in Congenital Tuberculosis.—In cases of congenital tuberculosis the situation and distribution of the lesions show some important differences as compared with those occurring from the ordinary means of infection and in adult animals. The bacilli in the congenital form are always distributed by the blood-stream, and, in consequence of the arrangement of the fœtal circulation, the blood first enters the liver; also a large volume of blood from the posterior vena cava enters the systemic circulation without first passing through the lungs.

The result of these peculiarities is that when the blood of the umbilical vein contains tubercle bacilli, the liver and its lymphatic glands are the chief seats of the lesions, while the number of tubercles in the lungs is generally small. Tubercles are found very frequently in the substance of the spleen, and may also be present in the cardiac muscle and in several groups of lymphatic glands, including the popliteal and other peripheral glands.

SYMPTOMS OF TUBERCULOSIS IN CATTLE.

The disease is usually insidious in its development, and slow and chronic in its course.

In the initial stages its presence may be overlooked, and not uncommonly well-marked and advanced lesions may be found in slaughtered animals that appeared perfectly healthy during life.

As the pulmonary form of the disease is most commonly met with, the symptoms, as might be expected, are chiefly in connection with the respiratory system.

In *the early stages*, and in those cases that appear to continue for an indefinite period without developing distinct signs of the disease, there are certain symptoms that may be present, although they cannot be regarded as by any means diagnostic. These symptoms are as follows: A short, dry, intermittent cough, easily induced, and especially observed when the animal first leaves its stall, particularly in cold weather, or when it is made to rise suddenly, or if driven for a short distance.

The respirations may be accelerated to a greater degree than is normal when the animal is forced to walk for some distance, and in some instances this is observed even during repose. Beyond these trivial symptoms, the animal may appear in good health, and may readily fatten. In other instances careful percussion and auscultation may reveal circumscribed areas of dulness and evidences of crepitation or wheezing sounds over the affected areas. These areas are usually isolated and circumscribed. A physical examination of the chest, however, is attended with considerable difficulty in the ox, in consequence of anatomical peculiarities and the sounds set up by the rumen.

A slight elevation of temperature may be observed at intermittent periods, but this is a symptom of no diagnostic value.

In *more advanced cases* the animal is "hide-bound," and there is usually loss of condition; but cases are met with in which the general condition has been good, and on the animals being slaughtered for food, extensive lesions may be discovered.

The cough becomes more frequent, and may be short, dry, or paroxysmal in character, or it may be moist. The alterations in the respiratory sounds become more marked, and vary according to the lesions present. The appetite may be lessened, and the milk in the case of a dairy cow may in some cases become altered as regards quality and quantity, while in others no alterations are observed in the early stages. Nasal discharge is not observed, as the expectorate is usually swallowed when it reaches the pharynx, or may be discharged through the mouth in the act of coughing; it contains tubercle bacilli. Other symptoms depend on the involvement of various organs. Thus, attacks of tympanites occurring after feeding depend on involvement of the glands in connection with the œsophagus or of the bronchial glands; these press on the œsophagus, and interfere with the eructation of gases from the rumen; or they may press on the vagus nerve, and interfere with the normal movements of the stomach.

When the disease becomes generalised, abdominal symptoms may be observed, such as diarrhœa, and enlargement of the superficial lymphatic glands may be noted.

In the later stages of the disease the animal shows marked wasting, accelerated respirations, frequent fits of coughing, sunken eyes, anæmia, and general debility. The temperature is generally raised to 104° F. or 105° F., and evidences of generalised tuberculosis, such as enlargement of superficial lymphatic glands and diarrhœa, become more marked. Auscultation of the chest reveals the presence of complex morbid sounds depending on the lesions that are present.

At any time an acute attack of pneumonia or pleurisy may develop in a tuberculous animal; in such instances the respiratory symptoms will be greatly exaggerated.

The superficial lymphatic glands may be involved in any region of the body, and show swelling and induration; in some cases large tubercular abscesses are formed, a favourite seat of these being in front of the stifle.

In cases of abdominal tuberculosis, absorption and assimilation are interfered with; there is marked loss of condition, and evidences of digestive disorder, such as intermittent attacks of tympanites after feeding, also diarrhœa. It is said that in some cases rectal exploration may reveal the presence of tubercles and enlarged glands on the rumen, in the mesentery, and in the sublumbar and subsacral regions.

When the genital organs in the female are involved, nymphomania and sterility may result. In cases where sudden generalisation occurs from an old bronchial or mesenteric lesion, in a pregnant cow, she frequently aborts. Chronic metritis may be observed, accompanied by a purulent discharge, which is fœtid in some instances. This discharge may be greyish-yellow in colour, grumous, or granular, and may accumulate in the depressions of the vagina.

In the male the testicle may show a tubercular swelling, which may also involve the epididymis. Hydrocele has been observed in some cases.

In tuberculosis of the udder we find a firm, hard, painless swelling of one quarter (usually one of the posterior quarters of the organ), and occasionally two quarters are involved. The secretion of milk is not at first interfered with, but later on it becomes thin and watery, and may contain flocculi, due to semi-coagulation. Tubercle bacilli are often present in the milk, but not invariably, and a diligent microscopical examination may fail to detect them even in cases where the milk has proved to be virulent by inoculation into guinea-pigs.

The affected quarters finally become indurated to a marked extent, and the disease may extend to the anterior quarters of the udder. The supramammary lymph glands become involved; they are swollen, and finally undergo caseation or calcification. The superficial inguinal glands may also become affected. According to Moussu, the lesions in the udder are generally diffuse, and show a tendency to hypertrophy and the formation of fibrous tissue and sclerosis. In time the entire secreting structure undergoes diffuse suppuration; fibro-caseous masses form in the depth of the tissues, and may undergo softening, deep-seated tuberculous abscesses being produced. Moussu also states that when the infection of the gland is slight the effects may escape notice for weeks, or even months, and the animal may only appear to suffer from subacute or chronic mammitis. In some cases the udder is greatly increased in size. He finds that the supramammary glands are always involved before the udder is seriously attacked.

In laryngeal tuberculosis, when the lesion is in the form of a tumour, a "roaring" sound is emitted when the animal is moved, or when placed in a warm cowshed.

In pharyngeal tuberculosis, with involvement of the retropharyngeal glands, respiration is accompanied by a "snoring" sound similar to that occurring in actinomycosis of the pharyngeal region. When suppuration occurs in these glands, extensive circumscribed abscesses may result; manipulation of the external pharyngeal region may show enlargement of the part, the head is protruded, and the "snoring" or roaring sound during respiration is very marked. Examination of the upper region of the pharynx by manipulation through the mouth will detect the presence of a fluctuating swelling.

Pharyngeal tuberculosis usually terminates in the generalised form of the disease, but we meet with cases in which it remains as a local condition for an indefinite period, so far as can be judged by the symptoms presented.

In tuberculosis of the brain and meninges, evidences of excitement, convulsions, or vertigo, are observed, succeeded by paralysis and coma. Amaurosis is a common symptom, and a staggering gait or a tendency to move in a circular direction may also be observed. In a typical case of tubercular meningitis recorded by Ainsworth Wilson, to which we have previously referred, the prominent symptoms were: "Head held at various angles—high, low, on one side; boring in corner of stall; periods of excitement, during one of which the animal got cast in the manger; pupils widely dilated; blindness; vessels of the retina and conjunctival vessels markedly injected; frequent rotatory movements round the box, mostly from left to right; high stepping with fore-feet; difficulty in rising; nose protruded, and neck held stiffly in turning; staggering gait; occasional sweating; twitching of facial muscles; pupils contracted later; inability to rise; convulsive movements of the limbs; and opisthotonos." When the spinal cord is involved, locomotion is interfered with, and finally paralysis results.

In tuberculosis of bones and joints the symptoms vary according to the location of the lesions. When the cervical vertebræ are affected, stiffness of the neck is observed; and when the dorsal vertebræ are involved, a staggering gait and difficulty in turning may be observed. Tuberculosis of the large joints of the limb is manifested by a hard, persistent swelling, and by evidences of acute synovitis.

DIFFERENTIAL DIAGNOSIS.

This is conveniently considered under the following headings:

A. Clinical Diagnosis.
B. Bacteriological Diagnosis.
C. Diagnosis by the Use of Tuberculin.

A. Clinical Diagnosis.—The majority of practitioners admit that, with the exception of far advanced cases of the disease, it is not possible to arrive at a definite diagnosis by the observation of symptoms or by a physical examination of the case. Moreover, we cannot by clinical methods alone determine whether a given animal is free from tuberculosis or otherwise. In cases termed "clinically affected," in which marked emaciation, cough, and evidences of chronic pulmonary disease, are present, these symptoms, taken in conjunction with the others mentioned on p. 141, and with the history of the case, may enable us to give a positive diagnosis.

As already pointed out, considerable lesions may be present without giving rise to any symptoms which would lead us to suspect the animal to be affected with tuberculosis.

On the other hand, we see cases which present symptoms suggestive of the disease, but on post-mortem examination tuberculous lesions are absent; also we meet with similar instances where the application of the tuberculin test proves that the diagnosis was erroneous.

Again, the tuberculin test demonstrates how frequently tuberculosis exists in apparently healthy cattle.

The differential diagnosis of tubercular mastitis is of special importance, and it must be admitted that considerable difficulty is often experienced in dealing with the subject during the later stages of an attack of acute mastitis, when the affected quarter of the udder is hard and swollen and the milk altered in its physical characters. Also, in cases where the mastitis commences as a chronic interstitial inflammation, the condition simulates that of the tubercular form. The employment of tuberculin will not definitely decide the question, as a reaction will result provided tuberculous lesions are present in any region of the body. A differential diagnosis must be arrived at by the employment of bacteriological methods (see p. 145).

Differential diagnosis by means of a harpoon or trocar was adopted by Walley, Nocard, McFadyean, and Ostertag. In this method a small portion of tissue is removed under aseptic precautions from the affected quarter by means of a special harpoon, and examined microscopically for the presence of tubercle bacilli. It may happen, however, that the lesion may be tuberculous, and yet it may be difficult to demonstrate the presence of bacilli, or they may be absent from the portion of tissue removed.

In the differentiation of tubercular mastitis from ordinary mastitis by clinical methods, the history of the case and a consideration of certain phenomena will prove of assistance. The tubercular form is insidious in its onset, and never arises suddenly like ordinary mastitis. Its painless character on manipulation, the absence of heat, its chronic nature, the non-occurrence of resolution, and the steady increase in volume and density of the affected quarter, are strongly suggestive of tubercular characters.

With reference to the statement that the quantity and appearance of the milk are not appreciably altered at the commencement of the attack in tubercular mastitis, we have observed that it is not unusual to find the disease much more progressive in some cases than in others, the affected quarter increasing in size to a corresponding degree, and alterations in the quantity and physical characters of the milk occurring far quicker than is usually the case.

Amongst other diseases which may require to be differentiated from tuberculosis, we may mention actinomycosis affecting the udder, and also the presence of actinomycotic lesions of the pharynx. For the differential diagnosis see Actinomycosis.

Jöhne's disease simulates a tubercular affection of the intestines.

The employment of avian tuberculin is now accepted as a diagnostic test for the former disease (see Jöhne's Disease, p. 202).

B. Bacteriological Diagnosis.—The examination of mucus obtained from the trachea of the suspected animal is an uncertain method of diagnosis; moreover, the material is difficult to procure. It is said to be best obtained by swabbing the throat, or by collecting it from the wall, manger, etc., in front of the suspected animal, on which it is ejected by the process of coughing. Some observers have collected the material for examination by holding a sheet of paper before the animal's nostrils during the act of coughing.

The detection of tubercle bacilli in the milk of a suspected animal by microscopical examination is not a satisfactory or reliable means of diagnosis, as the bacilli are not always present in sufficient numbers to be discovered by the microscope. According to Ostertag, Bang, and Nocard, it is only when the udder is affected that there is any probability of the bacilli being present in the milk. This view is not held by all authorities (see p. 195).

The most reliable method to determine the presence of tubercle bacilli in milk is to carry out inoculation experiments with the sediment of the suspected fluid. This method is also adopted in the differential diagnosis of tubercular mastitis. For this purpose guinea-pigs are preferred, and the inoculation test may be carried out by the intraperitoneal or by the subcutaneous or intramuscular method. In the former the milk, if infective, will produce abdominal tuberculosis in from three to four weeks after inoculation; in the latter, according to Ostertag, the period of time required is shorter, being within ten days. The lymphatic glands close to the point of inoculation assume the character of hard, painless nodules, and tubercle bacilli can be demonstrated therein. Some observers have found that the milk from a tuberculous udder may be infective only at intermittent periods.

Amongst other suspected materials which may be employed for inoculation purposes are the vaginal secretion, the uterine discharges, and extirpated lymphatic glands.

In cases of tubercular disease of the intestine a microscopical examination of the fæces may reveal the presence of tubercle bacilli.

C. Diagnosis by the Use of Tuberculin.—Tuberculin is chiefly employed for diagnostic purposes, and the tuberculin test is now recognised as the most reliable means for the diagnosis of tuberculosis.

Tuberculin was first introduced by Koch in 1890 as a curative agent, but without any success. It consists of a concentrated bouillon culture of tubercle bacilli, in which the latter have been killed by heat and afterwards removed by filtration. This preparation is known as "old" tuberculin, in contradistinction to "new" tuberculin, a modified form introduced by Koch in 1897, and prepared by taking young virulent cultures grown in the same manner as in the old tuberculin, separating the bacilli, drying them *in vacuo*, and disintegrating them by means of an agate pestle and mortar. The powder thus obtained may be treated in various ways, so as to form different kinds of tuberculin.

By adding the powdered bacilli to a 10 per cent. solution of caustic soda, stirring and filtering same, a preparation termed "T.A." (alkaline tuberculin) is produced. If, after trituration, the powdered bacilli are treated with distilled water and centrifugalised, the clear fluid decanted has the properties of the old tuberculin, and is known as "T.O." ("O." standing for *obere*, signifying upper).

If the remaining deposit, after the removal of the T.O., be again dried, triturated, treated with distilled water, centrifugalised, and decanted several times, the clear fluids obtained are mixed together, and constitute what is known as "T.R." ("R." signifying residue). Tuberculin R. is said to contain the substances present in the bacilli, which are insoluble in glycerine, and only produce a tuberculin reaction when employed in large doses.

Koch was again hopeful that repeated and increasing doses of T.R. would produce immunity against T.O., and against living and virulent tubercle bacilli. But only a very limited success, if any, could be claimed for T.R. as a remedial agent.

Old tuberculin is the form used for diagnostic purposes in animals, the new tuberculin being employed as a therapeutical agent for the disease in man.

Large doses of tuberculin subcutaneously injected into healthy animals produce in a few hours fever, malaise, and accelerated respirations.

The usual dose for diagnostic purposes in cattle—viz., 3 c.c.—produces no effect in healthy animals, but gives rise to certain symptoms in animals affected with tuberculosis, the most important being a gradual rise in temperature. It is thus a valuable diagnostic agent, and the one generally relied on to detect the presence of the disease.

The following directions are suggested by Sir John McFadyean for the employment of tuberculin:

"1. While under the tuberculin test cattle ought to be kept in the house, fed on their usual food, and protected from draughts. They ought not to be allowed to drink large quantities of cold water between the sixth and eighteenth hours after injection. It is well to take their temperature at least once on the day preceding the test.

"2. The dose of tuberculin for a medium-sized cow is 3 c.c., or 50 minims, and it may be varied above or below that according to the size of the animal. Large bulls ought to receive 4 c.c.

"3. It ought to be injected under the skin with a clean hypodermic syringe. The most convenient points are in front of the shoulder or on the chest wall behind the point of the elbow. The best form of syringe is one with an asbestos piston, as the whole instrument may be sterilised by boiling it in water for five minutes before use.

"4. The tuberculin must be injected into the subcutaneous connective tissue, and care must be taken that the whole dose is introduced.

"5. Ordinarily the temperature must be taken at the time of injection, and at the ninth, twelfth, fifteenth, and eighteenth hours afterwards. When there is any reason to suppose that the animal may have been already tested with tuberculin during the preceding two or three weeks, it is advisable to take the temperature at the third and sixth hours as well as at the times just mentioned.

"6. Animals in which the temperature during the eighteen hours following the injection rises *gradually* to 104° F. or more may be classed as *tuberculous*, and those in which it remains under 103° F. as *not tuberculous*. When the maximum temperature attained is under 104° but over 103° F., the case must be considered doubtful, and the animal may be retested after a month.

"7. The test is not reliable in the case of animals in the last stage of the disease, or in those in which the temperature is over 103° F. before injection.

"8. The tuberculin should be kept in a cool place, and protected from light. Should it become turbid or cloudy, it must not be used.

"9. The tuberculin test does not render the milk in any way injurious."

Tuberculin, besides producing a thermal reaction in tuberculous cattle, may give rise to a systemic reaction, which is manifested within twelve hours after the injection of the agent. A capricious appetite, slight interference with rumination, a staring coat, arching of the back, rigors, diarrhœa, and slightly accelerated respirations, may be observed, especially in milch-cows, but these symptoms only last for a short time. Villar has found that a falling off in the milk-yield usually occurs for twenty-four hours or so, but in cases of severe reaction this diminution may be more marked, and he has several times observed that the milk-yield was diminished by 50 per cent. for some days, while in exceptional instances cows did not return to their normal milk-yield until after the next calving. He also found that the systemic reaction, even in animals badly affected with tuberculosis, is not always shown, and that in some cases, even when the thermal reaction is well marked, constitutional symptoms and interference with the milk-yield may be absent.*

A local reaction to tuberculin administered by subcutaneous injection in cattle is not observed. Ainsworth Wilson, however, informs us that a local reaction may occur in the udder in some cases of mammary tuberculosis. Within twenty-four hours after a hypodermic injection of tuberculin, given in the usual manner, the affected quarter or quarters become swollen and œdematous, there is effusion into the subcutaneous tissue, the skin is hot and pits on pressure, and there is more or less pain on manipulation; the supramammary lymphatic gland is swollen, and its posterior aspect is more or less prominent and painful.

Tuberculosis may be diagnosed with certainty in a suspicious udder when the mild inflammatory phenomena therein are associated with the general test, with or without a rise of temperature. But this local reaction in the udder may be slight or fail to appear. Thus the absence of a reaction in an affected quarter of the udder cannot be regarded as evidence that the gland is not tuberculous.

Villar records a case in which an apparently healthy cow showed a chronic enlargement of one hind-quarter of the udder, and, on being tested, the rise of temperature only amounted to 1·6° F., the maximum temperature not reaching 104° F., so that from the chart she could only be regarded as a suspicious case of tuberculosis; but on microscopical examination of the milk yielded by the enlarged quarter, large numbers of tubercle bacilli were detected therein.

The average normal temperature of cattle which are to be submitted to the tuberculin test should, according to some practitioners, be obtained by taking the temperature every morning and evening for the three days preceding the test. Obviously it would be difficult to carry this out in practice, especially when large numbers of animals have to be treated; hence in most instances we depend on taking the temperature on the day previous to injection and also at the time of injection.

Villar points out the importance of registering the temperature up to the twenty-first hour after injection, as he has found that in some cases the characteristic thermal reaction is not produced until this period is reached. He also draws attention to some other important practical details in connection with the subject, which we quote from the paper already mentioned as follows:

"Ordinarily a temperature which, though risen, has dropped by the twelfth hour would not be a positive or even a suspicious reaction; an infected animal's temperature would not be on the decline at this hour, therefore in practice the first time at which it is usually necessary to take the temperature is at the twelfth hour. . . . In cases where the rise in temperature—for in healthy cattle there is usually some slight rise—has not much exceeded one degree, and has fallen by the eighteenth hour, there is no need to continue the observation; but where the temperature rises to 103° F. at the eighteenth hour, then, in my opinion, we are not justified in leaving the twenty-first hour temperature unobserved. . . . But not all cattle in which there is a rise from a normal initial temperature to 104° F. must be regarded as tuberculous. A sudden rise in the temperature to this point or more, with an equally rapid fall—a sudden jump in the temperature—is usually associated with some other morbid condition than tuberculosis. The ideal chart of tuberculin reaction should have a good curve, the temperature should commence to rise between the sixth and ninth hour, should continue to gradually rise, have some persistence at its highest point, and then gradually fall. . . .

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* Paper on "Tuberculin and Mallein," National Veterinary Association, 1909.

"The evening temperature of yearling bulls and heifers exceeds that of cows; in such, an evening temperature not exceeding 103° F. cannot be regarded as abnormal, and I always without hesitation test such animals; but it is not advisable to test a cow or stock bull whose evening temperature by much exceeds 102° F. The temperature of young calves, especially those fed from the pail, is very inconstant. In a herd of ten, all in apparent health, the evening temperature was found to vary from 99·8° to 104° F. It is well known that from indigestion and other slight causes the temperature of these young animals often varies to an extraordinary extent within twenty-four hours. This erratic tendency of the temperature of calves makes the testing of such animals untrustworthy and undesirable."

Although the tuberculin test, if properly carried out, usually gives accurate results, failures or inaccuracies are occasionally recorded.

Such instances would appear to be more common on the Continent than in Great Britain, as, according to Friedberger and Fröhner, the failures have been estimated at 13 per cent. in Germany and 10 per cent. in Denmark. The so-called failures occur when cases which give a typical reaction to tuberculin, on being slaughtered, show no tuberculous lesions; or when obviously tuberculous animals do not react to the agent; or when a certain animal or animals react at one time, and on being retested in about six months afterwards pass the test successfully; or when an animal certified as being free from the disease according to the result of the tuberculin test, is retested later, and is found to react. These so-called failures or inaccuracies may be explained on different grounds as follows :

1. Certain diseased or abnormal conditions other than those of tuberculosis may be present, or occur during the period of testing, and produce a thermal reaction which may lead to erroneous deductions. Sexual excitement or strange surroundings may sometimes bring about a thermal reaction. Some observers state that animals within three weeks of parturition are unduly susceptible to tuberculin, but Villar found that this is not always the case. Attention to the temperature curve is of importance in obviating erroneous deductions; also care should be taken not to apply the test in cases where the temperature at the time of injection exceeds the limit already stated.

2. Animals may show a very marked thermal reaction and constitutional symptoms as the result of tuberculin, and the lesions may be insignificant and not easily discovered at the post-mortem examination; in some instances the only evidence of disease may be the occurrence of caseation in a small lymphatic gland. Such lesions may easily be overlooked, unless the practitioner conducting the autopsy is an expert in morbid anatomy. This will account for some, at least, of the recorded failures.

3. In far advanced or extensively generalised cases of the disease tuberculin may fail to produce a reaction; some pathologists believe that the probable explanation of this phenomenon is that in the systems of such animals tuberculin is produced and held, and, as a result, they do not respond to the agent when employed as a test.

4. During the period of incubation of tuberculosis the tuberculin test may fail to indicate the presence of the disease. The period of time which elapses between the entrance of tubercle bacilli into the body—*i.e.*, after infection—and the capability of the animal to react to tuberculin is very variable. Experiments have been carried out by McFadyean, Nocard, and others, to determine this period, and, as a result, it was estimated as from eight days to seven weeks.

5. The disease may become arrested, and, according to Villar, it is possible for an animal to even recover from it in a few months if the lesion be slight; hence, in such an instance a reaction to tuberculin may be observed at one period, and later on the same animal would cease to react.

6. Repeated injections of tuberculin produce a degree of tolerance to the agent. This fact is made use of by unscrupulous persons for fraudulent purposes in cases where it is imperative that cattle should go through the test successfully before being permitted to land in certain

countries, or where stock-owners insist on the test being applied to animals prior to purchase.

The plan adopted is to inject and reinject an animal until after a time it will cease to react. Others administer febrifuges and liberal amounts of cold water, and by these means reduce the temperature and prevent the reaction occurring.

Under such conditions, of course, the test may prove misleading. This matter will be again referred to later on.

7. The tuberculin employed may have become deteriorated by age or bad preservation, or it may be useless on account of faulty manufacture, and hence the test fails.

From the above remarks it will readily be seen that, in basing a diagnosis on the tuberculin test, great care is essential in order to prevent erroneous conclusions, and that it is possible for the latter to occur in at least some instances, in spite of all precautions.

In order to secure uniformity of strength and to prevent the fraudulent use of the agent, its manufacture and sale ought to be controlled by the State.

With reference to the tolerance which cattle may obtain to the action of tuberculin, it is both of interest and importance to ascertain the period of time that should elapse before retesting can be carried out, so as to obtain accurate results. This knowledge is necessary in connection with cases in which the first testing gives a reaction of a doubtful character, and it is essential that a retesting should be adopted. Judging by the results of Nocard's experiments in 1892, an interval of from twenty-five to thirty days should elapse between the two injections, so as to insure a typical reaction on the second occasion.

In order to combat the designs of fraudulent dealers and owners, who inject tuberculin a short time previously to the employment of the test, Vallée advised that in cases where this species of fraud is suspected a double dose of tuberculin should be injected, and the temperatures should be taken from the second hour after injection, for every two hours up to the fourteenth or fifteenth hour. By this procedure the reaction will occur in tuberculous animals even when a primary dose has been injected as early as thirty-six or forty-eight hours before the test is applied. The reaction to the double dose is intense, sudden, and transitory, and may disappear after the twelfth hour. Jowett* has adopted this method on several occasions, and found it reliable; he observed that the maximum rise of temperature occurred about the seventh hour after injection.

* *Journal of Comparative Pathology and Therapeutics*, March, 1911.

The usual method of employing tuberculin is by subcutaneous injection; there are, however, several other ways in which various observers have used modified tuberculin as a test for the presence of tuberculosis.

Briefly, these methods may be enumerated as follows: (1) The conjunctival; (2) the cutaneous; (3) the ophthalmo-cutaneous; (4) the epidermal; (5) the intradermic; (6) the mixed methods.

1. THE CONJUNCTIVAL TEST was introduced in 1907 by Calmette and Wolff-Eisner, with reference to the diagnosis of tuberculosis in the human subject, and was applied to cattle by Vallée. Moussu, Lignières, and others, also studied the subject. It is often termed "Calmette's ophthalmo-reaction." A few drops of ordinary tuberculin, prepared without glycerine or carbolic acid, are instilled into one eye, the other eye being untouched for comparison. If the animal be tuberculous, certain phenomena occur in from six to ten hours in the conjunctiva of the tested eye. According to Ainsworth Wilson, who has devoted considerable attention to the subject,* "the conjunctival reaction shows itself in three ways: (1) In opaque catarrhal or muco-purulent discharge; (2) redness and swelling of the palpebral conjunctiva, including the membrana nictitans and caruncula; (3) injection or engorgement of the capillary vessels of the ocular conjunctiva covering the sclerotic, accompanied by a diffuse reddening of the membrane, most marked towards the outer canthus. No. 3 is often slight in the early stages, but it increases in intensity even after Nos. 1 and 2 have commenced to fade. . . . A slight conjunctivitis sets in usually between the fourth and tenth hours after instillation; it seldom appears before the sixth hour, and very exceptionally its onset is delayed for twenty-four hours. There is an increased flow of tears as a rule; the conjunctiva lining the lids, hair, and caruncula, may be uniformly swollen or infiltrated here and there with serum. It is much congested in marked reactions, and usually associated with a more or less profuse purulent discharge, which streams from the inner canthus in a lineal direction down the face. The pus frequently has a yellowish tinge; it soon dries and drops off, or remains crusted on the face. On the other hand, it may persist for three or four days. In slight reactions the three phenomena mentioned above are much less apparent; one of them—*e.g.*, the discharge—may be absent, although close examination will usually reveal a few flakes in the conjunctival sac. . . . A slight opaque discharge or a slight reddening of the conjunctiva occurring alone should be regarded as negative."

As regards the duration of these phenomena, the same observer found that "the positive results were most marked at the fifteenth hour after instilment, a few were fading at that hour, a considerable number culminated about the twenty-first hour, while a small percentage were delayed till the thirty-sixth or forty-eighth hour. In almost every case, however, a definite diagnosis could be made during the twenty-four hours following the instilment of the tuberculin."

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* *Vide Journal of Comparative Pathology and Therapeutics*, March, 1911.

The test produces no constitutional disturbance; it has no effect on the untreated eye, and causes no irritation on the eyes of sound animals except at the time of instillation, nor does it induce in them any of the phenomena mentioned. In cases that react, the cornea and deeper structures of the eye are unaffected, marked protrusion of the membrana nictitans and closure of the eyelids is not observed, and photophobia is slight.

The reports on the value of this test differ considerably. Vallée asserts that if an animal is capable of reacting, it will do so positively and repeatedly to this test.

Mohler considers it unreliable. Ainsworth Wilson says: "The disadvantages of the ophthalmic test may be summed up in the one word 'unreliability.'" He points out that "it fails to agree with the subcutaneous method in 8 to 15 per cent. of cases, while post-mortem examinations have shown that it misses a certain number of tuberculous cases." He also considers that "the best results are obtained by combining the two tests, either by applying them simultaneously, or, better still, by employing the subcutaneous method seven days after the ophthalmic method. . . An indefinite (doubtful) systemic reaction, accompanied by a positive ophthalmic reaction, may safely be regarded as proof that the animal is tuberculous, and *vice versâ*."*

It has been suggested that the ophthalmic test is useful in cases where fraud is suspected, as the previous injection of tuberculin has no effect on the reaction of the former. We should prefer, however, in such instances to depend on the subcutaneous test carried out according to the method of Vallée, as it is far more reliable.

As the ophthalmic test can be employed in cases where a persistent high temperature occurs, it should prove useful in such instances.

2. THE CUTANEOUS TEST was introduced by von Pirquet in 1907. Vallée was the first observer to apply this test to cattle. It is carried out as follows: The site selected is usually the neck or withers; the skin is shaved, disinfected, and lightly scarified until blood begins to ooze therefrom. A few drops of ordinary tuberculin (old), diluted with an equal part of sterile saline solution or of boiled water, are then brushed into the part. In the case of tuberculous animals, an œdematous swelling

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* *Journal of Comparative Pathology and Therapeutics*, March, 1911.

appears in about twenty hours after the application of the test, and reaches its maximum in from twenty-five to thirty hours. This persists for four to five days; then a crop of papules appear, which last for about two days before desquamation takes place, and a serum-covered surface is left beneath. If the scarification is too deep, the part becomes very painful and indurated. No general or thermal disturbance is observed. In healthy cattle the reaction is slight and transient.

The phenomena of this test are markedly diminished by a previous subcutaneous injection of tuberculin, but the cutaneous test does not appear to have any effect on a subsequent test (*i.e.*, one carried out a few days later) by the subcutaneous method.

Trotter scarified a portion of skin about the size of a penny at the base of the skull, and applied tuberculin with a camel's-hair brush. In thirty-one animals so tested the post-mortem examinations showed that the test gave accurate results in twenty instances. Reinecke found that in twenty-five animals so tested by the cutaneous method one showed a trifling skin reaction, but no lesions were found post mortem; the remaining twenty-four showed no reaction, but eight of these were proved to be tuberculous on post-mortem examination.

The reliability of the cutaneous test does not compare favourably with that of the subcutaneous method.

3. THE OPHTHALMO-CUTANEOUS TEST has been applied by Guèrin and others. It consists in first applying the ophthalmic test, and a few days later testing by means of the subcutaneous method. In tuberculous animals the conjunctival reaction is intensely revived, producing what is termed the "second-day reaction." The consensus of opinion favours the employment of this combined method, as it appears to frustrate any attempts at fraud, such as the subcutaneous injection of tuberculin by the owner prior to the official test. Litteljohn speaks highly of this test, and states that the secondary conjunctival reaction was most marked at the fifteenth hour after the employment of the subcutaneous injection of tuberculin.

4. THE EPIDERMAL TEST consists in rubbing into the skin an ointment prepared with equal parts of tuberculin (old) and lanolin, or a few drops of tuberculin alone. In tuberculous cattle, the skin treated as described shows in from twenty to twenty-four hours a red, swollen appearance, followed by a crop of vesicles. In healthy cattle no reaction occurs. Lignières and Vallée have reported results of this test, but they do not appear to be very favourably impressed by it. Vallée stated that out of eighteen cattle proved to be tuberculous by the employment of the subcutaneous method and by subsequent post-mortem examination, only

ten gave a distinct reaction to the epidermal test, and five failed to show any reaction.

5. THE INTRADERMIC TEST.—In this the tuberculin is injected into the substance of the skin, and thus differs from the cutaneous method. Moussu applied it to cattle, selecting a fold of skin at the base of the tail as the seat for injection. Two minims of a 10 per cent. tuberculin (old) were administered in this manner, and in tuberculous cattle an œdematous swelling of the size of a walnut developed, and persisted for three or four days. Similar results occurred in swine, goats, and sheep, but in swine the swelling was hæmorrhagic in character. Vallée has only tried this method on seven tuberculous cattle, and obtained over 70 per cent. of correct results. If a subcutaneous injection of tuberculin be given a day or two beforehand, the reaction to the intradermic test does not occur.

6. THE MIXED METHODS of testing may be carried out if considered advisable, particularly the ophthalmo-cutaneous test, the latter being specially indicated when the temperature of the animal is such that the subcutaneous method cannot be carried out.

A consideration of the various methods leads us to the conclusion that the most reliable is undoubtedly the subcutaneous test, the next in order being the intradermic method.

In making arrangements for testing large herds of milking cows, one fact has to be borne in mind, and that is with reference to the effect on the yield of milk. Although tuberculin does not render the milk of cows undergoing the test injurious in any way, it undoubtedly decreases the yield of milk in "reacting" cows in a material manner for the first three milkings after the application of the test. We know of instances in which the milk-supply has been decreased to as much as two-thirds the usual quantity in herds where the number of reactors have been as high as 50 to 60 per cent.

When large contracts have to be regularly supplied, this is often a serious matter, and one likely to cause some business difficulty, if not dissatisfaction with the test. In such cases, therefore, it is advisable to obviate the inconvenience by testing the herd in sections.

II. TUBERCULOSIS IN SWINE.

As already remarked, swine are, next to cattle, the commonest subjects for tuberculosis. In the German abattoirs the number of tuberculous pigs met with was from 2 to 4 per cent., while in Denmark from 10 to 14 per cent. were found affected. Young animals are chiefly attacked, the majority of cases occurring in pigs less than one year old.

With reference to the type of bacilli present, the Royal Commission on Tuberculosis found that pigs may be naturally infected with either the bovine, human, or avian types of tubercle bacilli, but apparently in the great majority of cases the bovine type is the one which is generally present. In those cases where the human and the avian type were present the disease at the time of slaughter was found to be localised.

Infection probably occurs in most instances by way of the alimentary tract, the nature of the food offering special facilities for the entrance of the bacilli by ingestion, more especially when raw offal and the by-products of dairies are supplied to the animals. Infection may also occur from the milk of tuberculous sows, and instances are known where all the members of a litter have become affected from this source.

Cases are recorded of infection occurring after castration, the mode of entrance being the operation wounds.

McFadyean records an outbreak of tuberculosis in a litter of young pigs, seven weeks old, in which the development of the disease was very rapid ; and although the character of the lesions pointed to infection by ingestion, the exact source of the infection was not determined, as the sow appeared to be healthy. The lesions were both advanced and generalised, consisting of tuberculous nodules in the spleen and miliary tubercles in the lungs, some undergoing central necrosis ; the bronchial glands were enlarged, caseous, and slightly gritty from calcification. At the autopsy of another pig from the same litter (twelve weeks old), the glands of the throat and neck were found much enlarged, and partly caseous on section ; the inguinal, precrural, and popliteal glands were also enlarged and slightly caseous. The spleen contained four tuberculous nodules about the size of peas, and all caseous at their centres. The lungs showed a dense miliary tuberculosis, and were almost solid throughout, but not visibly caseous. The bronchial glands were enlarged and slightly caseous. In the last three feet of the small intestine about a dozen minute crater-shaped ulcers were found. The stomach, large intestine, liver, and kidneys, appeared normal. Microscopic examination of one of the cervical and one of the mesenteric glands showed calcification at the centre of some of the caseous parts.

McFadyean is of opinion that "at least in the case of pigs, and not improbably in cattle also, a most extensive development of tuberculous disease make take place well within two months, and that within the same period tuberculous lesions may undergo extensive caseous degeneration, and even become partially calcified."* This is a very important point from a medico-legal aspect.

Tuberculosis in swine is characterised by the presence of primary lesions in the mesenteric glands, the spleen, liver, and intestine. There is a marked tendency to early generalisation, and to caseation and calcification. The submaxillary lymphatic glands are frequently involved, also the retropharyngeal and the superior cervical lymphatic glands; the tonsils and pharynx may also show tuberculous lesions, and occasionally ulceration of the tongue may be met with.

The spleen may show tumour-like tuberculous growths, and the liver may present discrete lesions, or may be affected by miliary tuberculosis.

In the intestines the lesions are chiefly met with in the jejunum and sometimes in the cæcum, and consist of crater-like ulcers in the mucosa. Tubercles may occur in the muscular and subserous coats of the intestine, and the mesenteric glands are generally involved.

In the lungs the lesions may be those of a miliary tuberculosis or of a chronic caseous broncho-pneumonia. In some cases vomicæ and abscess formation may be observed. The bronchial glands may be enlarged, caseous, and show calcification. The serous membranes are not so commonly affected as in cattle. The inguinal, precrural, and popliteal lymphatic glands, may be enlarged and show caseation. Tubercular lesions may be found in the voluntary muscles, especially in the vicinity of affected joints. Skin lesions have been observed by some authorities; an erythematous eruption and the formation of hard nodules under the red spots (Williams); dark grey crusts covering the skin (Friedberger and Fröhner). Tuberculosis of the middle and internal ear is described by Schütz and by Friedberger and Fröhner. According to the former, the infection extends from the pharynx by way of the Eustachian tube to the tympanic cavity, the resulting lesions being periostitis and necrosis of the petrous temporal bone, and extension of the disease to the cerebral meninges. Occasionally the external auditory meatus may be involved, and a tubercular tumour may be formed therein.

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* *Journal of Comparative Pathology and Therapeutics*, June, 1902.

Tuberculosis of the bones, joints, and vertebral column, has also been observed in swine.

According to Ostertag, the distribution of the lesions in generalised tuberculosis of pigs is as follows : Lungs, 100 per cent. ; liver, 90 per cent. ; spleen, 80 to 85 per cent. ; mouth, larynx, and intestines, 80 per cent. ; kidneys and serous membranes, 30 per cent. ; bones, 15 to 20 per cent. ; udder, 5 per cent. ; testicles, uterus, and articulations, 1 per cent.

In the fifty-nine cases of tuberculosis in swine examined by the Royal Commission on Tuberculosis, the disease was stated to be localised in twenty-six cases, and the lesions examined were in the submaxillary glands, and in the remaining cases in the mesenteric glands. In a certain number of cases the disease was said to be localised in the glands of the neck, and in others to be generalised.

Symptoms.—In young pigs suffering from the intestinal form of the disease, gradual emaciation and digestive disturbances, such as diarrhœa, colicky pains, tympanites, and vomiting, may be observed; also a capricious appetite, pallor of the visible mucous membranes and of the nose, an unhealthy appearance of the skin, with occasionally an eruption, as already mentioned. The internal temperature varies, and is subject to fluctuations ; the eyes are sunken, and the abdomen presents a pendulous appearance. The emaciation becomes extreme, and death occurs from exhaustion.

When tubercular lesions of the brain are present, various nervous phenomena may be observed, such as walking in a circle, convulsive movements of the limbs, nystagmus, etc. The head may be carried to one side or in various positions, the tongue may show unilateral paralysis, and ptosis may be present. The case terminates in general paralysis and coma. When the lungs are involved, accelerated respirations and a dry paroxysmal cough are observed, also rapid emaciation.

Course.—In young pigs the course is rapid, but in older animals it varies from some weeks up to a few months. It is more rapid when lesions occur in the lungs and abdominal organs simultaneously.

Differential Diagnosis.—In tuberculosis affecting young pigs in a litter the diagnosis is generally based on a post-mortem examination of one of the affected animals and the discovery of tubercle bacilli in the lesions.

The chronic form of pulmonary lesions in swine fever might be mistaken for tuberculosis ; here, again, a bacteriological examination is necessary, or the presence of the latter disease may be proved by experimental inoculation of guinea-pigs.

The tuberculin test may be applied, provided the temperature is not raised above normal. According to Thiro, quoted by Friedberger and

Fröhner, the normal temperature of young pigs averages 104° F., and in adults 102° F.; and the dose of tuberculin is from 1½ to 5 minims.

According to Williams, a characteristic symptom of the disease in pigs is the presence of an enlarged submaxillary gland.

Lymphadenoma may be met with in the pig, and is to be differentiated from tuberculosis by a microscopical or by a bacteriological examination of the lesions.

Prophylaxis.—Affected animals should be destroyed, and all litter, etc., burnt; the sties should be thoroughly disinfected.

Breeding sows showing any suspicious symptoms should be tested with tuberculin.

The food should receive attention, and if there is any reason to suspect infection from the skimmed milk, etc., obtained from dairies, it should be sterilised by heating to 185° F.

III. TUBERCULOSIS IN THE SHEEP.

As already remarked, the disease is very rarely met with in this animal. According to McFadyean, the explanation of this fact is that sheep lead an outdoor life, and the risks of infection are thus much lessened. Formerly the rarity of the disease in sheep was ascribed to a natural immunity or insusceptibility to infection, but this view is not in accordance with facts, and the explanation given above is probably the correct one. The disease has been conveyed by means of experimental inoculation, and also by repeated administration *per os* of material rich in tubercle bacilli. Moussu records a case of the disease in a sheep that had been kept for two years in contact with some tuberculous cows.

The first undoubted case of tuberculosis in the sheep in Great Britain was recorded by McFadyean in the *Journal of Comparative Pathology and Therapeutics*, vol. xiii., 1900; but the occasional occurrence of the disease was noted in Continental abattoirs prior to this. From time to time the affection was said to have been met with, but its presence was not demonstrated by bacteriological methods, and the similarity of the macroscopic lesions arising from parasites in the lungs, such as the *Strongylus rufescens*, to those of tuberculosis must be taken into consideration, also those occurring in the disease termed "caseous lymphadenitis" or "pseudo-tuberculosis."

In the case referred to the lesions were situated on the inner aspect of the thoracic walls and on the upper surface of the breast-bone, and were similar to those of "grape" disease in cattle. They showed on section a fibrous capsule enclosing caseous contents which were partially calcified. At the posterior region of the sternum one growth was found which contained caseo-purulent material. Tubercle bacilli were not discovered in the lesions, but the diagnosis was confirmed by experimental inoculation of rabbits.

Another case is recorded by McFadyean in 1902, in which the lung was "to a large extent destroyed, and replaced by completely caseous and partially calcified material, the diseased areas varying from the size of a pea to that of a pigeon's egg." The bronchial glands were enlarged, caseous, and calcified; also a mediastinal gland was involved; the spleen contained numerous caseous tumours; the liver presented similar lesions; enlarged lymphatic glands were found in the gastro-hepatic omentum, and in one kidney a yellowish-white tubercle about the size of a barley grain was present. The nature of the disease was determined by experimental inoculation. In the same journal another case of ovine tuberculosis is recorded by Dr. Foulerton, in which both lungs were extensively consolidated, and contained numerous tubercles. The pleuræ were also involved, the tubercles varying in size from that of a pin's head to that of a pea. Tubercles were also present in the liver, spleen, and both kidneys. A tuberculous mass containing caseous areas was found in the inguinal glands of the right side, and the anterior mediastinal, sternal, cervical, mesenteric, lumbar, and dorsal glands, were enlarged, and contained caseating patches. The tubercles in various places showed either a caseous or a calcareous degeneration, the latter being especially well marked in the cervical glands and in the liver. The diagnosis was confirmed by experimental inoculation.

Law found that a common seat of tuberculosis in sheep was in the region of the throat, or on the side of the cranium and face.

Ramussen records a case in which the first lumbar vertebra and the last two dorsal vertebræ showed tuberculous lesions, in addition to those occurring in other organs.

Symptoms.—These are by no means diagnostic, as they are common to other affections. Marked emaciation, anæmia, debility, cough, and digestive disturbance, have been observed.

Differential Diagnosis.—It is seldom that the necessity will arise for a differential diagnosis in the living animal, as the disease is so rarely met with. Should, however, the affection be suspected, tuberculin may be employed, as the symptoms are obscure; the dose would probably be from $\frac{1}{3}$ to $\frac{3}{4}$ minim. A differential diagnosis is more likely to be required in the case of the dead animal in connection with the procedure of meat inspection.

As already mentioned, the two conditions which are most likely to be confounded with tuberculosis in the sheep are—(1) The presence of certain parasites in the lung, around which miliary nodules may form, and can easily be mistaken for true tubercles, or pulmonary lesions may be induced which simulate tuberculosis of the lung; (2) pseudo-tuberculosis, also known as "caseous lymphadenitis" or "caseous adenitis." In pseudo-tuberculosis the lesions bear a close similarity, both macroscopically and microscopically to tuberculosis, and also occur in the usual locations of the latter disease (see p. 208).

In one instance where a butcher in Ireland was prosecuted for supplying the carcass of a sheep alleged to be tuberculous to a workhouse, both the qualified meat inspector and the expert (a professor of human pathology) endeavoured to prove the existence of tuberculosis, without, however, demonstrating the presence of tubercle bacilli in the lesions, or adopting the ordinary bacteriological tests. For the defence, it was clearly proved that the lesions were due to pseudo-tuberculosis, the expert for the defendant having demonstrated the presence of the specific bacillus of this affection in the lesions; the lungs were also found to contain specimens of the *Strongylus rufescens*. The defendant was acquitted, and the District Council paid him £65, to cover expenses and costs.

IV. TUBERCULOSIS IN THE GOAT.

We have already remarked that the disease is of rare occurrence amongst goats in Great Britain, but not uncommon on the Continent. This is accounted for by the fact that these animals are not by any means numerous in Great Britain, while in Continental countries goats are extensively kept for milking purposes, and also used for food, so that the opportunities for infection are similar to those occurring in the case of cattle. The goat can with facility be infected experimentally.

According to McFadyean, probably the first authentic case of tuberculosis in the goat or in any of the smaller domestic ruminants in Great Britain was recorded by Edgar in 1892.* The animal was a she-goat, five years old; the illness occurred about eight months after giving birth to a kid, and lasted for a fortnight; the prostration was so extreme that the animal had to be destroyed. Up to the time of illness she was in fat condition, but frequently showed lameness. On examination of the lungs post mortem, they were found uniformly studded with greyish-yellow tubercles; the bronchial glands were enormously enlarged and hardened; the pleuræ were not involved. The abdominal viscera were not examined, but were described by the owner of the animal as containing white patches. The microscopical examination showed typical tubercles which were caseating, and also the presence of tubercle bacilli.

Another case is recorded by Robinson, in which a goat was companion to a tuberculous cow, and, on being tested with tuberculin, reacted. The post-mortem showed the lesions of generalised tuberculosis.

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* *Journal of Comparative Pathology and Therapeutics*, vol. v.

V. TUBERCULOSIS IN THE HORSE.

General Remarks.

Up to the year 1888 equine tuberculosis was regarded as a rare disease in Great Britain. No doubt in some of the earlier volumes of the *Veterinarian* we find cases recorded, but, as already remarked, the clinical descriptions and the lesions read more like those of glanders than of tuberculosis.

The disease was recognised on the Continent by Nocard and other observers from 1878. From 1882 to 1896 the number of published observations of genuine cases was close on one hundred. To McFadyean must be accorded the credit of drawing the attention of the profession of Great Britain to the existence of tuberculosis in the horse, and of recording the first two cases of the disease in this country subsequent to the discovery of the tubercle bacillus. These appeared in vol. i. of the *Journal of Comparative Pathology and Therapeutics*, 1888, and from that date to 1892 twenty-eight cases were recorded in that journal, in all of which the tubercle bacillus was demonstrated in the lesions, while about forty cases altogether of the disease were investigated by him during this period. Up to that time the disease was confounded with a condition termed "lymphadenoma," affecting the spleen, and the diagnosis was not determined by microscopical or bacteriological examination of the lesions. McFadyean, however, demonstrated that this so-called "lymphadenoma" was in reality a tuberculous lesion, which was present in the large majority of cases of equine tuberculosis.

In Robertson's "Equine Medicine," second edition, 1892, we find it stated "that this constitutional condition of scrofula may in our patients exhibit itself in the form of acute tuberculosis is not impossible. The probabilities, however, in adult life are largely in favour of its appearance as localised tubercle, although even in this development I am rather doubtful if I have ever encountered it."

This author further states that the situations of localised tubercle are chiefly the gland structures of the abdomen, and also that "specific arthritis" (joint-ill), in well-pronounced and malignant cases, is of a tuberculous character, while, independent of the latter, he regarded equine tuberculosis as a comparatively rare disease. In the same work "lymphadenoma of the spleen" is described, but it is admitted that in three or four cases tubercle bacilli in immense numbers were found in the growths.

Needless to remark, the above views as regards joint-ill are erroneous, but the author and reviser were approaching the real facts of the case when they described the gland structures of the abdomen as the common seat of the disease, and the presence of tubercle bacilli in the lesions of "lymphadenoma."

Although equine tuberculosis cannot be described as a common disease, it is very probable that a number of cases have been overlooked in the past, or ascribed to other affections, as the symptoms of the malady are often very obscure. The number of cases recorded cannot be taken as a correct index of the frequency of the disease, as post-mortem examinations are not always carried out to the extent that is desirable in veterinary practice. When we consider, too, that considerable doubt existed in the minds of many practitioners as to the existence of the disease in the horse, it is apparent that careful attention was not always directed to the affection either from a clinical or a pathological point of view prior to the period mentioned.

In the *Journal of Comparative Pathology and Therapeutics*, from 1888 to 1910, thirty-one cases of equine tuberculosis were recorded, and in every instance the diagnosis was confirmed by the discovery of the tubercle bacillus in the lesions. In the *Veterinary Record* from 1896 to July, 1911, sixteen cases of the disease were recorded.

An analysis of forty-three cases shows some interesting features with reference to the etiology, lesions, and symptoms of the affection.

As regards age, old horses do not appear to be specially liable. The above forty-three cases included one yearling, seven four-year-olds, four five-year-olds, nine six-year-olds, three seven-year-olds, three eight-year-olds, four ten-year-olds, two eleven-year-olds, one twelve-year-old, one thirteen-year-old, one sixteen-year-old, one eighteen-year-old, and six in which the age was not stated.

As regards breed, highly-bred horses are in the minority. Thus eighteen were cart-horses, ten harness horses, two well-bred animals, one troop-horse, one Belgian stallion, two ponies, one cob, one yearling (hackney), and seven horses in which the breed was not stated.

Some authorities, including Nocard, held that as the result of experiments both the horse and donkey resisted all attempts to inoculate them with tubercular material.

McFadyean, however, conducted experiments, and demonstrated that neither animal possessed any powers of resistance to either subcutaneous or intravenous inoculation. In one instance material from a tuberculous spleen inoculated into the cellular tissue of a horse at the base of the ear produced no alteration in the animal's appearance for the first two weeks, but on the sixteenth day the respirations were accelerated, there was an occasional cough, the temperature was irregular, and on the twentieth day dulness was marked and the respirations were hurried. On the twenty-first day the animal was unable to rise in the stall, and was destroyed. The autopsy showed no lesion at the seat of inoculation; the lungs were voluminous and firm in character, and the entire pulmonary tissue crammed with minute grey translucent tubercles; the latter showed incipient necrosis towards their centres. The spleen was normal in size and dark in colour, but showed no tubercles; the kidneys were enlarged, but presented no tuberculous lesions. The pulmonary lesions were exactly similar to those observed in ordinary cases of equine tuberculosis, which were permitted to run their course.

A peculiar feature in connection with tuberculosis in the horse as compared with the disease in cattle is that the distribution of the lesions points to the intestine as being the path by which the bacilli gain entrance to the tissues. The primary lesions generally occur in the abdominal cavity, especially in the spleen and mesenteric glands, while the pulmonary lesions are secondary.

This indicates that ingestion is the mode of infection, and leads us to inquire as to the etiology of the disease in horses. In some of the cases recorded there is clear evidence that the animals were reared on cow's milk in consequence of having lost their dams shortly after birth, and it is also well known that weakly foals or horses suffering from debilitating diseases are not unfrequently fed on cow's milk; if the latter contain tubercle bacilli the source of infection is tolerably clear.

Cohabitation with tuberculous cows is also a possible source of infection, while the ingestion of forage and litter contaminated by bovine tubercle bacilli may convey the disease to horses. Some of the cases recorded were traced to farms on which tuberculosis was prevalent amongst the cattle.

Davis* recorded four cases of equine tuberculosis which occurred on one farm—viz., a four-year-old hackney mare, a yearling filly, the daughter of said mare, a six-year-old pony, and a mare which was the grand-dam of the filly. The yearling filly was reared on cow's milk, and its constant associate was the tuberculous pony.

Nocard was of opinion that the abdominal type of equine tuberculosis was due to infection by avian tubercle bacilli, while the pulmonary type depended on infection with either bovine or human tubercle bacilli. He formed this opinion as the result of experiments which demonstrated that the guinea-pig possesses a considerable degree of resistance to inoculation with tubercular material from the abdominal form of equine tuberculosis, and also to avian tubercle bacilli, and hence concluded that infection in this type of the equine disease arose from tuberculous fowl.

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* *Veterinary Record,* vols. xvii. and xviii.

McFadyean, however, conducted experiments which showed that the guinea-pig was susceptible to inoculation with material from a case of equine abdominal tuberculosis, and that a generalised form of the disease was produced as the result, which, judging by the degree of generalisation of the lesions, was not of the avian type.*

In the five cases of equine tuberculosis investigated by the Royal Commission on Tuberculosis, the cultures obtained grew similar to those of the bovine tubercle bacillus. But the experiments carried out showed that, although the bacilli found in equine tuberculosis resembled the bovine bacilli in their cultural behaviour, the virulence of the former was not in all cases equal to that of the latter, the slight virulence for the calf being especially well marked. The conclusion was arrived at that the viruses of equine origin resembled more closely in their properties those of the bovine type than either the human or the avian type.

Morbid Anatomy.—As already remarked, the lesions are chiefly found in the abdominal cavity.

Spleen.—This organ is the most frequent seat of lesions in equine tuberculosis, and was involved in thirty-five of the forty-three cases of the disease already referred to.

The lesions consist of numerous tumour-like growths of various sizes, which may project above the surface of the organ, and a number of smaller tumours may be found on the capsule. Some may be firm in consistence, and on section may present a whitish-yellow colour, and be devoid of any distinct evidences of caseation. In other cases they are firm in texture, but on section show caseation, or they may be found to contain a thick caseo-purulent material. In some instances the spleen may be greatly enlarged, weighing from 16 to 23 pounds. The lymphatic glands along the hilus may also be enlarged.

The enlargement of the organ is not always well marked, and in some of the cases recorded the weight varied from 1 pound 14 ounces to 8 pounds.

In one case recorded by McFadyean "the spleen weighed 3¼ pounds, and its capsule presented several peculiar fibrous whitish patches and numerous *perlsucht* nodules up to the size of a split pea. The fibrous patches were not raised above the surface, and all of them had a more or less distinctly radiate shape, suggestive of old cicatrices. The largest of them was nearly as big as the palm of the hand, and on section it was seen to be composed of fibrous tissue, and to correspond with a whitish tumour-like mass in the substance of the spleen. The central part of the tumour was caseous. The other fibrous patches of the capsule overlay peculiar indurated, not sharply-defined areas of splenic substance. These had the consistence of cirrhotic liver, and a brownish colour, with greyish-white streaks. The rest of the splenic tissue was crammed with grey miliary tubercles. The splenic lymphatic glands were enlarged to the size of pigeon's eggs, but were not caseous."*

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* *Journal of Comparative Pathology and Therapeutics*, vol. ix., 1896.

Mesenteric Glands.—These were involved in thirty-seven of the forty-three cases mentioned. In some instances they were very much enlarged and showed extensive caseation, and formed a large mass in which the individual glands could not be distinguished.

In others the enlargement reached the size of a cocoa-nut, and the caseous material had undergone softening, a purulent material resulting, which was very rich in tubercle bacilli. In other cases, again, the glands are only increased to the size of a walnut or of a hen's egg.

Peritoneum.—This structure was not frequently involved. When lesions were present they were chiefly found on the peritoneal surface of the diaphragm, and consisted of fungoid or soft villous growths. In other instances the peritoneum was covered by tubercles.

Flat fleshy nodules, the largest of which were mushroom-shaped, were observed on the peritoneal surface of the diaphragm in some cases. Occasionally ascites was present.

Liver.—Tubercles were found in this organ in a small number of cases. In one instance the presence of irregular pale yellow tumours was recorded. It is interesting to note that one case of cirrhosis of the liver and two of amyloid degeneration of this organ have been met with by McFadyean in horses suffering from tuberculosis.

Kidneys.—Tubercular lesions in these organs were only met with in one case of the series mentioned. Marked enlargement of the kidneys, however, was not an uncommon condition, being probably due to chronic venous congestion.

Stomach and Intestines.—Judging by the cases recorded, tubercular lesions in these viscera must be of rare occurrence. In one instance the peritoneal surface of the stomach was covered with small tubercles. In another, a half-bred mare, four years old, in which the spleen, mesenteric glands, portal lymphatic glands, liver, and peritoneal surface of the diaphragm, were involved, one pea-shaped caseating nodule was found by McFadyean in the submucosa of the first part of the jejunum and two others in the ileum.

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* *Journal of Comparative Pathology and Therapeutics*, vol. v.

Lungs.—An analysis of the forty-three cases already mentioned shows that the lungs were involved in twenty-one instances.

The lesions varied in type. In many cases miliary tuberculosis was found; in others whitish, sarcomatous-looking nodules, about the size of a pea, were observed, which were firm in consistence and without central softening; in others, again, the lesions took the form of a caseous broncho-pneumonia, the parenchyma of the lung being studded with small abscesses enclosed by a firm capsule, or a purulent infiltration in patches was observed. Enlargement of the lungs may be present. Devine* records a case in which the lungs weighed 77 pounds; they were "of a cream colour, with red streaks at intervals through them, and on palpation felt hepatised. . . . On section the lungs appeared more or less completely filled with a cream-coloured material of a waxy consistency; in fact, there was a very small amount of real lung tissue remaining."

Pleuræ.—Lesions of the pleuræ were not commonly met with; in the series of cases mentioned this structure was involved in only seven of the forty-three instances. In some cases the disease was confined to the surface of the diaphragm. The lesions resembled those found in cattle; in one instance small rounded fleshy growths were observed.

Bronchial Glands.—These were found enlarged and caseating in some of the cases.

Mediastinal Glands.—Enlargement and caseation of these glands has been observed; in one instance a large tuberculous tumour was found in the anterior mediastinum, and on section it was caseous and partly calcified.

The prepectoral, prescapular, inguinal, and other superficial lymphatic glands, may show lesions. The submaxillary gland was involved in three of the series mentioned; it showed enlargement and induration in ten instances, and a deep-seated abscess in one case.

The pharyngeal lymphatic glands were sometimes involved; in one case recorded these glands were much enlarged and indurated, and on section were found beset with caseating centres.

Lesions of the Vertebræ.—McFadyean pointed out the occurrence of lesions of the cervical vertebræ in equine tuberculosis, and recorded the first case in the *Journal of Comparative Pathology and Therapeutics*, September, 1892. He regarded the lesions as an ostitis and periostitis, most probably due to infection by way of the lymphatic vessels of the neck, either from the throat or from the thoracic cavity. In forty-three cases of equine tuberculosis, five instances of lesions of the vertebræ were observed, but one more may reasonably be added to the latter, as all the symptoms of this condition were present, although the vertebræ were not examined post mortem.

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* *Veterinary Record*, July 1, 1911.

In the first case the lesions of the vertebræ recorded by McFadyean were as follows: The whole seven cervical vertebræ were diseased; the lesion took the form of an irregular scaly and warty growth of new bone, and was confined to the exterior of each vertebra, the spinal ring and articular surfaces being normal. The new bone had a light and porous appearance, and, while for the most part it formed only a thin layer, at some points it had a thickness of ½ inch.

No. 2 showed the lower cervical vertebræ extensively diseased; the texture of the bone was softened. At other places the outer surface of the bones was studded with small spiny projections of new bone.

No. 3: All the cervical vertebræ were more or less diseased, with the exception of the atlas. The second and third were most involved, the lesions consisting of considerable thickening due to new, rough, spongy bone formation. Spiny points of new bone were developed around some of the articular surfaces, and in one vertebra the articular head was involved by the disease.

No. 4: In one or two of the cervical vertebræ the spinal canal was involved.*

No. 5: In this case the dorsal vertebræ were involved; several of the vertebræ were firmly attached, and showed nodular growths on their surfaces.†

Occasional Lesions.—In a case recorded by Spreull, the tubercular lesions consisted of a number of pedunculated tumours occurring in a cavity situated in the region of the anterior spinous process of the ilium, with extensive suppuration. The autopsy showed the presence of a mass of tumours, which occupied the right and left iliac as well as the right and left lumbar regions, extending over and around the kidneys, and continuous through the walls of the abdomen with the tumours mentioned above. The spleen was also involved.

In another case one ovary was found to contain an abscess, while the spleen, liver, mesenteric glands, and mediastinal glands, showed the usual tuberculous lesions, and a large tubercular mass was present at the entrance to the thoracic cavity. In one instance tubercular lesions of the brain were discovered in addition to evidences of generalisation of the disease. At the base of the membranes a thickened infiltration was present, which on microscopic examination proved to be tubercular granulation tissue. Giant cells, small-celled infiltration, and connective-tissue proliferation, were present.*

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* Anderson's case, *Veterinary Record*, vol. xi.

† Brown's case, *Veterinary Record*, vol, xi.

One case is recorded in which the primary lesions occurred in the pharyngeal lymphatic glands, and the affection spread down the neck and into the thorax by way of the lymphatic vessels. These glands on both sides were much enlarged and indurated, and on section were found beset with caseating centres. The anterior mediastinum contained a large tuberculous tumour, which was found on section to be caseous and partly calcified. The prescapular lymphatic glands were enlarged and tuberculous ; a large tumour was found underneath the trachea at the entrance to the chest. This tumour was mainly fibrous in structure, but contained some tuberculous and caseating centres ; it was connected posteriorly with the prepectoral lymphatic glands, and in the anterior direction it pressed upon the œsophagus. The disease extended from the prepectoral glands to the lower end of the first rib on the left side, and to the anterior part of the sternum. The bronchial glands were enlarged and caseating, but the lungs were free from tuberculous lesions. The lesions in the abdominal cavity were of a more recent date than those mentioned above, and were probably due to a later infection from the ingestion of tubercle bacilli.†

Symptoms.—One of the commonest features in equine tuberculosis is a history of gradual emaciation. In many of the recorded cases the loss of condition was observed to follow an attack of influenza. The period of time during which this symptom was present varied from two to eighteen months ; in some instances it may occur more rapidly. The temperature is generally raised to 101° or 102° F., and in some instances to 103° F., but is subject to fluctuations. The appetite is often capricious. In some cases the animal feeds well until the disease is advanced ; in others there is a history of loss of appetite. The visible mucous membranes are pallid, and swelling of the limbs is not an uncommon symptom.

Polyuria occurs in some cases ; it may be temporary, or may persist for several weeks when the disease becomes advanced.

A scurfy condition of the skin may be observed, and in some instances a generalised eczematous eruption may be present.

When the lungs are involved, the respirations become disturbed, cough may or may not be present, and the physical signs will depend on the extent of the pulmonary lesions. Evidences of bronchitis may be observed, also a muco-purulent nasal discharge. In some cases the type of respiration present is that seen in asthma or " broken wind."

Dysphagia occurs when tuberculous lesions cause pressure on the œsophagus; colicky pains after feeding have been observed in some cases of the abdominal type of the disease.

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* Braid's case, *Veterinary Record*, May, 1903.

† McFadyean, *Journal of Comparative Pathology and Therapeutics*, vol. v.

Occasionally an examination *per rectum* may reveal the presence of enlarged lymphatic glands. Enlargement of the superficial lymphatic glands may be observed in some cases.

In one instance a tumour could be felt in the region of the left flank; in another case a hard swelling was present in front of the sternum, and a similar one appeared higher up in the neck. These interfered with the movements of the neck, and also with deglutition; the animal grunted when moved or excited in the slightest degree, and constantly stood with his nose poked out, the attitude being similar to that occurring in acute laryngitis.

When the vertebræ are involved, the symptoms presented are very characteristic. In one case the head was carried so that the poll was horizontal with the withers; the animal was able to lower the head, but not to raise it. He was disinclined for movement, walked in a "spread-out" style, and turned in a very wide manner on the near side; over the region of the lower cervical vertebræ a small, hard, painful swelling was found. In this case the lower cervical vertebræ were chiefly affected. In another instance in which all the cervical vertebræ were more or less diseased, with the exception of the atlas, the animal was unable to bend his neck; over several of the vertebræ the tissues presented a hard lumpy appearance, and marked tenderness was evinced on pressure of this region.

Amongst other symptoms observed in the cases recorded were inability to feed off the ground, the neck appeared as if fixed, and if it was forcibly bent to either side pain was evinced; the nose was "poked out" and turned slightly upwards and to the right side; also extreme atrophy of the muscles covering the cervical vertebræ was present, so that the prominences of the latter were readily seen and felt. In one instance where the dorsal vertebræ were involved marked stiffness in progression was observed, both in the fore- and hind-limbs, also immobility of the neck; the near fore-leg was kept in advance of the off fore, and the animal was lame in the former. Later on paralysis of the hind-quarters occurred. In other cases the horse showed marked difficulty in turning or in backing, and if suddenly forced to carry out these movements he would lose his balance and fall down.

Course.—In the cases recorded the animals became so debilitated and emaciated that either death ensued from exhaustion or they had to be destroyed. In one instance, however, emaciation was not so marked as usual.

Differential Diagnosis.

Although the presence of tuberculosis may be suspected when well-marked emaciation occurs in the absence of any apparent cause, or in association with the other symptoms mentioned, the only reliable diagnostic measure is the employment of the tuberculin test.

This is carried out in the same manner and with similar precautions as the procedure in the case of cattle. Care should be taken that the temperature at the time of injection does not exceed 101·5° F. The dose of tuberculin is similar to that employed for cattle—viz., 3 c.c. or 50 minims for a full-sized horse, while for a cob 45 minims, and for a pony 40 minims will be sufficient.

In the tuberculous horse a firm swelling practically always appears at the seat of injection; this may attain to the size of a walnut, or may be of considerable dimensions, and disappears in from eighteen to twenty-four hours.

When the submaxillary gland is enlarged and indurated, and emaciation is present, if the tuberculin test gives negative results, then the mallein test may be employed in order to decide the presence or absence of glanders.

At the autopsy a differential diagnosis between pulmonary tuberculosis and pulmonary glanders may be necessary. Regarding the macroscopical diagnosis, Hunting points out that the nodules of pulmonary glanders are seldom if ever found in such numbers and so evenly distributed throughout the lungs as to be confounded with miliary tuberculosis. Nor can they be mistaken for the other lesions found in pulmonary tuberculosis in the horse. In tuberculosis the bronchial glands are always involved when the lungs are the seat of marked lesions; in glanders this is not so.* The differential diagnosis can also be carried out by microscopical examination of the lesions and by bacteriological methods (see Glanders, p. 89).

With reference to the differential diagnosis of affections of the spleen and tuberculosis of this organ, a microscopical and bacteriological examination of the lesions will be necessary.

TUBERCULOSIS IN THE DONKEY AND MULE.

In this animal the disease is very rarely met with. Cases have been observed by Nocard, Blanc, and others, and the disease has also been transmitted by experimental inoculation by Nocard and Stockman. Nocard, by keeping two donkeys for twenty-eight months in the same building with tuberculous cattle, found that one of them became infected.

A case of abdominal tuberculosis in a mule has been recorded by Pleindoux.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xv., p. 230.

VI. TUBERCULOSIS IN THE DOG.

By HENRY GRAY, M.R.C.V.S.,
Kensington, W.

Until the last twenty-five years tuberculosis was looked upon as a rare, if not an unknown, disease of the dog. It is true that Blaine, in the first (1817) and subsequent (1845) editions of his "Outlines of Canine Pathology," and many other writers, referred to it as occurring in this species, but it was not accepted until the bacillus of Koch was demonstrated in the lesions, sputum, and pleuritic, pericarditic, and ascitic fluids, and conveyed by inoculation to other animals. Thanks to the labours of Jensen, Eber, Galli-Valerio, and more particularly to Cadiot, and in a lesser degree to others, we have now quite an amount of literature placing the subject on a firm basis. The affection also occurs in wild canine animals confined in zoological gardens.

It is not a rare disease in the dog, although it is encountered in him less frequently than in the cat. Though it may occur in young animals, it is generally seen in adult or aged ones, especially those that live in close proximity to their owners. It runs as a rule a chronic course, but occasionally it may take an acute form. Very frequently it has silently progressed to an extensive degree before the animal shows any serious manifestations of its ravages, and in many instances it is not suspected until a post-mortem examination is made. From a *public health point of view* it is important to recognise the disease early, as it may become a dangerous menace to the health of human beings in contact with such animals.

In a great number of cases the dog contracts the disease from human beings suffering from phthisis. In some cities canine tuberculosis is more prevalent than in others. In Paris, where it is more frequently encountered than in London, a large majority of dogs affected belong to restaurant-keepers. In this case, no doubt, they contract it by ingesting the contents of the spittoon containing the sputum of phthisical subjects. Licking the lips of consumptive people is also a fruitful source. I saw, in consultation, a tubercular dog belonging to a medical gentleman, who was, as well as his wife, the victim of tuberculosis, and both were passionately fond of the animal. I have also encountered at the same time two tubercular dogs, whose owner, a young man, was suffering from phthisis. Similar cases to these are far from rare, and have been witnessed on various occasions by other practitioners.

Raw meat or offal, or unboiled milk from tubercular animals, are also possible sources of infection; but I believe the commonest source is that from mankind.

All dogs are, however, not equally susceptible to infection, even to the experimental method, in which large masses of tubercular material or large quantities of pure cultures have in a very great number of instances failed to induce the disease. Therefore it seems rational to conclude that a special predisposition exists in those cases that contract the infection.

Dogs are refractory to the avian bacillus, even when large quantities are injected into the veins. This type of bacillus also fails to confer immunity to the mammalian type.

I believe, in spite of the now classical experiments of Tappeiner, Bertheau, Weichselbaum, Frerichs, Cramer, and others, that tuberculosis in the dog is rarely, if ever, contracted by *inhalation*. In my opinion it is introduced by *ingestion*, although I willingly admit it may also occur by *inoculation*, which rarely, if ever, takes place under natural conditions.

Lesions.—The disease affects principally the lungs and liver, but no organ is exempt, and very often it is encountered in a generalised form. Occasionally it may confine itself to one part, to be ultimately extended to the various visceral organs.

When the *lungs* are affected, the tubercles are of a greyish colour, and generally of a fibrous consistence. Although caseation is not a common characteristic of canine tuberculosis, older tubercles may undergo softening in their centres and contain a little greenish-grey pus. When very extensive they may coalesce, forming large lobulated masses, which are usually raised to a marked extent above the surface of the lung. Occasionally such masses undergo softening, and form large cavities by the softened centres coalescing. In a few cases both lobes of the lung have numerous small tubercles scattered all through their substance.

Secondary changes, such as acute or chronic pneumonia, sclerosis, congestion, emphysema, bronchial dilatation, chronic bronchitis, etc., extending to the greater portion of one or several lobes, and in particular to the posterior ones, may be found associated with pulmonary tuberculosis.

The lung may exceptionally be affected with *caseous pneumonia*, in which the disease extends to a large portion of one or both of these organs. The diseased lung during the early stage presents, when cut into, a firm, dry, and slightly granular appearance. Around the caseous portion of lung a zone of hepatised tissue is noticed. When the disease is allowed to run its course, the caseous material undergoes softening, and ultimately leaves large cavities. This is also witnessed in the cat.

The *bronchial tubes* do not very often show well-marked lesions. The bronchioles in the vicinity of a tubercle often become dilated, and their mucous membrane is congested or inflamed.

The *larynx* and *trachea* are occasionally the seat of tubercular ulceration, which usually granulates and gives rise to extensive vegetations.

The *bronchial and also the mediastinal glands*, especially the former, are commonly attacked at the same time as the lung, and perhaps the pleura. They may, however, be the chief or only seat of the disease. They are generally enlarged to an enormous extent, giving rise to the idea that they are new growths, such as cancer or sarcoma, for which they were formerly mistaken. The lesions may affect the glands of one side only, but usually they extend to those of both sides. Although they are occasionally firm or fibrous in consistence, they frequently undergo softening in the centre, giving rise to large cavities, which usually contain a clear, amber-tinted, sticky fluid. They may, however, contain a milky, cheesy, or pultaceous material. These cavities are often intersected by fibrous bands, which give the interior a honeycombed or reticulated appearance.

The *pleura* is commonly the seat of the morbid process, and is generally associated with lesions of the lung or peritoneum. Occasionally it is confined solely to this membrane. When the disease is acute, the pleural surface is covered with false membranes, composed of fibrin, leucocytes, red blood-corpuscles, and desquamated epithelial cells. These false membranes are usually encountered only when the tubercles are situated immediately beneath the pleural membrane. As a rule, however, pleural lesions are better marked on the mediastinal and parietal than on the visceral pleura. The mediastinal pleura is often very much thickened, discoloured, and covered with large irregular masses, having on their surface fine granulations. These granulations are generally fibrous and dotted over with fine tubercles, which may, here and there, be found in small patches, undergoing softening, caseation, or cretification. The *pleural sacs* are often found to contain a large amount of a clear, amber-tinted, serous fluid; or the effusion may be turbid and contain flakes of fibrin; or it may be greenish-grey, bloody, or even purulent in appearance. Tubercular pleurisy is not always accompanied by effusion; it is occasionally dry in character, and in such instances the slight fibrinous exudate often brings about adhesion of the visceral to the parietal layer of the membrane, forming *pleural symphysis*, which is not a rare condition in the dog. Occasionally the pleural membranes may be adherent at two points, leaving a clear interspace, which contains a variable amount of fluid (loculated effusion).

Tuberculosis of the *pericardium* is often met with even when the lung and pleural membrane are not affected. It is also encountered when the bronchial and mediastinal glands are the only other parts attacked. The visceral membrane may alone be the seat of tubercles, but as a rule both layers are included at the same time in the morbid process. The *pericardial sac* may contain fluid, which may be either clear, amber-tinted, or bloody in appearance. In some instances the amount of fluid may be very extensive, and cause marked distension of the structure. The pericardial membrane may, like the pleural membrane, be the seat of well-marked changes, such as thickening, vegetations, etc. It may also become adherent to the cardiac wall, to the costal pleura, or to the diaphragm.

The *heart* itself may be the seat of tubercles. These are whitish in colour, and vary from the size of a millet-seed to that of a bean. They may run into one another, forming large masses of an irregular outline and fibrous consistence. They may infiltrate the walls of the ventricle or auricle, interventricular wall, and the valves. Eber found the arch of the *aorta* thickened, and the middle and adventitious tunics contained lardaceous centres.

The *intestines* are rarely the seat of tubercles. They may, however, be the seat of ulceration, especially the ileum and cæcum, and sometimes the rectum. The ulcers may be several in number or only solitary; they vary in extent and depth, and may even cause perforation and peritonitis; or, if they are deep, but do not penetrate the peritoneal layer of the intestine, they set up a localised peritonitis, to which often a portion of the adjoining viscera, notably the omentum, becomes attached. The ulceration may become extensive. Very often a chronic catarrhal intestinal condition is encountered. When there is intestinal ulceration, the lacteals and corresponding mesenteric glands become involved in the morbid process. These glands may often be found independently affected; they may be enlarged to an enormous extent, and show a round or irregular outline, and often in their centre they are found to be undergoing softening or cystic degeneration, or to contain a purulent material.

The *peritoneum* is not rarely affected. The parietal layer may remain normal, although the visceral layer, especially that of the mesentery and omentum, may show numerous isolated tubercles, usually of small dimensions, scattered all over its surface. At times the parietal layer is the seat of a generalised miliary tuberculosis. When the mesenteric glands are attacked, we may find the mesentery and even the omentum considerably thickened and tough, and often of the consistence of fibrous tissue. They are frequently so crowded with small tubercles that the latter are almost in juxtaposition with one another. Occasionally the mesentery and omentum are covered here and there with vegetations. The peritoneal cavity may contain a variable amount of fluid; often it is present in enormous quantity. It may be of a clear citron colour, or greenish-grey, or bloody in appearance.

The *liver*, next to the lung, is more often the seat of tubercle than any other organ. It may be attacked when the bronchial or mediastinal glands are the only other organs affected. Usually, however, it accompanies tuberculosis of the lung. The tubercles vary in size and appearance. Sometimes they are greyish or whitish in colour, and fibrous and hard in consistence, and rarely exceed the size of a pea. Those occurring on Glisson's capsule are generally conical or hemispherical in shape, but those extending into the substance of the liver are circumscribed, and have very often a crenated outline.

The *kidneys* are often the seat of disseminated tuberculosis. Either one or both glands may be affected. The tubercles are chiefly scattered in the substance of the cortical layer, from which some may extend towards the capsule, which they often raise up and give rise to a large tubercular eminence on the surface of this organ. They may also be encountered in the medullary layer, from which they exceptionally extend to the pelvis of the kidney, and cause ulceration of its mucous membrane. The tubercles may be the seat of caseous or lardaceous degeneration. Very often interstitial nephritis is associated with these changes, but as this chronic renal condition is so commonly met with in aged non-tubercular dogs, considerable care should be exercised in ascribing it to a tubercular origin. As in the cat, the lesions of the disease may be confined to the kidney only.

The *bladder*, *prostate*, *urethra*, *testicle*, *tunica vaginalis*, *uterus*, and *ovary*, are often affected with isolated or confluent tubercles, and the uterus may contain a quantity of purulent or cheesy material, and its walls are often thickened in patches, which may be hard or soft in consistence. Similar conditions to these are also occasionally found in the cat.

The *spleen* and *pancreas* are usually exempt from tubercle. They may, however, contain a few small tubercles, which in the spleen stand out prominently against its dark red ground; but in the pancreas, in consequence of their similarity in appearance to this organ, they may be easily overlooked.

The *nervous centres* are occasionally attacked with tubercle. When lesions are present they are usually found scattered over the surface of the brain, but they may also extend into its substance. *Ocular lesions* may be present, such as iritis, choroiditis, hydrophthalmos, etc.

The *submaxillary, retropharyngeal, prescapular, and inguinal glands*, are exceptionally found enlarged; when involved, their size may be increased to an enormous extent. Their centres have generally undergone softening, and occasionally they may open externally by means of a fistula, which gives exit to a clear or dirty coloured, sticky fluid. They may also be the seat of extensive ulceration.

The *skin*, especially on the under surface of the cervical region, thorax, and outer surface of the parotid region, may occasionally undergo extensive ulceration, the base of which is granular or covered with vegetations, and from which a sticky or oily fluid exudes.

In *acute miliary tuberculosis* the lungs and pleuræ, as well as the other viscera and serous membranes, are covered with minute translucent greyish tubercles of a fibrous consistence. The *lungs* are uniformly swollen, congested, emphysematous, and friable. The *pleural* membranes are sometimes covered with false exudate, and in this case the pleural sacs contain a serous or bloody effusion. The spleen is swollen and congested. Cadiot describes a tubercular septicæmia in which there are no appreciable tubercles.

In this acute condition one generally finds some old or pre-existing lesion either in the lungs, mesenteric, mediastinal, or bronchial lymphatic glands.

Symptoms.—The disease very often runs an insidious or silent course, until almost suddenly the animal manifests signs of effusion into the thoracic, pericardial, or peritoneal cavities. These may in some cases be simultaneously affected, when the respiration becomes disordered, and is often followed by gradual emaciation and weakness. At other times these effusions are especially noticeable, following a chronic cough, obstinate ulceration of the skin, or swollen lymphatic glands in the region of the throat, neck, or shoulder.

In many instances there is an occasional short cough, which terminates in the animal expectorating sputum into its mouth and then swallowing it. This goes on for weeks, perhaps months, and occasionally the cough disappears, and is followed by evidences of an effusion into the pleural, pericardial, or peritoneal cavity, or by some cerebral or spinal disturbance. The soft tissues of the temporo-maxillary and dorsal regions become attenuated, allowing the bony parts to stand out prominently, and thus giving the attenuated parts a sunken appearance. The flanks seem to be slightly fallen in, and the abdomen appears pendulous. The mucous membranes are pallid, and the facial expression is not bright. The coat is dull and open. The appetite is capricious, and the animal tires easily. A catarrhal discharge of a mucous or fatty-looking material may occasionally be found issuing from the eyes or nostrils. It is not profuse, and rarely arises from an injected mucous membrane.

If the larynx, trachea, or the larger bronchial tubes, become the seat of ulcerations, which, if the animal lives long enough, ultimately give rise to profuse granulations, so as to resemble papillary growths, the cough is frequent, the breathing is noisy and difficult. On inspection of the larynx, these ulcerations, when situated on this organ, can easily be seen.

When the lesions are confined principally to the bronchial and mediastinal glands, there may be an occasional muffled cough, which is sometimes followed by expectoration; the inferior third of the thoracic walls, corresponding to the sterno-costal cartilages, is contracted, almost vertical, or perhaps even slightly concave externally, and on percussion and auscultation this region is found dull; but the regions above it have their normal sounds very highly intensified, those of the heart being plainly heard all over the chest.

When the lesions in the lung commence to undergo retrogressive changes, there may be present a dry, hoarse cough, which may be induced by making the animal move. After a time this cough may become louder, more frequent, and looser, and during the effort a mucous or muco-purulent discharge may be ejected from the mouth, or even vomition may take place.

When the pleural membrane is affected, the respiration becomes difficult, short, and sharp, and very often accompanied by moaning or groaning at each inspiratory effort; but this is sometimes noticed when the pleura remains indemnified, while the bronchial and mediastinal glands are extensively implicated. If pleural effusion be present, the flanks heave or have a pumping-like action. Very often pleurisy is preceded by physical signs indicating the presence of cavities within the lung substance. Pericardial effusion will be evidenced by the usual symptoms of that condition.

When the liver is involved, we may occasionally observe a slight icteric condition of the mucous membranes, evidences of enlargement of the organ, and perhaps abdominal effusion.

Occasionally, when the disease attacks the kidney, we may find polyuria and great thirst, and an examination of the urine may reveal the presence of albumin, casts, and perhaps blood and tubercle bacilli.

When the intestines and mesenteric glands are affected, diarrhœa, dysentery, vomiting, constipation, colic, or tenesmus, may be manifested. Very often the diarrhœa is followed by constipation, and the fæces may contain blood. On manipulation of the abdomen, the mesenteric glands, if enlarged, can easily be detected. When they are voluminous, they may give rise to vomiting.

When the pancreas is the seat of lesions, we may notice polyuria, great thirst, emaciation, and icteric symptoms, and perhaps the presence of sugar in the urine.

In those occasional cases in which the nervous system is involved, we may observe great restlessness, howling, convulsions, and delirium; in other instances, vertigo, paralysis, or coma, may ensue. Some animals are dull, others irritable and even vicious on the slightest provocation, and still others docile and bright. At times there may be shivering, the skin is hot, and the nose dry.

The internal temperature, when abnormal, is usually an oscillating one, the evening reading being more marked than that of the morning. It may vary from 102·8° to 105° F. in the twenty-four hours, or it may even descend below the normal average (101·5° F.). It is, however, most irregular, but may remain quite normal.

When the eyeball becomes affected, iritis, choroiditis, partial or total loss of vision, or an amaurotic state of the pupil of one, but usually of both, eyes may be observed.

The *external localisations of tuberculosis* are far from rare, but generally overlooked as such, and attributed to other causes. They consist of enlargement of the submaxillary, retropharyngeal, cervical, prescapular, inguinal, etc., glands. These very often undergo softening in the centre, rupture, and give rise to a large granular wound, which is very obstinate to treatment. When this contracts, if not treated, it leaves a fistulous opening, from which issues an oily or syrupy material, matting together the surrounding hairs as if liquid gum had been applied thereto. When these glandular enlargements undergo resolution, they are very often followed by acute miliary tuberculosis, pleural or pericardial effusion, etc.

In other instances the skin alone undergoes the morbid process; it becomes necrotic, sloughs off in a patch or patches, leaving a latticed or reticulated granular base, which heals with very great difficulty. The surrounding skin becomes indurated, the edges are not inclined to approximate, and very often a fistulous gap is left. If healing takes place, the part is likely to open again and continue its morbid course. It is, like the glandular enlargements, liable to give rise to secondary disseminations.

The joints and limbs very rarely become involved in canine or in feline tuberculosis; when lesions occur in these regions, lameness is observed.

As the disease progresses, the cough and emaciation increase, the appetite becomes very capricious, and the animal is generally destroyed as incurable.

It is surprising what a great number of times a case of tubercular ascites can withstand the operation of tapping. I observed two cases on which the operation was performed over thirty times in each case respectively—on an average once a week.

Although in a large number of cases tubercular dogs may live for years without suffering much disturbance, others, if allowed to live, rarely survive a year, and in many other instances they succumb to some intercurrent affection, such as gastro-enteritis. In other cases death quickly supervenes from pneumonia, pleuritic, pericardial, or abdominal effusion, which may or may not be due to tubercular infection.

Diagnosis.—The diseases with which tuberculosis is likely to be confounded are the *chronic broncho-pneumonia of canine distemper*, *generalised carcinoma* and *sarcoma*, *lymphoma*, *lymphosarcoma*, *lymphadenoma*, *chronic bronchitis*, *asthma*, *chronic gastro-enteric catarrh*, *verminous pleurisy* or *pneumonia*, *coccidiosis*, and *pseudo-tuberculosis*.

If we take into consideration the history of the case, the *general appearance of the animal*, and the course of the disease, and submit the nasal, tracheal, or wound discharges (which are generally small in amount), or the pleuritic, pericardial, or ascitic fluid, to a bacteriological examination, and finally the animal to the various tuberculin tests, we believe the difficulty of arriving at a correct diagnosis is greatly reduced, if not absolutely removed. There are, however, many cases in which the tubercle bacillus cannot be readily demonstrated, but in this case some of the diseased material or fluid should be inoculated into the peritoneal cavity of a guinea-pig.

The *dose of tuberculin* is from 0·5 to 1 c.c., according to the size of the dog. I would, however, approximate it at $\frac{1}{6}$ to $\frac{1}{3}$ minim to the pound of body-weight. It should be injected under the loose skin inside the thigh or below the abdomen. The operation should be performed in the morning, and the temperature taken *every hour*, as very often the reaction takes place within a few hours (from two to five hours), and declines to normal within twenty-four hours.

In some cases of advanced and generalised tuberculosis tuberculin is said to be uncertain in action, and may give rise to hyperthermia and death. It has also been asserted that in some instances it has caused a reaction in dogs that are not tuberculous. I believe that close attention should be paid to those cases in which the tuberculin apparently fails to give its regular specific action, with the object of placing its employment on a better-defined basis than it is even at present.

Treatment.—Although some cases of tuberculosis might recover spontaneously or with proper treatment, I believe that as soon as a correct diagnosis is arrived at it is the bounden duty of the practitioner to advise the owner, on public health grounds, to have the animal, more especially if a pet or house dog, destroyed. I would go farther, and suggest that the disease should be compulsorily notifiable by scheduling it under the Diseases of Animals Act. If there remain any doubt about the transference of tuberculosis of the ox to man, and *vice versâ*, there should be none so far as the dog and cat are concerned.

If, however, treatment is demanded by the owner, the animal should be sent into the country, preferably to the seaside, and housed in the open, where it can not only get plenty of fresh air, but also exercise. Highly nutritious food, such as milk, raw meat, and tripe, should be given. Cod-liver oil, virol, or petroleum emulsion, might be tried. Creosote or creolin inhalations are advisable. Internally guaiacol, cacodylate of soda, atoxyl, or soamine, should be administered. The opsonic-index treatment should be undertaken, as much with the object of proving its value in the treatment of tuberculosis as in the cure of the animal.

The external ulcerations often respond to the repeated local applications of iodine or nitrate of silver.

VII. TUBERCULOSIS IN THE CAT.

By HENRY GRAY, M.R.C.V.S.

Tuberculosis is a commoner affection in the cat than in the dog, and adult or aged animals are more liable to it than kittens. These facts were observed by Blaine as far back as 1817. Although it mostly originates in these animals from drinking tuberculous milk or eating raw meat or uncooked offal of tubercular cattle, and also from infection from diseased cats, it is frequently contracted from tubercular people. In one instance I encountered two cases of tubercular pleurisy during the same week coming from the house where a phthisical young man resided.

Wild feline animals confined in zoological gardens frequently become affected with tuberculosis. The disease may become generalised, but usually in this species it confines itself to one or two localities, and the lungs often remain free from it. It is commonly found attacking the mesenteric glands alone. This latter localisation and that of the skin around the throat or breast are the two commoner seats of predilection. Miliary tuberculosis of the lung is not rare; it is generally secondary to some other lesion in a remote part.

Symptoms.—It is characterised by progressive emaciation, a persistent dry cough, sometimes retching or vomiting, and frequently a chronic diarrhœa. The appetite is capricious and irregular; the respirations are short, jerky, and after exertion become precipitous (pumping-like action of the flanks) and very difficult, so much so that the animal keeps its mouth open, gasping for breath, and in these paroxysms falls down exhausted. There may be constipation, especially in the case of an enlargement of the mesenteric glands, which often causes intestinal obstruction. The temperature may rise three or four degrees (104° to 106° F.) above the normal (101° to 102° F.). After a time the emaciation is so great that the muscles, especially those of the temporo-maxillary region and of the dorsal region, become wasted, and the eyes appear sunken, thus giving the animal the "tubercular expression." The mucous membranes become pallid and anæmic; the respirations are quickened and short, sometimes accompanied by moaning or grunting, and become difficult under the least exertion. Sometimes a discharge of yellowish-white material issues from the nostrils or mouth during a fit of coughing.

On auscultation of the chest, the pulmonary sounds may be of a bubbling, splashing, rattling, or gurgling character, and sometimes the breathing is tubular. There may also be signs of effusion into the thoracic cavity, and evidences of cavities within the substance of the lungs.

When the abdomen is the seat of the lesions, there may be, besides great emaciation and weakness, an effusion into the peritoneal cavity; and when the mesenteric glands are enlarged (and such enlargements are easily detected in this species by simple manipulation), there may be symptoms of colic or constipation. When the kidneys are extensively involved, they may sometimes be found by palpation to be enlarged, rough, uneven, or nodulated. The association of an amaurotic pupil and a degenerative or vascular choroiditis with the renal lesions mentioned is quite pathognomonic of tuberculosis. When the uterus or ovaries are affected, there may be in the pregnant cat abortion, and in the non-pregnant animal a whitish leucorrhœal discharge issuing from the vagina.

In some instances the eyeball becomes involved, and is the first condition to attract attention, in consequence of the defective sight or total blindness of the animal. Iritis may be present, but more commonly an amaurotic condition of the pupil is observed. Examination with the ophthalmoscope shows that the choroid has in parts lost its brilliant lustre, and has a dull, slaty or vinaceous appearance, while abnormal tortuous vessels are found extending all over its field. Very often the retina is found detached. A special study of this form of tuberculous localisation has been made by Hancock and Coats from cases submitted to them by A. J. Sewell.*

If the disease is located in the parotid lymphatic glands under the ear, we may find after a time a chronic ulceration and a dirty sticky discharge flowing from unhealthy-looking fistulous wounds in this region.

Occasionally tubercular enlargement of the lymphoid tissue in the region of the posterior nares is witnessed, giving rise to a polypoid obstruction to the inlet and outlet of air through the nostrils to the respiratory organs. At times the joints, especially the coxo-femoral joint, become the seat of a tubercular ulceration, giving rise to lameness. The skin of the nose, throat, and breast, and other parts of the body, undergoes a destructive ulceration, which has a tendency to spread, and has no inclination to heal even under treatment; the resulting wounds appear dirty, and give forth a thin, colourless, sticky material, which mats the surrounding fur. The anal glands, the toes, and the tissues in the axilla or groin, may also be the seat of the disease—in fact, any part of the body is liable to be attacked. Sometimes the disease takes on a rapid course and simulates various other acute affections, such as pneumonia, distemper, etc. Usually death takes place within one to six months, but occasionally the animal may live longer, and in rare instances recovery is possible.

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* *Vide Veterinary Record*, January 14, 1911, vol. xxiii.

Diagnosis.—It is likely to be mistaken for many other chronic diseases, such as the wasting form of chronic distemper, cancer, sarcoma, lymphadenoma, chronic liver disease, pleurisy, verminous pneumonia, pseudotuberculosis, coccidiosis, nephritis, chronic gastro-enteric catarrh, etc.

The use of tuberculin, or the microscopical examination of any discharge or exudate, should be adopted when necessary. The thoracic or ascitic exudate, when present, may be drawn off by a fine trocar and examined for the bacillus. The dose of tuberculin for the cat is from 1 to 5 minims, and after its injection the temperature should be taken every hour, as the maximum is reached in a few hours (about the fifth), and soon declines to reach the normal. Unless this precaution is taken, wrong conclusions are likely to be arrived at.

Treatment.—As the disease is generally incurable, and is communicable to mankind and to other animals, the affected animal should be destroyed, and its cage burnt or its house cleansed and disinfected.

VIII. AVIAN TUBERCULOSIS.

By G. L. INGRAM, M.R.C.V.S.,
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Tuberculosis is a fairly common disease amongst poultry, and may also be met with in pheasants, pigeons, turkeys, parrots, and other birds.

According to Rabinowitsch, parrots may be affected with the avian, the bovine, and the human type of organism; in the latter case they become infected from phthisical owners. In the Berlin clinic, of 700 parrots treated, 25 per cent. were found to be tuberculous.

In poultry, infection generally occurs by ingestion of food contaminated by the fæces of affected fowl. In such, intestinal lesions are common, and as a result the discharges contain large numbers of tubercle bacilli.

In parrots, according to Friedberger and Fröhner, infection usually takes place through the respiratory apparatus, and is due to inhalation of human tubercle bacilli, but infection by ingestion of food contaminated by similar bacilli may also occur.

Microscopically, avian tubercle bacilli are almost identical with the human and bovine type, but are as a rule a little longer, although in a strain recently isolated from a naturally affected hen the organism was so short as to resemble in form a small coccus. Growth of the organism takes place on the same media as used for other types, but is much more rapid than is the case with mammalian tubercle bacilli. A good growth is obtainable on Dorset egg medium in a fortnight, and the range of temperature is from 35° to 45° C. The appearance of a young culture is somewhat moist and waxy, but old cultures become dry, and then more resemble a culture of the human variety. It seems probable that the avian bacillus is a variety or strain of the human type of tubercle bacillus which has become modified by continued sojourning in birds, subject to different body fluids and to the higher body temperature.

As a rule it is difficult to experimentally infect poultry with the human type of bacillus—*e.g.*, by feeding on phthisical sputum. Guinea-pigs inoculated subcutaneously with avian bacilli show a considerable degree of resistance, and when inoculated intraperitoneally the lesions are different from those caused by the human type of bacillus.

Nocard, by placing a small quantity of a culture of human tubercle bacilli enclosed in collodion sacs in the peritoneal cavities of poultry, and leaving them *in situ* for some months, succeeded after several such transplantations in altering the bacilli so that they rapidly produced the lesions of avian tuberculosis when inoculated into healthy poultry. The results of the experimental work of the Royal Commission on Tuberculosis tend to cast some doubt on the accuracy of these experiments, as they have never been successfully repeated.

Lesions.—In poultry the lesions are chiefly confined to the digestive apparatus. The liver is probably the organ most commonly affected; it presents large numbers of yellowish-white nodules, varying in size from a pin's head to that of a pea, or they may be fewer in number and up to the size of a hazel-nut. The tissue between these nodules seems slightly softer and more friable than usual, and Nocard stated that ruptures of this organ in tuberculous birds were not rare. The spleen is often involved, also the intestines; the latter may show tuberculous ulcerations.

Invasion of the peritoneum may be observed, and may be associated with ascites; the mesenteric glands may show tuberculous lesions. The lungs are rarely involved, but miliary tuberculosis of these organs may be found in some instances. Histologically the lesions resemble those met with in bovine and human tuberculosis, but as a rule there are few, if any, giant cells present.

A tuberculous lesion of the foot may be met with in the common fowl; this is popularly known as "bumble-foot." The part becomes swollen; fissures may form thereon, and give exit to a purulent material in which the presence of large numbers of tubercle bacilli can be demonstrated.

Lesions of the bones and joints may be observed in some cases.

Some observers state that tuberculous lesions in fowl show a marked tendency to caseation, and that in the completely caseous lesions numerous bacilli can be found.

In parrots tubercular lesions of the skin are very common, especially in the region of the head, neck, and beak; these occur in the form of new growths of horny skin, and may be of considerable size; they can be removed with facility, and in the granulating bases left numerous tubercle bacilli can be demonstrated. On the legs and feet the lesions may present a raw surface. The tongue, buccal mucosa, conjunctiva, and larynx, may also be involved, the lesions appearing in the form of more or less rounded vegetations; the commissure of the beak is a favourite seat of the disease. Warty growths on the eyelids may also be present. Miliary tuberculosis of the lungs is frequently met with, but in some instances the tubercles may attain to the size of a pea or of a bean.

Lesions of bones and joints may also be observed. According to Eberlein, the tuberculosis of parrots is characterised by the presence of a very large number of bacilli, which may be demonstrated in the new growths, fæces, nasal discharge, and saliva.

Symptoms.—There are no symptoms characteristic of the disease. Marked emaciation gradually appears, with debility and loss of appetite; in some cases vomiting and diarrhœa may be observed, the combs and wattles become anæmic, flaccid, and dried up, the visible mucous membranes are pale, the eyes sunken, the wings and tail droop, and lameness develops when the joints of the limbs become affected. Later on paralysis occurs, and the birds are unable to stand. The course is generally more or less chronic.

Diagnosis is based on a microscopical examination of the localised lesions, staining being carried out by the Ziehl-Neelsen method. Nocard obtained a temperature reaction in poultry by using tuberculin prepared from avian tubercle bacilli. Avian tuberculin is now employed as a diagnostic test for Jöhne's disease in cattle (see p. 202).

Treatment is obviously not only useless, but also dangerous, as the healthy birds are liable to become infected.

Prophylaxis is of importance. All affected birds should be destroyed, and all houses and sheds which have been occupied by them should be thoroughly disinfected. The surface layers of earth should be removed from the enclosed poultry-run, and disinfected with quicklime, carbolic acid, etc., and the ground surface thoroughly disinfected.

TREATMENT OF TUBERCULOSIS.

It is obvious that in the case of all animals, once a definite diagnosis of tuberculosis is arrived at, treatment is out of the question. Not only can it prove of no avail, but there is also the risk of the disease being conveyed to other animals or to mankind.

PROPHYLAXIS.

The prevention of tuberculosis in animals is a subject of vast importance, not only with reference to the interests of the stock-owner, but also because the necessary measures adopted will assist in reducing the risks of the disease being conveyed from animals to man.

The subject has been investigated in all civilised countries, and discussed both by pathologists and practitioners at many scientific gatherings, and various suggestions have been made from time to time with a view to eradicating the disease and insuring a pure milk and meat supply. An ideal plan in theory would be the compulsory testing of all cattle with tuberculin, and the "stamping out" of all animals proved to be affected with the disease; but in consequence of the enormous cost that such a system would involve, it cannot at present be considered as practicable.

Two methods of controlling the disease have been adopted with considerable success: one by Bang in Denmark, and also by Délépine, Malcolm, and others, in Great Britain and Canada; the other by Ostertag in Germany.

Bang's Method.—This consists in testing the herds with tuberculin, and then segregating the "reactors" from the animals that have successfully passed the test. The latter are housed in tubercle-free buildings, and no cattle or other animals are permitted to come into contact with them without first being subjected to the tuberculin test. The reacting cattle are also kept in separate buildings or fields, and bred from and milked for a time as usual, gradually being fed off and slaughtered. The calves of "reactors" are separated from their dams immediately after birth, and are reared on food free from tubercle infection; they are then submitted to the tuberculin test on one or more occasions, and on being proved free from tuberculosis are drafted into the tubercle-free herd when they are about a year old.

In this manner a tubercle-free herd is gradually secured, while, on the other hand, the herd of "reactors" correspondingly diminishes, such results being obtained by the exercise of some trouble and care on the part of the owner, but without causing serious interference with the dairy business generally carried on by farmers in Denmark.

Ostertag's Method is to immediately slaughter all animals affected with "open" tuberculosis—*i.e.*, cases in which the disease occurs in those parts of the body having natural outlets, and thus being dangerously infective to other animals and to man. This method has the advantage of rapidly clearing away infective cattle; the disadvantage is the difficulty in determining when non-infective animals may become infective. There is always the liability of having an infective animal in a herd for some time before its presence is discovered, and such an animal is distributing the infection freely amongst its companions.

This method was practically the one adopted by the British Board of Agriculture when the Tuberculosis Order was issued in 1909. It has marked advantages, and should not be despised; consequently it is a matter for regret that the Board withdrew an Order which in this country would have been a first attempt on a national scale to control tuberculosis in animals.

In our opinion, what is urgently required is compulsory notification of animals suspected of being affected with "open" tuberculosis; veterinary inspection should follow, with immediate slaughter of animals found to be so affected, and compensation up to carcass value should be allowed to the owner, the funds for compensation being provided by the State.

Combined with this a serious attempt should be made to induce stock-owners to voluntarily put into force Bang's method of eradicating the disease in individual herds.

This could probably be carried out by the State supplying the owners of stock with free tuberculin-testing by qualified veterinary surgeons, and free disinfection of buildings in which the tubercle-free animals are to be housed.

A capitation grant for each animal found to pass the tuberculin test, given for a limited number of years, should be allowed to each owner for the purpose of building up a tubercle-free herd.

In carrying out the tuberculin test it is sometimes found that "reactors," if retested at an interval of three months, may go through the test successfully. Some observers are of opinion that such animals that fail to react after several testings, and show a healthy appearance, may be considered as safe to return to the herd, as they have probably recovered from the disease. This opinion, however, is not generally believed in, and many authorities hold that if an animal has once definitely reacted it should be considered as tuberculous, the subsequent non-reaction being ascribed to the disease being arrested, but not cured. Hence it is impossible to predict when the disease may again become active, and the animal may consequently prove a dangerous source of infection.

PROTECTIVE INOCULATION.—In 1902 protective inoculation was introduced by von Behring as a means of preventing tuberculosis. He found that the dried bacilli of human tuberculosis injected intravenously into healthy cattle produced an increased power of resistance to subsequent experimental infection. He formed the opinion that a single inoculation is sufficient to produce immunisation against natural infection. The inoculation is carried out with 0·06 grain of tubercle bacilli in a drachm of water, and the animals selected are calves from four to six weeks old.

These calves are specially treated after birth—*i.e.*, they receive the colostrum of the cows for the first two days, and after this are fed on sterilised milk.

The inoculation is carried out by injection of the preparation mentioned into the jugular vein. A single inoculation suffices if a febrile reaction results; otherwise a second inoculation is carried out with twenty-five times the amount of tubercle bacilli.

Further research is required before it can be asserted that the procedure is free from danger, and before its general adoption can be advised.

According to Nonak (1909), no practical value can be attributed to the use of von Behring's "bovo-vaccine," particularly as regards preventing the spread of tuberculosis among bovines; but, on the other hand, it seems to have no detrimental effect on the health of the vaccinated animal.

COMMUNICABILITY OF TUBERCULOSIS FROM ANIMALS TO MAN.—As the result of much patient investigation by eminent authorities, it is now universally recognised that bovine tuberculosis can be conveyed to man by the ingestion of the meat and of the milk of affected animals. The last Royal Commission on Tuberculosis was appointed in August, 1901, and the first Interim Report appeared in 1904, which showed that, as the result of preliminary investigations, bacilli obtained from certain cases of human tuberculosis produced in cattle lesions which could not be differentiated from those occurring in bovine tuberculosis.

Other Interim Reports appeared in 1907 and 1909, and the final report was issued in 1911, dealing with the whole of the inquiry into tuberculosis of man and the various animals, also with the avian form of the disease.

As already mentioned (see p. 112), three types of tubercle bacilli were distinguished—viz., the human, the bovine, and the avian type.

The bovine type, although morphologically indistinguishable from the human type, was identified by its cultural characters, taken into consideration with the effects produced by inoculation into calves and rabbits, and it was recognised as the only type of tubercle bacillus present in bovine tuberculosis.

Amongst the conclusions arrived at were the following:

"The bovine tubercle bacillus produces a fatal tuberculosis in cattle, rabbits, guinea-pigs, chimpanzees, monkeys, goats, and pigs."

"The human tubercle bacillus readily produces a fatal tuberculosis in guinea-pigs, chimpanzees, and monkeys, but causes, even when administered in large doses, only slight and non-progressive lesions in cattle, goats, and pigs."

"In other words, we have always found that guinea-pigs, chimpanzees, and monkeys, are all highly susceptible to the effects of either the human or the bovine tubercle bacillus, and that the disease produced in these animals by both types is histologically and anatomically identical."

"We have investigated many instances of fatal tuberculosis in the human subject in which the disease was undoubtedly caused by a bacillus of the bovine type, and by nothing else. We have compared the lesions from such cases with those obtained from parallel cases of fatal tuberculosis in which the human tubercle bacillus alone was discovered. Except for the difference in the type of bacillus found in them, these two groups of cases presented similar features; the clinical histories of the patients were alike, the cases all terminated fatally, and the lesions examined after death were found to be anatomically indistinguishable."

"Man must therefore be added to the list of animals notably susceptible to bovine tubercle bacilli. . . ."

"Our researches have proved that in a considerable number of cases of the human disease the lesions contain, and are caused by, bacilli which are in every respect indistinguishable from the bacilli which are the cause of tuberculosis in cattle. In all such cases the disease, therefore, is the same disease as bovine tuberculosis. . . ."

In connection with this subject it is important to note that in twenty-nine cases of primary abdominal tuberculosis in man which were investigated, fourteen yielded bovine tubercle bacilli and thirteen human tubercle bacilli, and two contained a mixture of the human and bovine type of bacilli. In two cases of primary pulmonary tuberculosis in man the bovine type of bacillus only was found in the sputum, and it was concluded that the bovine bacillus may be responsible for some cases of tuberculosis in man which primarily involve the lungs, as well as those cases in which the disease primarily affects the alimentary canal and its associated glands.

In nine cases of cervical gland tuberculosis in man, three yielded bovine tubercle bacilli.

In ten cases of lupus, three yielded the bovine type of tubercle bacilli so far as the cultural characters were concerned, but these bacilli possessed less virulence for the calf and rabbit than the ordinary bovine type.

With regard to the dangers of transmission of tuberculosis from swine to man, it was found that in the great majority of cases of the disease in the former species the bovine type of bacillus was present, while in a few instances the human and avian type were observed.

Hence the conclusion: "Bovine tubercle bacilli are infective to human beings, and we have no reason to suppose that they can be rendered less so by previous residence in the bodies of pigs."

From the above extracts it is apparent that an efficient system of inspection of meat and of dairies is of the highest importance in preventing the transmission of tuberculosis from animals to man.

Referring first to the inspection of meat, we may remark that it forms a special study in itself, and requires a definite course of training. For full information on the subject the reader is referred to the many excellent works on meat inspection which are now published.

The Royal Commission on Tuberculosis, in its Report issued in 1898, recommended that the following principles should be observed in the inspection of carcasses which present tuberculous lesions:

The entire carcass and all the organs may be seized—

1. When there is miliary tuberculosis of both lungs.
2. When tuberculous lesions are present in the pleura and peritoneum.
3. When tuberculous lesions are present in the muscular system or in the lymphatic glands, embedded in or between the muscles.
4. When tuberculous lesions exist in any part of an emaciated carcass.

The carcass, if otherwise healthy, shall not be condemned, but every part of it containing tuberculous lesions should be seized—

1. When the lesions are confined to the lungs and the thoracic lymphatic glands.
2. When the lesions are confined to the liver.
3. When the lesions are confined to the pharyngeal lymphatic glands.
4. When the lesions are confined to any combination of the foregoing, but are collectively small in extent.

In view of the greater tendency to generalisation of tuberculosis in the pig, the presence of tubercular deposit in any degree should involve seizure of the whole carcass and of the organs. In respect of foreign meat, seizure shall ensue in every case where the pleuræ have been "stripped."

With reference to the risks of infection to man from avian tuberculosis, the conclusion arrived at by the Commissioners, as stated in the final Report (1911), was as follows:

"We must conclude, as the result of our investigations, that the unmodified avian tubercle bacillus is a negligible factor in the production of human tuberculosis."

INSPECTION OF MILK AND OF DAIRIES.—This forms one of the most important duties of the veterinary officer of health. From the knowledge we now possess with reference to the dangers of transmission of tuberculosis from animals to man through the medium of milk containing tubercle bacilli, it is apparent that the most rigid inspection of dairy cows and their surroundings is essential for public health. In addition to the source of infection from tuberculous lesions of the udder, we must take into consideration that in cases of "open" tuberculosis there is danger of bacilli entering the milk through the discharges from the affected animal, more especially when the milker, as so frequently happens, is careless and ignorant of ordinary precautions and of cleanliness.

In the final Report of the Royal Commission on Tuberculosis (1911), some important conclusions are found with reference to the dangers arising to human beings from the consumption of milk derived from tuberculous cows; these conclusions also appeared in the Third Interim Report, and are as follows:

"Bovine tubercle bacilli are apt to be abundantly present in milk as sold to the public when there is tuberculous disease of the udder of the cow from which it was obtained. . . . But these bacilli may also be present in the milk of tuberculous cows presenting no evidence whatever of disease of the udder, even when examined post-mortem. Further, the milk of tuberculous cows not containing bacilli as it leaves the udder may, and frequently does, become infective by being contaminated with the fæces or uterine discharges of such diseased animal. We are convinced that measures for securing the prevention of ingestion of living bovine tubercle bacilli with milk would greatly reduce the number of cases of abdominal and cervical gland tuberculosis in children, and that such measures should include the exclusion from the food-supply of the milk of the recognisably tuberculous cow, irrespective of the site of the disease, whether in the udder or in the internal organs."

The above conclusions are of vast importance, as hitherto there was a prevalent idea that, provided no lesions were present in the udder, the milk was not infective. They also emphasise the necessity of adopting the tuberculin test, more especially in the case of private owners where only one cow is kept to supply milk. If such an animal is suffering from the disease, it is clear that the risks of infection are intensified. Moreover, the general appearance of the cow cannot be relied on as a guarantee that she is free from the disease; the results of the tuberculin test and also of post-mortem examinations demonstrate what extensive lesions may be present without any evidences of their existence being shown during life. Again, we have experience of milch-cows that were regarded as types of healthy animals, and suddenly developed acute pulmonary symptoms without apparent cause, evidences of clinical tuberculosis rapidly appeared, and extensive lesions were found on post-mortem.

From our present knowledge we must regard such cases as distinctly dangerous, and advise that, more especially when a milch-cow is kept for the supply of milk for children, the animal should be tested with tuberculin.

This plan is now being adopted by many private owners, who recognise the dangers of infection from milk, and they also insist on the test being applied prior to the purchase of every new cow.

As the public become better educated in such matters, it is highly probable that the adoption of the tuberculin test will become universal in all dairies.

Unfortunately, at present sufficient importance is not attached to the matter by the public, while the owners of dairies object in many instances to the employment of the test, fearing a considerable loss should a number of the cows react.

We must emphasise the importance of having every new cow added to a tubercle-free dairy tested with tuberculin, as otherwise a source of infection may be introduced.

With reference to the danger arising to man from the ingestion of butter and cheese prepared from milk infected with tubercle bacilli, the investigations of Continental authorities—also of the U.S. Bureau of Animal Industry (1909)—would indicate that these products prove virulent. Petri (1898) examined 102 samples of butter, and found that 32·4 per cent. contained tubercle bacilli. Other observers found that the percentage varied from 2·12 to 32·4. Constant storage at an icy temperature, or the addition of salt, does not affect the virulence; even salted butter retained its virulence for six months. The bacillus of Rabinowitsch was frequently found in company with the tubercle bacillus in the samples examined.

In cheese the tubercle bacillus survives for a long or short period, according to the mode of manufacture. According to Harrison,* the bacillus is killed in 33 to 44 days in Emmenthal cheese, while it disappears after 65 days in Canadian Cheddar, and its virulence is only lost after 90 days in cheese manufactured in Berne. According to Galtier, living tubercle bacilli are found even after eleven months in certain cheeses. In some of the American experiments, the infected cheese employed was 261 days old, yet it was found that even at this age the tubercle bacilli retained sufficient vitality to cause slight tuberculous lesions in one of the fed and in four of the inoculated test animals.

Margarine may also prove a source of infection ; Morgenoth (Berlin), out of ten samples examined, found that eight were infected. On another occasion one out of ten was infected. Annett (Liverpool, 1900) examined thirteen samples, and found tubercle bacilli in one of them.

In conclusion, we think it is of importance to emphasise the dangers that are likely to arise from the presence of tuberculous dogs, cats, and parrots in dwelling-houses. From a public health point of view this matter has not received the attention it merits. The existence of the disease in these species is frequently overlooked, and when we consider the intimate relations between them and human beings, especially in the case of pet dogs, it is clear that the practitioner should not hesitate to point out the risks when any suspicious instances come under his notice. We have sufficient evidence to prove the transference of the disease from man to these species. Instances are recorded by Gray in which he has observed the presence of tuberculosis in these species when the owners or inmates of the same house were tubercular. Hence it is reasonable to conclude that infection may occur in the opposite direction, although we may not be able to offer specific instances in support of this view.

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* *Centralblatt für Bakteriologie*, Band. xxxi., 1902, p. 250.

JÖHNE'S DISEASE

By G. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.,
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Synonyms.—Chronic bacterial enteritis of cattle; Specific chronic enteritis of cattle.

French : L'entérite chronique spécifique des bœufs.

German : Die spezifische chronische Enteritis des Rindes; Der Infektiöse Darmkatarrh des Rindes; Die spezifische hypertrophische Darmentzündung des Rindes.

Definition.—A contagious chronic inflammation of the intestines of ruminants, particularly bovines, characterised by great emaciation and a usually persistent diarrhœa.

This disease appears to have been first described by Jöhne and Frothingham in 1895, and next by Markus in 1904. Since that date it has been described as occurring with a rather wide distribution on the continent of Europe, the British Isles, the Channel Isles, and North America, by numerous writers, including B. Bang, Liénaux, van den Eeckhout, Bougert, Stuurman, Miessner, Leclainche, Bugge, McFadyean, Stockman, O. Bang, Pearson, and others. The disease affects cattle principally; Stockman has described it as affecting sheep in Scotland, and McFadyean has recorded a case in a deer. The small experimental animals (rabbits and guinea-pigs) appear to be insusceptible.

ETIOLOGY.

Jöhne's disease is due to a small acid-fast bacillus, varying in length from 1 to 4 microns. When stained with carbol-fuchsin and treated with 25 per cent. solution of sulphuric acid (Ziehl-Neelsen process), many of them stain uniformly, while others present a granular appearance, in this respect resembling the tubercle bacillus. On account of this morphological similarity, some investigators have expressed the view that the bacillus of Jöhne's disease is the same as the bacillus of tuberculosis

(Liénaux, van den Eeckhout, Bougert). This view, however, cannot be supported, since the organism is non-pathogenic for rabbits and guinea-pigs. Moreover, this disease exists in the Channel Isles, where bovine tuberculosis is non-existent. In those cases where tubercles have resulted from the inoculation of material from cases of Jöhne's disease, it is probable that there was a mixed infection (McFadyean).

Attempts to obtain the bacillus in artificial culture have proved negative on the part of most investigators, but Twort and Ingram have obtained pure cultures in a medium on which the former was able to cultivate the lepra bacillus of man.

Twort* explains his method as follows: "In view of the close relationship between the tubercle bacillus and the lepra bacillus, it appeared highly probable that these two organisms would require the same chemical substances for building up their protoplasm, which could be elaborated from the ordinary media only by the tubercle bacillus. It was thought that if these substances could be supplied already formed to the lepra bacillus it might grow, and the easiest method of supplying these substances would be by adding to some good medium the ground-up bodies of tubercle bacilli containing them. Accordingly, a number of tubercle cultures were taken and inoculated on to Dorset's egg medium. When sufficiently grown, the tubes were steamed, and the growth of tubercle scraped off the surface, care being taken to avoid the medium containing the waste products of tubercle growth. The tubercle bacilli so obtained were ground up with glycerine and saline, steamed for half an hour, and added to the yolk and white of new-laid eggs in the following proportions: Eggs, 75 parts; 8 per cent. sodium chloride, 25 parts; mix well, and add tubercle bacilli 1 per cent., and glycerine 5 per cent. or less. The medium was placed in test-tubes, heated to 60° C. for one hour, and on the following morning incubated at 38° C. for six hours, and again heated in a water-bath at 60° C. for one hour, and set in slopes at 58° C." Referring to the growth of the bacillus of Jöhne's disease, Twort continues: "Working with Ingram on the same medium, I have succeeded in isolating and growing the acid-fast bacillus found in the intestine of cows in Jöhne's disease. The first generation of this bacillus grows often long, with occasional branching and club formation; in subcultures it gradually grows smaller, and in the second or third generation is about the size of the tubercle bacillus. The growth is only just visible to the naked eye, and subcultures on ordinary laboratory media show no evidence of multiplication. The cultures were incubated at 40° C."

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* Twort, *Veterinary Journal*, February, 1911. Reprinted from Proceedings of the Royal Society, B., 1910, vol. lxxxiii.

With these cultures Twort and Ingram claim to have produced the disease in bovines by ingestion and also by intravenous inoculation. In each case they were able to recover the organism from the intestinal lesions.

Since Twort and Ingram reported as above, they have experimented with numerous acid-fast bacilli to see if other organisms might be used instead of the tubercle bacillus in the preparation of their medium, and they have found that the Timothy grass bacillus, dried over caustic potash *in vacuo*, and ½ per cent. added, well powdered, to Dorset's egg medium, gives the best results of all (personal communication to the writer).

MORBID ANATOMY.

The lesions consist of a specific enteritis, affecting both small and large bowels. The small intestines are mainly affected, particularly the ileum, while the duodenum may even escape. In mild cases it may be difficult to observe any macroscopic change, but in more severe instances the bowel wall immediately gives the impression of being thicker than normal. On exposing the mucous membrane, the latter shows a more or less wrinkled or corrugated appearance, which is most obvious in the last portion of the ileum. In the large intestine the wrinkled appearance is rarely so marked as in the ileum. In no portion of the intestine is congestion a marked feature. In those cases where the bowel has been suitably fixed soon after death there is no loss of superficial epithelium, and no evidence of ulceration. Desquamation of epithelium and necrotic appearance of villi are only post-mortem changes. The mesenteric lymphatic glands are usually somewhat larger than normal, and on being cut into have a juicy or œdematous appearance.

On microscopic examination in advanced cases the villi appear to be distorted as the result of formation in the submucous coat of epithelioid cells, amongst which may be seen occasional giant cells. Varying numbers of acid-fast bacilli are observed in and between these cells, and may also be found in scrapings from the surface of the mucous membrane. Sometimes they are very numerous and in clumps, while in mild cases they may be difficult to discover. Similar histological changes and bacilli may be found in the lymphatic glands.

The situation of the lesions suggests that the natural method of infection is by the ingestion of food or water contaminated with fæcal discharges from previous cases.

SYMPTOMS.

The first symptom to be observed in this disease is loss of condition and a diminution of the milk yield, in spite of a good appetite and a plentiful supply of food. The unthrifty condition is manifested by a slowly progressive emaciation and a staring, harsh coat. The animal may remain in this state for a considerable time—a year or more—fluctuating in condition, with perhaps occasional relaxation of the bowels. The temperature varies—sometimes normal, sometimes elevated several degrees. Eventually the diarrhœa becomes profuse and persistent, and often fœtid, and in such cases recovery is exceedingly rare. Parturition has frequently a very marked effect, and sometimes cows not suspected of being affected with the disease suddenly develop it in its acute diarrhœic form after calving, waste away rapidly, and die in a few weeks. Feeding and rumination may continue up to the last day or two, and death is due to exhaustion from the diarrhœa. In sheep the symptoms correspond exactly with those of cattle affected—namely, emaciation, followed by diarrhœa.

PROGNOSIS.

As a rule prognosis is unfavourable. An early diagnosis where possible is of the greatest importance in this disease, owing to the fact that it is not very amenable to treatment, and it becomes a question as to the advisability of slaughter or treatment of the animal affected. It is doubtful if recovery ever occurs, though it is possible that there are mild non-fatal cases. In those cases, taken in hand early, the diarrhœa and emaciation may be checked by astringents and dry, highly nutritious foods. Such animals may continue to exist for a year or more, but when diarrhœa is well established death is usually only a matter of weeks.

DIAGNOSIS.

There is no doubt that in former times animals affected with this disease were included in the general description of " wasters " or " piners," the other conditions included being tuberculosis and parasitic gastro-enteritis. A positive diagnosis can sometimes be made by microscopic examination of fæces, which should be obtained direct from the mucous membrane of the rectum. If on staining by the Ziehl-Neelsen method one finds acid-fast organisms arranged in groups or clusters, it may be taken as certain that the animal from which the specimen is taken is affected with Jöhne's disease. It is not, however, always easy to discover the organisms in affected animals, and therefore failure to demonstrate them does not justify a negative diagnosis.

O. Bang, of Copenhagen, succeeded in finding an apparently trustworthy diagnostic agent in Koch's old tuberculin prepared from avian tubercle bacilli, and corresponding exactly with the old tuberculin test for tuberculosis. Affected animals give a thermal reaction of from 2° to 4° F. between the ninth and fifteenth hours after hypodermic injection of the agent, the temperature gradually falling to normal by the twentieth or twenty-fourth hour. It is advisable to first submit the suspect to the ordinary bovine tuberculin test a week or so before applying the avain test, since tuberculous animals may react to the latter. The converse, however, does not hold, so that if a suspect gives a negative result to the bovine tuberculin test, and subsequently a positive reaction to the avian tuberculin test, the animal may be regarded as affected with Jöhne's disease. For obvious reasons this test is not applicable in the case of animals with a febrile temperature, but Horne of Christiania has reported a positive result to the conjunctival instillation of avian tuberculin.

To differentiate the condition from tuberculosis, moreover, the absence of cough, etc.,. assists. In parasitic gastro-enteritis the presence of numerous ova of nematodes in the fæces assists in diagnosis. Age and breed are not of any assistance, for all breeds, whether old or young, are susceptible to each condition.

TREATMENT AND PREVENTION.

As will have been gathered from what has already been said under the heading of Prognosis, medicinal treatment does not meet with much success. There is little doubt that if an animal has exhibited clinical symptoms, the best plan to adopt is to slaughter at once, in order to diminish the risks of spreading infection. The diarrhœa may be checked for a few days or weeks by means of intestinal astringents and disinfectants, and by feeding on dry, highly nutritious foods containing decorticated cotton cake. Opium, catechu, and prepared chalk in combination, are useful in some cases, salol in others, while Male claims to have obtained the best results by the administration of Cyllin in drachm doses, in water, once or twice daily. It is, however, impossible to improve the emaciated condition, and it is doubtful whether there is any practical advantage obtained beyond prolonging the animal's life. Such a proceeding could only be warranted from an economical standpoint if the cow was in an advanced stage of pregnancy, and the expected calf was, by virtue of its pedigree, likely to prove a valuable one.

On the other hand, however, when diagnosis has only been made by the use of avian tuberculin or other diagnostic means, and where no clinical symptoms have been evinced, then such animals may be isolated and housed, and an attempt made to fatten them for the butcher. All the excreta from affected animals, and all soiled litter, should either be destroyed or spread on arable land and ploughed in deeply. Disinfection of houses and premises should also be as thorough as possible.

The great difficulty to be overcome in any attempt to exterminate the disease from any herd or locality is due to the fact that animals may be affected and spread the malady, although showing no clinical symptoms. Reliance must be placed on some diagnostic agent, such as avian tuberculin, and the disease systematically attacked, as in the case of other contagious diseases. Cognisance must also be taken of the fact that, as Stockman has recorded, sheep are susceptible to infection, and may be factors in the spread of the disease. Any attempt to exterminate the disease from a farm, therefore, must include attention to sheep, slaughter being probably the best plan of action should any animals prove to be infected. The pastures where affected animals have been grazing should be left vacant of ruminants for some time, probably months being required. It is impossible, however, to state definitely the period required for safety, pending some knowledge as to the length of time the organism may exist and remain infective outside the animal body.

MEAT INSPECTION.

On the question of fitness or otherwise for human food there is little to be said. So far as is known, the organism is non-pathogenic to the human being, and its habitat in an infected animal appears to be limited to the intestines and the corresponding lymphatic glands. The question must therefore be decided on the general grounds of the condition of the carcass. If it shows advanced emaciation and the tissues are dropsical, such as would be the case in animals killed in advanced stages of the disease, the carcass should be *ipso facto* condemned as unfit. If, however, the carcass was reasonably nourished, and the fat and other tissues set firmly, the carcass may be passed. The intestines and their lymphatics should be destroyed, and the former should not be used for sausage-skins.

PSEUDO-TUBERCULOSIS

By E. WALLIS HOARE, F.R.C.V.S., and G. L. INGRAM, M.R.C.V.S.

This term is applied to certain affections which present lesions resembling in their macroscopic appearances those of tuberculosis, but not caused by the tubercle bacillus. These lesions generally consist of nodules in the internal organs and lymphatic glands, which tend to undergo early caseation.

The most important of the diseases coming under the heading of "pseudo-tuberculosis" is the affection in sheep known as "caseous lymphadenitis." This we shall describe later on (see p. 208). Here we shall draw attention to the pseudo-tuberculous affections which have been met with in other animals and described by various authorities.

According to Ostertag, the causal agent in these types of pseudo-tuberculosis is multiform—thus, micrococci, bacilli, mould fungi, and certain parasites, are capable of inducing the lesions; but Preisz and Guinard state that all the bacterial forms of the disease depend on an identical micro-organism. In cattle a caseous broncho-pneumonia is described by Kitt, in which the causal agent was the bacillus of Preisz. Courmont described nodules in the pleura of cattle due to a bacillus (unnamed), and Stöhr observed a caseous pneumonia in sucking calves, and isolated a bacillus therefrom. Hinks described a case in a large ruminant in which tubercle-like formations were found in the lungs, caused by *Aspergillus fumigatus*. Certain parasites (strongyli) are capable of producing nodules in the lungs of cattle and sheep, and the lesions were erroneously termed "nematode tuberculosis."

In Argentine cattle, nodules in the small intestine due to sclerostomum larvæ are met with. A form of pseudo-tuberculosis in sheep and goats caused by *Strongylus rufescens* is described by De Jong, and nodules in the livers of pigs were found by Deleïdi to depend on a similar parasite. In the kidneys and lungs of dogs and cats nodules caused by cylindrically shaped worms were described by Ebstein and Nicolaïer, and Laulanie found similar nodules produced in the lungs by *S. vasorum.*

Mégnin and Mosny describe cases of pseudo-tuberculosis in the hare;

Malassez and Vignal met with cases in chickens ; and Hayem and Toupet record instances of the disease in man. According to Osler, pseudo-tuberculous processes are very rare in human beings. Cases are described by Flexner in which the lungs presented the appearance of a caseous pneumonia, and numerous tubercle-like nodules existed in the peritoneum. The micro-organism found in the lesions was a streptothrix, which differed greatly from the *Streptothrix actinomyces* and from the known forms of the tubercle bacillus.*

Silberschmidt observed the disease affecting the lungs of a goat, and described a pathogenic species of streptothrix as the causal agent. In guinea-pigs and rabbits pseudo-tuberculosis was described by Eberth, Pfeiffer, and others. Pfeiffer isolated the *Bacillus pseudo-tuberculosis*, and claimed that it was the causal micro-organism of the disease in these animals. Pseudo-tuberculosis of the mouse and rat was described by Preisz.

According to Schwäbel, a nodular pseudo-tuberculosis in cattle occurs in France, Germany, Denmark, and Russia. Animals of both sexes and of all ages were affected, and there was an absence of clinical symptoms. Only three cases were recorded by this observer in which the presence of the nodules was observed during life. The predilection seats of the disease were the subcutaneous tissues and the superficial muscles.

Hungerbühler† investigated the disease, and found that the majority of the nodules were situated in the skeletal muscles, and occurred with great frequency in the subcutaneous tissue ; but they were not limited to these locations, as they were also discovered in the nasal cavity, in connection with the tongue, in the frontal sinus, trachea, lungs, liver, udder, testicles, and tunica vaginalis. He also found that tuberculous lesions were frequently coexistent, and stated that when tubercle bacilli were discovered in the nodules they were derived from the true tuberculous lesions that were present. The macroscopic characters of the lesions did not resemble in any way those of tuberculosis, and even when the nodules were abundant in the muscles the nearest lymphatic glands showed no signs of disease. Experimental inoculation gave negative results as regards tuberculosis. The presence of a micro-organism was demonstrated in the lesions by staining, inoculation, and cultural experiments, and this micro-organism was shown to be capable of producing nodular suppuration in animals. There was an entire absence of acid-fast bacilli in the cases examined, and, except in one instance, the specimens contained but few bacteria of any kind.

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* Osler's "Principles and Practice of Medicine."

† Hungerbühler, "Inaugural Dissertation at the Pathological Institute in Stuttgart, 1910." Translated in the *Journal of Comparative Pathology and Therapeutics*, March, 1911.

It was not decided whether the micro-organism discovered was capable, either *per se* or in association with other bacteria, of causing the nodule formation, or whether the degree of virulence exerted any influence on the character and extent of the lesions.

The micro-organisms discovered were of various types, such as micrococci, diplococci, short chains of streptococci, and small organisms rather longer and thicker than the bacillus of swine erysipelas. In one case the bacilli closely resembled the *B. pyogenes*, and were present in large numbers, either singly or in clumps, in the necrotic centre, the fibroblast zone, and the foci of rounded cells. In another instance the bacillus, in view of its biological characters, appeared to be identical with the *B. pyogenes* and the Preisz-Nocard bacillus.

As regards the histology of the lesions, Hungerbühler examined fourteen cases of the disease, and found " that the smaller nodules showed an outermost zone composed of richly cellular fibrous tissue, the matrix being more distinctly fibrillated towards the outer part. On the inner side of this zone the cells could hardly be distinguished from epithelioid cells. Here and there were groups of lymphocytes, and bloodvessels were numerous. In some sections atrophied muscle fibres were seen enclosed in the connective-tissue zone, these fibres still showing well-marked transverse striation. . . . The centre of the nodules was composed of a homogeneous, or, in some cases, a laminated fibrous matrix, staining intensely with eosin. Degenerative changes were well marked. In this matrix, even in the smallest nodules, there were numerous round or oval abscess-like collections of rounded cells. . . . Bloodvessels were, as a rule, absent in the degenerated areas. A slight degree of calcification was present in some of the nodules. Small, well-defined nodules were frequently found around the larger ones. Some of the larger nodules were distinctly fibrous throughout. The histology of the nodules suggested that the process was one of chronic inflammation. The lesions were distinguished from true tuberculous lesions by the presence of foci of round cells in the necrotic centres, the absence of epithelioid and giant cells, and the small amount of calcification."

Rockl studied the histology of the lesions, and included them under the general term " granuloma." Liénaux examined the lesions in a case of the disease, and found that the nodules resembled tubercles, as their central parts were composed of epithelioid cells surrounded by connective tissue containing lymphocytes. Dürbeck described a case in which the nodules were composed of multiple cellular foci, partly confluent and partly necrotic, surrounded by an abundant fibroblastic layer containing leucocytes. Foulerton described the histology of the nodules as "that of granulomata in which there is no evidence of caseation or giant-cell formation."

Schwäbel described nine cases, in six of which the lesions were identical. In these six cases he recognised that in the nodules three zones could be distinguished—viz., "a peripheral connective-tissue zone, a zone composed of cells which had large cell bodies and clear vesicular nuclei, and a central area composed of a degenerated tissue in which a number of cellular foci were enclosed."

In the seventh case the retrogressive changes were more marked, and in the eighth case the mono- and polynuclear epithelioid cells were more numerous than in the first six mentioned, and multiple young foci were included in the same capsule. In the ninth case "the nodules were also compound, epithelioid and giant cells were numerous, and the degenerative changes already described produced an appearance suggestive of a large tubercle."

As regards the location of the lesions, which could be observed during life, the nodules, in a case occurring in a six-months-old calf, were numerous, up to the size of a bean, and scattered over the body just under the skin. Those occurring below the knees and hocks were firmly attached to the skin, and the subcutaneous tissue was thickened. Nodules of various sizes were found in the subcutaneous tissue, and the superficial muscles of the face, sides of the neck, chest wall, back, loins, croup, limbs, and tail.

In some cases nodules were found in the subcutaneous tissues and superficial muscles from all parts of the body. The favourite seats appeared to be the muscles of the back, shoulders, loins, and tail.

Hungerbühler directed attention to the differential diagnosis of the disease, and pointed out that in cases of multiple sarcomata in cattle numerous nodules may be firmly embedded in the muscles and partly adherent to the skin, and simulate the superficial lesions of pseudo-tuberculosis. The sarcomatous growths, however, are soft in consistence, and are characterised by a rapid and unlimited growth. A microscopical examination will complete the diagnosis.

I. PSEUDO-TUBERCULOSIS IN THE CALF.

By H. GRAY, M.R.C.V.S.

Definition.—A disease occurring in young calves about a fortnight old, and showing on post-mortem examination nodular lesions confined to the liver, all other organs being healthy, and due to a special bacillus resembling that of swine erysipelas.

History.—This disease was studied by Vallée in France in 1898, where it occurred yearly as an enzoötic on a particular farm.

Bacteriology.—The pulp of the diseased liver contained, besides other microbes, a fine bacillus in abundance, which retained the coloration of Gram. In sections coloured by Gram-Nicolle the same microbe was present. The bacillus is much finer than that of swine erysipelas, and is often found singly or sometimes in small masses confined to the lesions, in the centre of which it is indifferently distributed. It is not found in those tissues free from lesions. It is immobile, scarcely longer than broad, has rounded extremities, and is sometimes slightly curved. It is frequently found in the form of a diplococcus, but never in that of a streptococcus. It is a facultative anaërobe, and does not form spores. The cultures are pathogenic for the calf, guinea-pig, rabbit, and pigeon.

Etiology.—Infection takes place either by the umbilicus or by the alimentary canal. Experimental transmission by ingestion indicates that probably invasion is effected by the alimentary canal, the bacillus being taken in by milk contaminated in the udder or by accidental soiling.

Symptoms.—The calf is attacked one or two weeks after birth by symptoms of extreme weakness and total loss of appetite, and dies within four to eight days without manifesting any other phenomena beyond these.

Lesions.—All the organs appear normal except the liver. This organ retains its normal size, but its surface is crammed with innumerable fine greyish, tubercle-like granulations, which often become confluent. The glands of the hilum are not hypertrophied. The hepatic tissue is easily torn. The lesions are disseminated in the whole substance of the organ. The histological study shows in the centre of the foci an absolute degeneration of the glandular elements, with infiltration of the destroyed phagocytes, and at the periphery a zone of reaction.

Prevention.—The disease disappeared after a thorough disinfection of the shed, and feeding the calves on boiled milk.

Bibliography: Vallée, Sur une Nouvelle Pseudo-Tuberculose observée sur les Jeunes Animaux de l'Espèce Bovine, Recueil de Méd. Vét., 1898, p. 400; and Nocard et Leclainche, Les Maladies Microbiennes.

II. PSEUDO-TUBERCULOSIS IN SHEEP.

Synonyms. — Caseous lymphadenitis; Lympho-adenitis; Caseous adenitis.

French: Maladie caséeuse du mouton; Broncho-pneumonie caséeuse du mouton.

German: Pseudotuberkulose bei Schafes.

GENERAL REMARKS.

This affection deserves special consideration in consequence of its frequency in certain countries and the fact that it can easily be mistaken for tuberculosis; hence its importance in connection with meat inspection. The disease cannot be regarded as a serious one so far as the effects on the living animals are concerned. Gilruth, who has made a special study of the affection in New Zealand, states that, "so far as he is aware, the disease, when contracted naturally, is never fatal. At most, in long-standing cases, a condition of cachexia results." This observer has, in an excellent article on the subject,* fully described the etiology, bacteriology, and pathology of the disease, and to this article we are indebted for the material in this section.

Preisz and Guinard described the disease in France in 1891. In 1897 it was met with by Turski of Dantzig; in 1899 it was investigated by Cherry and Bull of Melbourne;† and in the same year by Nörgaard and Mohler in the United States.‡

GEOGRAPHICAL DISTRIBUTION.

The disease is common in New Zealand, Australia, the Western States of America, and the Argentine. It has also been met with in Germany France, Belgium, and Italy. According to Sivori, 10 per cent. of the sheep slaughtered in Buenos Ayres show the lesions of the disease.

According to Carré,§ the disease is very common in certain regions of France. In the region of Valreas, south of La Drôme, and north of Vaucluse, where lambs are raised for the Paris markets in large numbers,

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* *Vide Journal of Comparative Pathology and Therapeutics*, December, 1902.

† *Veterinarian*, vol. lxxii., 1899.

‡ Report of United States Bureau of Animal Industry, 1899, and *Veterinary Journal*, vol. liii., 1901.

§ Société Centrale de Méd. Vét., Séance du Novembre, 1910.

7,000 to 8,000 lambs out of 30,000 were attacked yearly. In certain flocks the proportion affected was 60 per cent.

Kerrigan (Canterbury, New Zealand) stated that, from the examination he made of a large number of lungs, he believed that 40 per cent. of the sheep passed through Fairfield are affected with nodules in the lungs. In another report the same observer remarked that the caseous nodules in the lymphatic glands occurred in 3½ per cent. of the sheep examined. Edgar found the average number affected to be 1 in 1,000 sheep.

Judging by the replies to inquiries we have made from the veterinary officers of health at some of the large abattoirs, the disease appears to be of comparatively rare occurrence in the British Isles.

ANIMALS SUSCEPTIBLE.

Although the disease may be found in any breed of sheep, it is admitted by all observers that the merino is more prone to be affected. According to Kerrigan, " the nearer the sheep is bred to the merino, the greater is the percentage of animals affected. . . . I have seen a thousand sheep from a certain place go through without the sign of a nodule; while, again, another flock of wethers from the same place and owner, but bred more to the merino, have been found affected to the extent of 3½ per cent. . . . The percentage of animals affected varies very much, but I might particularly mention that the old merino ewes have to be carefully inspected."

Edgar states that the disease was more prevalent among aged ewes and " broken-mouthed " sheep than in any other class. Lambs are the least affected.

The number of sheep and lambs condemned as unfit for food from May, 1901, to March, 1902, was given by Kerrigan as follows:

Lambs: 138,691 slaughtered, 29 condemned.
Sheep: 50,639 slaughtered, 89 condemned.

But in France, according to Carré, lambs are especially affected.

According to the results of experiments carried out by Gilruth, the disease can be transmitted to sheep, goats, guinea-pigs, and rabbits, but cattle appear to be immune.

In the outbreak described by Turski of Dantzig, out of 150 breeding ewes, 44 were affected to a greater or less extent.

ETIOLOGY.

The majority of observers state that the causal organism of the disease is the bacillus of Preisz (also known as the " Preisz-Nocard bacillus "), or at least a bacillus corresponding almost exactly with it.

This bacillus is also believed to be the causal organism of ulcerous lymphangitis (pseudo-glanders) in the horse, and of contagious pustular dermatitis in the horse and ox.

Nocard and Leclainche term this organism the "*bacillus of caseous suppuration.*" According to Carré, the microbe is polymorphic in cultures and also in the pathological products, and the term "Preisz-Nocard bacillus" should not be applied to it, as in the pus from the lesions in sheep the organisms are generally in the form of cocci arranged in groups. On the other hand, in ulcerous lymphangitis the bacillary form predominates.

Gilruth described the characters of the organism investigated by him as follows: "A short, irregular-shaped bacillus, varying in size and appearance from a coccus to a very short bacillus—*i.e.*, from 0·5 to 1 μ in length, and about 0·3 μ in breadth. In the pus of an early abscess the bacilli are frequently within more or less degenerated leucocytes, especially those in scrapings from near the periphery of the abscess or the lining of the fibrous capsule. In the centre of an old abscess the bacilli are usually very degenerated and irregular, occasionally being quite absent. Frequently the organisms are found in pus in the form of chains, which may almost be mistaken for streptococci, though careful examination demonstrates that these chains are composed of short cocco-bacilli, extremely small even in their longest diameter, and arranged side by side, not end to end. The bacilli are readily stained by the method of Gram. . . . The best method of demonstration is to stain the pus by Gram's method, and counterstain with eosin or weak fuchsin."

The bacilli isolated by Cherry and Bull in Australia had the same characteristics, and morphologically the bacillus isolated by Nörgaard and Mohler in America agreed in most respects with the organism described by Gilruth. Judging by experiments, however, the bacillus in the American disease seemed to be less virulent for rabbits, guinea-pigs, and sheep, than the organism isolated by Gilruth. As the result of many experiments, Gilruth obtained valuable information concerning the pathology of the disease.

He found in his first experiments that the early cultures were very virulent to guinea-pigs, rabbits, and even sheep. Passage through rabbits caused an evident diminution of the virulence. One sheep was inoculated in the flank with 0·5 c.c. of a three-days-old culture in serum condensation liquid. The animal succumbed in thirty-six hours, and the post-mortem showed a small amount of subcutaneous œdema at the seat of inoculation; the spleen was enlarged and pulpy, the contents being of a tarry consistency; the abomasum showed on its mucosa small patches of inflammation, each with its centre slightly ulcerated; the pleural cavity contained about a quart of serosanguineous effusion. No organisms were found in any of the tissues, beyond a small number present in the subcutaneous œdema at the seat of inoculation. "Tubes inoculated with the subcutaneous effusion developed the characteristic growth of the bacillus in a state of purity." The experiments on the other sheep were as follows:

No. 1 was inoculated in the thigh with 0·1 c.c. of pus taken from an affected lymph gland. Only slight lameness resulted for two days. The animal was killed four months later, and the characteristic lesions of the disease were found in the popliteal, precrural, and pelvic glands of the same side, and in the lungs. The pleuræ and thoracic lymphatic glands were normal. The specific organism was isolated from the affected glands and from the lung lesions. At the original seat of inoculation a small mass of greenish caseous matter was present, surrounded by a fibrous capsule ¼ inch thick.

No. 2 was inoculated on the inner surface of the fore-leg, above the carpus, with 0·05 c.c. of a three-days-old culture in liquid serum from an affected guinea-pig. After slaughter, three months later, the animal was found in excellent condition; the usual lesions of the disease were found, but in addition subcutaneous abscesses were observed extending round the leg at the seat of inoculation, and the lymphatic glands of the limb were involved.

No. 3: The sheep was fed, after a twenty-four hours' fast, with 4 c.c. five-days-old liquid serum culture made from original culture. On being killed four months afterwards, the left prepectoral gland was enlarged to the size of a marble, with a pea-sized centre of typical greenish pus surrounded by the usual capsule. Gilruth thought that in all probability this lesion occurring in one gland only was due to accidental inoculation from one of the other experimental sheep rather than to the large dose of culture ingested, which, if it proved infective, would have produced more marked lesions.

No. 4 was inoculated in the thigh with 0·25 c.c. of four-days-old broth subculture. The usual symptoms of lameness were evinced for a few days subsequently, and persisted slightly up till ten weeks later, when the animal was slaughtered. At the post-mortem a small amount of pus with much fibrous tissue was present near the seat of inoculation.

"The popliteal glands were much enlarged, and consisted simply of a thick fibrous envelope, with a firm, greenish, caseous centre. The pelvic lymphatics of the same side contained a pea-sized nodule of greenish pus, with a thick fibrous wall surrounded by apparently normal gland tissue. The other organs were normal."

A goat was also inoculated, and after showing lameness, fever, and constitutional disturbance for two days, gradually recovered, and on being killed two months after inoculation showed well-marked glandular lesions.

A calf six months old was inoculated in the thigh with 1 c.c. liquid serum subculture from a rabbit. On the following day marked pain, swelling, and lameness in the limb resulted; recovery was rapid. The animal was killed in ten weeks for examination, but beyond a small fibrous nodule the size of a bean near the site of inoculation, no lesions were found.

With regard to feeding experiments, in only one instance could the disease be transmitted by the alimentary tract, and this was in a rabbit, where typical pulmonary and pleural lesions were induced by feeding with 0·75 c.c. of a three-days-old broth subculture from a guinea-pig. Gilruth drew attention to the following points:

1. The similarity in the pathogenic action of the bacillus to that of the tubercle bacillus—*i.e.*, the general appearance of the nodules, the progression of the disease from gland to gland, with the ultimate affection of the lung.

2. Experiments showed that, provided death does not rapidly occur from, probably, the formation of a powerful toxin (as in the case of the sheep and of guinea-pigs and rabbits inoculated in the earlier experiments), and provided a sufficient time elapses, "*the lung ultimately becomes affected, even when the farthest extremity of the body is inoculated.*"

3. The distinctly green coloration of the pus in sheep does not appear to be due to any chromogenic action on the part of the bacillus, but rather to the reaction on the part of the ovine system.

MODE OF INFECTION.

This has not been definitely determined. The results of feeding experiments show that infection by ingestion is not probable. According to Edgar, the prevalence of the disease amongst aged ewes and "broken-mouthed" sheep points to a wound infection taking place while grazing, when the teeth are absent or much worn.

According to Carré, the first numerous abscesses that appear in lambs depend on umbilical infection, while in hoggets and adult sheep the infection occurs by way of the intestine.

MORBID ANATOMY.

The chief lesions are found in the lymphatic glands, the lungs, and the pleuræ. The lymphatic glands most commonly involved are the prescapular, the precrural, the inguinal, the popliteal, the bronchial, and the mediastinal, but various other glands may be affected, either primarily or secondarily. Lesions have not been observed in the pharyngeal, parotid, or sublingual lymph glands.

According to Gilruth, "the lesion commences by the arrest of the specific bacillus, generally in a lymph gland, where one or more are surrounded by and included within the phagocytes. The micro-organisms multiply within the cells, and ultimately cause the degeneration and death of the latter. Simultaneously a slow chronic inflammation occurs around the focus of attack; there is proliferation of connective-tissue cells, and the formation of more or less new fibrous tissue. As the process spreads outwards the centre degenerates, and the protecting wall increases in thickness—in fact, all the phenomena of the pathology of true tuberculosis in a gland occur, with the exception of the formation of giant cells. The degenerated centre of the nodule assumes a greenish tint, especially distinct at the time of exposure by the knife, but gradually becoming greyer afterwards. In older nodules, in the centre of the purulent or caseous mass (for the consistency varies from that of cream to that of cheese in different nodules) there are usually no bacilli present which can be demonstrated by the microscope or by cultural methods."

According to Ostertag, the caseous foci do not calcify, but dry up and exhibit an onion-like stratification. But Nocard and Leclainche, and also Sivori, assert that the caseous material may contain calcareous particles.

The pulmonary nodular lesions in these earlier stages of the disease generally occur close to the pleura on the supero-external region of the lung. They may be as small as millet seeds, and show a slightly greenish tint, even through the overlying pleura. (It has not been definitely decided whether the pulmonary and pleural lesions are ever primary, or whether the pleurisy is always secondary.)

"Gradually the pleura becomes involved, a fibrinous exudate results, and, later on, provided practical recovery with a slight permanent adhesion does not result, a quantity of pus or caseous material develops in the pleural cavity, localised always, more or less, by a thick fibrous wall" (Gilruth).

According to Edgar, the lungs may contain one or more of the abscesses or nodules. He has found them always isolated, the intervening portions of the lung being in most cases healthy.

"When situated in the costal pleura, they are less likely to become caseous or to heal, and there is more pus. In recent cases the pleura is acutely inflamed; in older ones it is thickened and attached to the lung by fibrous adhesions. Sometimes the abscess involves the costal muscles, and appears as a small bulging tumour between two ribs. The presence of these abscesses is usually confined to one portion of the body—most commonly the lungs and pleura—but it is by no means rare to find the muscles of the thigh or the lymphatic glands involved at the same time."

In a case recorded by Reid, "the subject, a wether in good condition, showed no symptoms during life. On opening the thorax, the left lung was found to be intimately adherent to the thoracic wall. Two-thirds of the lung tissue were simply converted into a large abscess filled with inspissated pus, the remaining portion of the lung being hepatised and showing the usual pneumonic lesions. The right lung was normal except in the posterior portion of the lower border, which was slightly affected with pneumonia."

Nodules have also been found in other portions of the carcass, such as in the intermuscular tissue, the udder, etc. Gilruth found in one instance an abscess the size of a bean situated in the centre of a "chop." He has no evidence that the disease ever affects to any great extent other organs, such as the spleen, liver, kidneys, and intestines.

In some cases of the disease specimens of *Strongylus rufescens* are observed in the lung in addition to the lesions of pseudo-tuberculosis.

According to Carré, a common feature of the disease in France is the presence of large abscesses, especially in the subcutaneous tissue.

SYMPTOMS.

In the large majority of cases no symptoms are observed. Occasionally in cases of long-standing, cachexia may be present; but the general experience is that the affected animals are in good condition, and the presence of the lesions is only discovered at the slaughter-house. In Reid's case, already referred to, the animal was in good condition, and showed no apparent symptoms during life, although the lesions were very extensive.

As already remarked, the experience of Gilruth was that the disease, when contracted naturally, never proves fatal.

Nörgaard and Mohler found that in cases where visceral metastasis occurred symptoms of chronic broncho-pneumonia or pleurisy, with progressive emaciation and anæmia, were manifested. According to Carré, although the mortality is slight, the development of the lambs is seriously interfered with, and their value may be very considerably reduced.

DIFFERENTIAL DIAGNOSIS.

As no symptoms are present during life, the differential diagnosis from tuberculosis will only be called for at the inspection of the carcass. The diagnosis must be based on a bacteriological examination, the demonstration of the specific bacillus, and on inoculation experiments.

Other points which will assist in the differential diagnosis are—In pseudo-tuberculosis the lesions never calcify, the nodules are more rapidly produced by inoculation, caseation occurs earlier and more quickly than is the case with tuberculous lesions, and no giant or epithelioid cells can be demonstrated in microscopic preparations of the lesions.

TREATMENT.

Obviously, as no symptoms are present, treatment cannot be adopted.

Carré states that prophylactic measures are of importance, and that by applying a suitable dressing to the umbilicus of newly-born lambs a very large reduction in the number of cases occurring has resulted. In France the disease appears to assume a far more serious economic aspect than in other countries, and the losses sustained by sheep-farmers and butchers are, according to Carré, considerable.

MEAT INSPECTION.

According to Ostertag, "the character of the process justifies the complete exclusion from the market of organs which are affected with the alterations in question, and of the meat which is sympathetically affected by the generalisation process."

III. STREPTOBACILLARY PSEUDO-TUBERCULOSIS.

By HENRY GRAY, M.R.C.V.S.

French: Tuberculose zoogléique; Pseudo-tuberculose bacillaire; Pseudo-tuberculose à streptobacille; Tuberculose streptobacillaire.

German: Pseudo-tuberculose.

Definition.—This is a nodular disease resembling true tuberculosis in its clinical characters, but caused by a specific streptobacillus.

History.—This disease was first studied by Malassez and Vignal in 1885, afterwards by Nocard, Eberth, Chantemesse, Charrin and Roger, Dor, Nocard and Masselin, Grancher and Ledoux-Lebard, Preisz, Pfeiffer, Parietti, Lucet, Galavielle and t'Hoen, Mégnin and Mosny, Lignières, Vallée, and others.

Animals Susceptible.—It has been found occurring spontaneously in the hare, rabbit, guinea-pig, fowl, and cat; also in a horse suspected of glanders by Pfeiffer and Parietti, and in an ox suspected of tuberculosis by Nocard and Masselin. It can be transmitted experimentally to these animals and also to the mouse.

Bacteriology.—The specific organism is widely distributed in nature, and only produces disease accidentally. It appears as a short, wide bacillus, measuring 1 to 2 μ in length, and having rounded extremities. This bacillus is often united end to end, forming a chain. In old cultures the short ovoid or rounded bacilli predominate. In the tissues every form is encountered, varying according to the age and seat of the lesions. Under any condition it has a tendency to unite into a mass or zooglea.

It is a facultative anaërobe, but grows preferably in contact with air. It is easily stained with the various simple or compound aniline dyes. It is, however, decolorised by the methods of Gram and of Weigert.

The following classification of the disease for descriptive purposes will here be adopted:

1. Bacillary Pseudo-Tuberculosis in the Hare.
2. Bacillary Pseudo-Tuberculosis in the Rabbit.
3. Bacillary Pseudo-Tuberculosis in the Fowl.
4. Bacillary Pseudo-Tuberculosis in the Cat.

1. BACILLARY PSEUDO-TUBERCULOSIS IN THE HARE.

French : Pseudo-tuberculose du lièvre ; Phtisie bactérienne du lièvre.

This disease is occasionally seen in the hare in its wild state, in such instances usually occurring as an epizoötic, and killing off the majority of the animals attacked.

It is due to a specific streptobacillus identical with that found in the bacillary pseudo-tuberculosis of the rabbit, cat, and fowl.

Symptoms and Lesions.—The animals are generally found dead or in a dying state in plantations and fields, and show extreme emaciation. Innumerable miliary nodules are scattered in the substance of the liver, and caseous foci appear in the lungs. At times the intestine, liver, spleen, kidneys, and lungs, are filled with abscesses, varying from the size of a pea to that of a bean. Miliary nodules are also sometimes found on the pleural and peritoneal surfaces, and accompanied by purulent effusion in the pleural and peritoneal cavities.

Diagnosis.—It should not be mistaken for the parasitic pseudo-tuberculosis due to the *Strongylus commutatis* or to coccidia.

Prevention.—Mégnin suggests that plantations should contain shrubs or herbaceous plants, such as eucalyptus, willow, artemisia, tansy, etc., so that the animals can feed off them, and a depurative effect may possibly be produced.

2. BACILLARY PSEUDO-TUBERCULOSIS IN THE RABBIT.

French : Tuberculose streptobacillaire chez le lapin ; Appendicite spontanée du lapin ; Pseudo-tuberculose des rongeurs.

German : Pseudo-tuberculose des Kaninchens.

This affection attacks rabbits of any age, and causes a very great mortality. It occurs as an enzoötic. Spontaneous outbreaks also occur in the guinea-pig.

Symptoms and Lesions.—The animals waste away, become very weak, and move about with difficulty, or are disinclined to shift for food, of which they sparingly partake. The coat is dull and open, their sides " lift," and they pant on the least exertion. The disease runs a slow course, and the animals die from exhaustion. The carcass is thin, soft, and discoloured. The lungs contain small nodules filled with caseous material. The liver is crammed with whitish circumscribed nodules of various sizes, containing cheesy centres. The kidneys may also contain a few nodules of similar character. The abdominal glands are very much enlarged. The intestines, especially the cæcum and colon, contain nodules. The cæcal appendix is enlarged, and contains nodules involving the whole thickness of the mucous membrane.

Diagnosis.—It should not be confounded with true tuberculosis, which occurs from time to time in rabbits kept in confinement. It cannot very well be mistaken for coccidiosis, or parasitic pseudo-tuberculosis due to the *Strongylus commutatis*.

3. BACILLARY PSEUDO-TUBERCULOSIS IN THE FOWL.

This disease occurs as an enzoötic, and its symptomatology resembles that of true avian tuberculosis. The lungs contain nodules varying in size from that of a rape-seed to that of a pea. These nodules are firm and dense, distinctly circumscribed, and do not appear to be encysted. On section they have almost a homogeneous appearance.

Diagnosis.—It might be mistaken for the caseous nodules found in some cases of fowl cholera and other microbic infections, and also for aspergillosis, true avian tuberculosis, coccidiosis, parasitic cysts, nodular tæniasis, etc.

4. BACILLARY PSEUDO-TUBERCULOSIS IN THE CAT.

This is occasionally seen in the cat, and, in its clinical manifestations, resembles true tuberculosis, parasitic pseudo-tuberculosis, generalised sarcomatosis or carcinomatosis.

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ULCEROUS LYMPHANGITIS IN THE HORSE

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Pseudo-glanders; Pseudo-farcy; Suppurative lymphangitis; Lymphangitis ulcerosa.

French: Lymphangite ulcéreuse du cheval; Lymphangite pseudo-farcineuse.

Definition.—A disease in the horse characterised by an ulceration of the skin and superficial lymphatics, which are also, in their course, the seat of abscesses due to the bacillus of Preisz-Nocard.

History.—This disease was first studied and described by Nocard in 1892,* and again in 1896.† In sixteen horses suspected of farcy, and which did not react to mallein, although pus obtained from the lesions in some of the animals, when injected into the peritoneum of guinea-pigs, produced an orchitis, Nocard found in the pus of the lymphatics a special bacillus which took Gram very well. In the exudate from the vaginal sheath of the inoculated guinea-pigs this special organism was found in a state of purity. Liénaux has also observed this disease in the ox.‡

Epizoötiology.—The disease is commonly observed in various parts of France, and possibly occurs everywhere, but is not recognised. It is doubtful, however, if the suppurative lymphangitis described by Moore § as occurring in India is of the same nature. Schwarzkopf || witnessed a tropical ulcerous disease in American horses during the war in the Philippines, but its precise nature was not determined. It also occurs in the ox in Belgium.

Etiology.—Infection seems to gain an entrance to the system by abrasions or wounds, and as this disease is more prevalent in winter, it is

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* Nocard, "Sur la Valeur Diagnostique de la Malléine," *Bull. de la Société Centrale de Médecine Vétérinaire*, p. 116, 1893; "Sur la Malléine," *ibid.*, 1894, p. 92.

† "Sur une Lymphangite Ulcéreuse Simulant de Farcin Morveux," *Annales de l'Institut Pasteur*, 1896, t. x., p. 609; *Rec. de Méd. Vét.*, 1897, pp. 1 and 99; *Journ. Comp. Path and Therap.*, 1896, vol. ix., p. 346.

‡ *Annales de Médecine Vétérinaire*, Juin, 1904.

§ *The Veterinarian*, 1897, p. 162.

|| *The American Veterinary Review*, 1902, vol. xxvi., p. 111.

very probable that cracked heels and other cutaneous lesions play a very important part in the entrance of the specific bacillus into the subcutaneous areolar tissue, and consequently into the lymph channels. The causal organism is widely spread in Nature, and exists as a common saprophyte. Pure cultures of this bacillus, when injected under the skin of the horse, set up a progressive ulcerous lymphangitis analogous to the natural disease. Identical results are obtained in the horse, ass, and mule by inoculation with the pus.

It may complicate contagious acne, which is due to an identical organism. Contagion plays very little, if any, part in its spread.

According to the researches of Dassonville,* the bacillus secretes a very acid toxin, to which the guinea-pig and rabbit are very sensitive, while the dog and cat are only slightly sensitive; also the symptoms and lesions produced by this toxin are very similar to those produced by the toxin of diphtheria. Again, the antidiphtheritic serum modifies the properties of the toxin of the Preisz-Nocard bacillus, paralysing its action and retarding its effects, sometimes for an indefinite period. Dassonville considers that the toxin of the Preisz-Nocard bacillus exhibits properties which entitle it to be classed with the diphtheria toxin, although it is not identical with the latter, and it is not definitely known whether the two are distinct species or two varieties of one species.

Vallée records a case of ulcerous lymphangitis in which recovery took place in a few days after an injection of antidiphtheritic serum.

It is interesting to note that Gilruth,† in his researches on the bacillus causing caseous lymphadenitis in sheep (which, as already remarked, is the Preisz-Nocard bacillus), observed the similarity of the organism culturally to the diphtheria bacillus, "particularly the yellow appearance on ox serum, the sparse growth on ordinary agar, and the growth on broth."

Symptoms.—The disease clinically somewhat resembles the lymphangitis of farcy by the swellings, ulcers, and corded lymphatics.

It usually starts at the lower parts, especially in the vicinity of the fetlocks, of the hind-limbs, which become swollen, painful, doughy, and hot, and show on the skin small buds, which increase in size and undergo suppuration, and then burst and discharge a thick, whitish, granular pus, which afterwards becomes liquid, oily, yellowish, or bloody. The resulting wounds from the open buds are circumscribed, winding, and deep, with salient and friable borders, and having an angry appearance identical to that of a farcy sore. These sores rapidly heal in a few days under the influence of ordinary antiseptics, leaving a slightly deformed and hairless cicatrix. But as soon as they heal up in one place, others are forming in another part close by, and thus the disease is kept up indefinitely.

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* Dassonville, *Rec. de Méd. Vét.*, December, 1907; *Journ. Comp. Path. and Therap.*, vol. xxi., 1908.

† *Journ. Comp. Path. and Therap.*, vol. xv., 1902.

The lesions are rarely confined to the skin, the lymphatics being nearly always invaded. The internal surface of the limb towards the groin shows a swelling, which is hot, somewhat painful, and sinuous in outline. After a time the cord becomes knotty, the knots undergo suppuration and afterwards burst, and pursue a similar course to the primary buds. In the ox there is no tendency for the lesions to undergo suppuration.

Course.—The disease does not always run the same course; sometimes the lesions are localised, and under prompt and correct treatment recover early. At other times the eruption gradually extends from the fetlock to the groin, udder, under surface of the abdomen, fore-limbs, neck and face, and the animal ultimately succumbs after a few months. Sometimes the limbs remain permanently enlarged. Between these two extremes all degrees of virulence and rapidity of spread are encountered.

Lesions.—The lesions may become generalised when the suppurative process extends from the prepelvic lymphatic trunks to the kidneys. The cortical layer of the kidney is crammed with abscesses, of the volume of a pea to that of a fowl's egg. Their walls are composed of a thin layer of fibrous tissue; the neighbouring parenchyma is not changed. The pus contains the bacillus in abundance. The lungs sometimes contain patches of hæmorrhagic broncho-pneumonia, consecutive to an arterial embolism of microbial origin.*

Differential Diagnosis.—From *glanders* this disease is differentiated as follows: In ulcerous lymphangitis the lymphatic glands do not become indurated, but the buds rapidly heal under the influence of ordinary antiseptic treatment. The contrary is the case with *glanders;* also glanders responds to the mallein test. Bacteriological examination of the pus also distinguishes these two diseases. Strauss's reaction is unreliable, as the pus of both diseases injected into the peritoneal cavity of the guinea-pig produces orchitis.

From *epizoötic lymphangitis* it is easily distinguished by the character of the ulcers and by the bacteriological examination of the pus (*vide* Epizoötic Lymphangitis.

Besides these specific forms, there are others—viz., *the lymphangitis*

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* Nocard et Leclainche, "Les Maladies Microbiennes des Animaux," tome ii., p. 173, 1903.

of strangles, which is due to the *Streptococcus equi*, and associated with other symptoms or lesions of that disease; the *radiating cutaneous lymphangitis*, resulting from harness and other wounds, usually on the back, shoulders, or neck, and rarely undergoing suppuration; and the *ulcerative lymphangitis*, causing somewhat irregular and deep wounds inside the thighs, and associated with cocci (Gray, Hunting, and McFadyean's unpublished observations).

According to Friedberger and Fröhner, another form of *pseudo-glanders* is described by De Haan and Hooskamer, as being due to the mould-fungus *Hyphomycosis destruens equi*, and characterised by a malignant mycosis affecting the skin, the buccal membrane, and nasal mucosa, resulting in the formation of ulcers and subsequent necrosis.

Treatment.—The wounds should be treated antiseptically. The abscesses should be opened early and curetted. The antidiphtheritic serum should be tried in obstinate cases.

CONTAGIOUS ACNE

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Contagious pustular dermatitis; Dermatitis contagiosa pustulosa Canadensis; Canadian variola; Canadian horse-pox; English equine variola; Canadian horse skin disease.

French: Dermite pustuleuse du cheval; Dermite pustuleuse contagieuse.

German: Acne contagiosa; Englischen Pocken.

General Remarks.—This is an epidemic affection of the skin, which we describe here, as the causal agent is the Preisz-Nocard bacillus. It was first observed by Goux in 1841-1842, amongst the horses of a battery in the French army, and again by Weber in Paris (1877). Bass met with cases of it in 1876 at Brussels and Paris amongst horses imported from Canada.

In Great Britain the disease was described by Axe in 1877, who stated that it was introduced by imported Canadian horses. In 1879 it was observed in various parts of Europe. German authorities allege that it was introduced into Germany by imported English horses, and they named the affection "English horse-pox."

Etiology.—The disease spreads from horse to horse either by direct contact or, more frequently, through the medium of infected harness clothing, grooming utensils, etc. The contagium is present in the pus from the skin lesions and in the crusts.

The regions of the body attacked are generally those which come in contact with harness, saddles, etc. According to Cadéac, the skin must be irritated, abraded, or inflamed, before infection can occur. Experimentally, however, it is sufficient to rub a few drops of water containing the specific bacilli upon the normal skin of a horse to produce the typical exanthema (Friedberger and Fröhner).

The bacteriology of the disease was studied by Grawitz and Dieckerhoff in 1885, and they discovered a specific micro-organism in the pustules, pus, and dried crusts, which they termed the "acne bacillus."

According to Nocard and Leclainche, this bacillus is identical with that causing ulcerous lymphangitis in the horse (Nocard's pseudo-glanders, see p. 220); also with that causing caseous lymphadenitis in sheep—viz., the Preisz-Nocard bacillus.

Period of Incubation.—The period of incubation, according to some authors, is from three to five days, while others regard it as varying from six to fourteen days.

Symptoms.—The eruption generally occurs on the back, on the area covered by the saddle, and sometimes on the croup and shoulders. The head, neck, and extremities, except in rare cases, remain free.

A *benign* and a *severe* form are recognised. In the *benign* form the skin of the affected areas is hot and slightly sensitive to palpation; small round or oval prominences appear. These consist of circumscribed inflammatory areas of various sizes, and on their surface vesicles are found, which become pustules. On the small areas the pustules may be from one to three in number, but on the larger areas from ten to twenty may appear. These pustules rupture, and light yellow crusts are formed, which cause agglutination of the hairs in the vicinity. In about seven days the crusts and hair fall off, and circular spots remain. In some cases small ulcers form, and cicatrices are left, deprived of hair and pigment. Pruritus is absent; also no constitutional symptoms are observed. A slight temporary tumefaction of the submaxillary glands may be present.

In the *severe* or grave form of the disease the pustules become confluent; the infection is more virulent, and the affected areas are larger. The affection is aggravated by keeping the animals at work, as the harness produces marked irritation of the affected parts. The skin becomes thickened, large painful nodules are formed, which extend deeply and suppurate in their centres; ulcerating cavities are produced, and suppurative inflammation of the lymphatics results.

The course of the disease is prolonged, and it may continue, with fresh attacks, for five or six weeks, or even two months, permanent cicatrices remaining.

In rare cases the extremities may be involved and the joints may be affected. As a rule, constitutional symptoms are slight and pruritus is absent. The disease is differentiated from equine variola by the absence of eruptions on the hollow of the pastern and on the mucous membrane of the mouth, and by its tendency to invade certain areas of the body; also by the absence of febrile symptoms.

Prophylaxis.—Preventive measures are similar to those advised in the case of equine variola (see p. 582).

Treatment.—The affected animals should be withdrawn from work. The skin lesions may be dressed with a solution of aluminium acetate (7 per cent.). Nocard and Leclainche advise a lotion composed of acetate of lead 2 parts, alum 1 part, and water 50 parts, or solutions of 1 to 2 per cent. sulphate of copper and sulphate of zinc may be employed (Trasbot). Dieckerhoff advises an ointment containing acetate of lead and tannic acid.

In the severe cases the resulting ulcers should be dressed with antiseptic solutions, such as Cyllin (3 per cent.), or perchloride of mercury (1 in 1,000), or solution of peroxide of hydrogen.

TETANUS

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Lockjaw.

French : Tétanos.
German : Wund-starrkrampf.

Definition.—An infective disease, occurring in all animals, but especially in horses, asses, and mules, characterised by tonic spasms of various muscles, with great increase of reflex irritability, causing marked exacerbations of the muscular spasms. It depends on the entrance of tetanus bacilli into a wound; these form toxins, which are carried to the nerve centres, and exert a special action thereon.

GENERAL REMARKS.

Even prior to the discovery of the tetanus bacilli, there were observers who recognised that the disease depended on some form of wound infection. Carle and Rattoni, in 1884, were the first to demonstrate the possibility that tetanus could be transmitted by inoculation from one animal to another. They produced the disease in rabbits by inoculating them with the products of a wound from a case of tetanus in man.

In 1885, Nicolaïer produced the disease in animals by inoculating them with garden mould and street dust. In the pus of the wounds he discovered a bacillus which he believed to be the cause of tetanus. In 1889 pure cultures of the bacillus isolated by Nicolaïer were obtained by Kitasato. The bacillus of Nicolaïer is recognised as the cause of tetanus in all animals, and also in man. This discovery has exerted an important influence on the treatment of the disease, but even more so on the means adopted for its prevention. Although we cannot claim that with a correct knowledge of the pathology of the affection a successful line of treatment for acute cases has followed, we can at any rate assert that, having abandoned the heroic therapeutical measures of former times, and recognising the natural powers of recovery, more successful results are obtained. Preventive measures based on the etiology and pathology of the disease have proved eminently successful in lessening the number of cases, which formerly were of comparatively frequent occurrence, as the result of infection of both accidental and surgical wounds. Tetanus is met with in all countries, but appears to be more frequent in certain districts than in others. The late Professor Williams drew attention to the fact that during his practice in the district of Bradford he met with only two cases of the disease in ten years, and the late Mr. Cartwright, of Whitchurch, never saw a case in his district during a practice of forty-five years.

Most practitioners have observed that a series of cases may occur at indefinite intervals, and also that on certain farms, wounds in horses are very liable to be followed by tetanus. The disease is more common in hot than in temperate climates, and is less frequently met with when wounds are treated under good hygienic surroundings and with antiseptic precautions.

ETIOLOGY.

It has been clearly demonstrated, both on bacteriological and on clinical grounds, that the cause of tetanus is the bacillus of Nicolaïer, which, gaining entrance to a wound, grows and forms toxins that are carried to the nervous centres, and produce the characteristic phenomena of the disease.

Kitt, von Behring, Nocard, and other investigators, have transmitted the disease to horses and other animals by inoculation with the pus taken from wounds of subjects affected with tetanus, also by means of pure cultures of the tetanus bacillus. Moreover, the characteristic bacilli have been discovered in the pus of wounds occurring in animals suffering from tetanus.

BACTERIOLOGY.

The tetanus bacillus is met with in a sporeless form and in a spore-containing condition.

In the sporeless form the rods are 6 to 8 μ in length, and 0·2 to 0·3 μ in thickness; they are straight, and rounded off at the edges, and occur either singly or in groups of two to four. Some authorities give the length of the bacillus as from 4 to 5 μ.

In the spore-containing condition, which is the more mature form, the rods are of the same length as the above, but have at one end a spherical and refrangent spore, which causes them to resemble in shape a pin or a drumstick. The bacilli may form long threads.

Movement.—The rods have a weak automatic power of movement. This ceases when spore-formation occurs.

Mode of Life.—They are obligatory anaërobes — *i.e.*, they grow only when air is excluded, and thrive best in hydrogen.

Growth.—They flourish best at a temperature of 97° to 100·5° F. Growth ceases at a lower temperature than 57° F. At the temperature of the blood spores form in thirty hours, and at 68° to 77° F. spore formation occurs in seven days.

Resistance.—The resistance of the bacilli is very great, especially in the form of spores. The bacilli are killed at a temperature of 160° F. The spores are destroyed by exposure to a temperature of 212° F. for two to five minutes in a steam steriliser. In cultivations the spores are killed by a 1 per cent. solution of nitrate of silver in one minute, by $\frac{1}{10}$ per cent. solution of the same agent in five minutes, by a 5 per cent. solution of Cyllin in five minutes, by a 1 per cent. solution of permanganate of potassium in ten minutes, by a 5 per cent. solution of carbolic acid mixed with $\frac{1}{2}$ per cent. solution of hydrochloric acid in twenty-five minutes, and by a solution of iodine in six minutes. Chlorine, chlorinated lime, and tar, have been found to have a destructive action on the spores.

According to Kitasato, a 5 per cent. solution of carbolic acid has no effect on the spores.

Dried pus, containing tetanus spores, retains its virulence for over sixteen months, according to Kitt. According to Nocard, clams employed for castration, which have become infected, may transmit the disease for eighteen months, even after being immersed for five minutes in boiling water, or in a 5 per cent. solution of carbolic acid.

According to Tizzoni and Cattani, the spores are not affected by concentrated mineral and organic acids. They also prove resistant to the digestive fluids, especially to the gastric juice; and, according to Sormani, they may grow and increase in the intestine.

The spores also resist putrefaction for a long period, and, according to some authorities, may remain virulent in putrefying material for two and a half months.

Staining.—The bacilli stain well with ordinary aniline dyes, also by the method of Gram. For the spore-containing form Ziehl's double staining proves satisfactory.

Cultivation.—The bacilli grow well in slightly alkaline peptone-containing gelatine, agar-agar, and broth. Growth is accelerated by adding 2 per cent. of grape-sugar or 1 per 1,000 of sulphindigotate of soda to the culture medium.

The colonies formed in gelatine plate cultures show a dense opaque centre with fine divergent rays. At the termination of the second week the gelatine commences to liquefy, and a small amount of gas is formed. The cultures give off an empyreumatic odour.

Distribution.—The tetanus bacillus is widely distributed, and may be justly described as ubiquitous. It is found in garden mould, especially when rich in animal manure; also in the fæces of horses, which seem to form a specially suitable nidus for its existence. Hence its presence in stable manure, gardens, on the floors of stables, in street dust, and in stable yards, where it exists as a saprophyte. According to Nicolaïer, it is not found in the soil of forests nor in the deeper layers of garden earth. Marchesi failed to find it in the soil at a depth below 2 metres.

The tetanus bacillus is also found in the fæces of cattle, pigs, dogs, rabbits, cats, guinea-pigs, mice, geese, and ducks, and in the intestinal canal of healthy horses and cattle.

Location of the Bacilli and their Action in the Body.—An important characteristic of the tetanus bacillus is that it does not pass into the blood or general tissues, but remains at the seat of infection—*i.e.*, in the wound at which it gains an entrance. Here the bacilli elaborate toxins, which reach the nervous centres by the blood-stream and along the course of the peripheral nerves. According to Marburg, the toxins may be taken up by the axis-cylinder of the peripheral nerves, and thus conveyed to the nervous centres. Various experiments tend to show that the toxins act especially on the central nervous system and on the spinal cord, and produce the general tetanic spasms.

Characters of the Toxins.—The toxins are formed by the local colony of bacilli in the wound, being elaborated during their growth. They possess actions on the nervous system similar to those of strychnine. A number of these toxins have been discovered—viz., tetanin, tetano-toxin, spasmo-toxin, and a hydrochloric acid toxin. According to recent investigations, the most important toxin is *tetano-toxalbumin.* This, as proved by Knorr, is a most active poison, a dose of ¼ milligramme proving fatal to a horse weighing 1,100 pounds. It produces not only the characteristic spasms of tetanus, but has also a destructive action on the blood-cells. According to Courmont, it occurs in large amounts in tetanised muscles, in the blood and urine of affected animals, and in the milk, according to Brieger. It is rendered ineffective by solutions of permanganate of potassium and other chemical agents, also by the gastric juice and the intestinal epithelium, and by exposure to a temperature of 302° F., according to Fermi, and to one of 149° F., according to Kitasato.

The latter authority found that, by inoculating animals with the toxins remaining in the pus of the infected wound, or with those obtained from virulent cultures, from which the bacilli have been removed, all the symptoms of tetanus were produced. In these cases the symptoms appear as soon as the toxins are absorbed, and there is no period of incubation, such as occurs when the unaltered cultures are employed.

MODES OF INFECTION.

Experiments conducted with a view to transmission of the disease by inoculation with blood, nervous matter, and other tissues, apart from the infected wound of affected animals, have proved negative. According to Kitasato, cultures cannot be obtained from any part of the body with the exception of the pus, secretions, and tissue of the infected wound. This authority, however, by injecting mice with the blood taken from the heart of a fresh cadaver that was the subject of tetanus, produced typical tetanic symptoms and death in from one to three days.

It has also been demonstrated that animals may be fed for long periods on pure cultures of tetanus bacilli without any ill effects.

There is no evidence to show that consumption of the flesh of animals affected with tetanus has proved detrimental to human beings.

Experimentally, it has not yet been demonstrated that infection can take place by way of the intact mucous membrane of the intestine or by the lungs ; thus it is not proved that the disease can be conveyed by ingestion or inhalation under ordinary conditions. According to Thalmann, infection may be conveyed when the respiratory mucous membrane suffers from catarrh. Other authorities believe that the minute perforations in the intestinal mucosa caused by certain parasites may render infection by ingestion possible.

The commonest mode of infection, however, is the entrance of the bacilli into a wound, and it is probable that spores may enter as well as bacilli. The source of the infection is usually earth, street dust, or stable manure, which contain the bacilli and spores ; and when the above agents gain entrance to a wound, the disease is likely to be produced. Experiments have demonstrated that mice, guinea-pigs, and rabbits, inoculated with garden mould develop typical tetanus, especially when the mould is rich in animal manure, such as horse-dung. The entrance of foreign bodies into wounds, such as splinters of wood, contaminated with soil or dust containing the bacilli, is a common source of infection. Cobwebs and the dust of hay, etc., may prove carriers of infection.

The infection in ordinary cases is generally a mixed one—*i.e.*, pyogenic organisms are present in addition to tetanus bacilli, and it is believed that the former favour the growth of the latter and tend to render infection more certain. Although a wound in any region of the body may be followed by tetanus, should it become contaminated by any material containing the bacilli or spores of the disease, we know from clinical experience that in certain regions infection is more likely to occur. Thus, wounds of the feet caused by gathered nails, pricks in shoeing, penetration by particles of glass or by a sharp portion of gravel, etc., furnish a large number of the cases.

Here we may remark that it is often insignificant wounds, receiving no attention, which are most likely to be followed by the disease; also that tetanus may occur after wounds have already healed and cicatrised. Punctured wounds of the extremities, especially those involving tendons or fasciæ, and those in which a foreign body remains, also punctures in the region of the forearm, elbow, and coronet, are liable to be followed by tetanus. Wounds of the feet and extremities are obviously more liable to become infected by material containing the bacilli, such as earth, manure, and dirt from stable floors. In our experience wounds in the region of the head, often of a superficial character, form a large proportion of the cases of tetanus occurring in country districts.

In some instances trivial abrasions of the skin may become infected by tetanus bacilli, such as collar and saddle galls, abrasion arising from the crupper when breaking in young horses, etc. The late Professor Williams met with a case in which the disease occurred after the application of a moderate blister to a fore-leg. Gray observed three cases in which the infection was carried by the same saddle, the bacilli entering through an abrasion of the skin of the withers. Occasionally suppurating sand-cracks, corns recently pared out, also treads, are followed by tetanus. The entrance of foreign bodies into a carious tooth is also regarded as an occasional cause of the disease.

Tetanus, although rare in cattle, may occur in cows after parturition, the mode of infection being, according to Friedberger and Fröhner, injuries to the uterus and vagina during difficult births, and the retention of a decomposed placenta. According to these authorities, the majority of cases of the disease in cattle occur after parturition, and they have also observed it as a sequel to abortion.

Tetanus is not uncommon in newly-born animals, especially foals and lambs, the mode of infection being most probably by the umbilicus.

The disease may occur after surgical operations, particularly castration, docking, and the treatment of umbilical and scrotal herniæ by means of clamps. Since the introduction of antiseptic methods and the observance of surgical cleanliness, this unfortunate complication is now of comparatively rare occurrence. But when operations of this kind are performed by empirics, it is by no means uncommon for tetanus to follow; and this is not surprising when we consider the crude methods that are adopted, and the total disregard of antiseptic precautions.

Occasionally the disease may follow simple operations, such as the insertion of setons, pyropuncture, and even subcutaneous injections. Operation wounds may become infected directly by the entrance of tetanus spores in the form of dust, by earth or dirt containing the bacilli; and indirectly by the hands of the operator, or by the instruments, which may be contaminated with the secretions from wounds of affected animals.

The presence of inert substances in operation wounds, such as silk threads or portions of dressings left behind, is believed to favour the development of tetanus bacilli, and it was proved experimentally that silk threads soaked in a suspension of the bacilli and placed beneath the skin produced the disease far more readily than if the bacilli were injected subcutaneously in small numbers and suspended in fluid.*

The employment of non-sterilised gut in operations has given rise to tetanus. Tetanus is of comparatively frequent occurrence in lambs after castration, and in some districts it may appear enzoötically after this operation. It is also seen in the pig and calf after castration.

Formerly an *idiopathic* variety of tetanus was recognised, under which were included those cases occurring without the presence of a wound. This was also termed the " rheumatic " form. Modern authorities, however, are of opinion that all cases of the disease depend on infection through a wound, although the latter may be so insignificant as to be overlooked, or may be present in regions where it cannot be detected. Thus, small wounds may be present in the mucous membrane of the mouth or that of the nasal cavities, and, as already mentioned, it is possible that lesions of the intestines may form the starting-point of the disease.

There is reason to believe that tetanus spores may occasionally enter a wound and be transported therefrom into the general circulation, and become deposited in various parts of the body, persisting therein for indefinite periods without germinating or multiplying. Such latent subjects may develop tetanus when they experience severe traumatism —*e.g.*, a fracture—without fresh infection. Or, in the absence of any wound, the spores may, from some unknown cause, start to germinate and multiply, and thus produce the disease. It is probable that some at least of the so-called idiopathic cases depend on this mode of latent infection.

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* The Bradshaw Lecture, " On the Treatment of Wounds," by Sir W. Watson Cheyne, December, 1908.

ACCESSORY CAUSES.

Chill is sometimes regarded as an accessory cause. Probably, when the disease follows exposure to cold and wet, the infection is already present in the system, and, the vital resistance of the animal being lowered, the bacilli become active. When the disease occurs in sheep after shearing, it must be attributed to skin wounds occasioned by the process rather than to the effect of chill. High-bred, nervous horses appear to be more susceptible to tetanus than heavy horses of a placid disposition.

When we consider the ubiquitous character of the tetanus bacillus, the frequency of wounds, the marked neglect of antiseptic precautions that prevails, and the lack of cleanliness in the surroundings of horses, it is a matter for surprise that more cases of the disease do not occur. Probably there exists some resistance to the growth of the bacilli and to the action of the toxin which is as yet unexplained.

ANIMALS SUSCEPTIBLE.

The horse appears to be most susceptible to the disease, if we judge this point from the number of cases of the disease met with in practice. Next to the horse come mules and donkeys. The disease is more common in sheep than in the bovine species, while it is rare in the pig, and more so in the dog.

Birds offer marked resistance to experimental inoculation with tetanus bacilli. According to Kitasato, fowls are immune to the disease, but other observers have succeeded in inoculating them, large doses of the bacilli being necessary.

IMMUNITY.

An attack of tetanus does not confer immunity. Although second attacks are very uncommon, still, they have been observed occasionally. Experiments go to prove that perfect immunity is not conferred.

MORBID ANATOMY.

The post-mortem appearances are not constant or characteristic. The wound may be either cicatrised or in a purulent condition. The nerve trunks leading from the wound may show infection of the nerve sheaths, minute hæmorrhages, and swelling and softening of the nerve fasciculi. Various changes are described as occurring in the brain and spinal cord, but the lesions are neither pathognomonic nor constant.

These consist of congestions in various parts, perivascular exudations, and granular changes in the nerve cells. Congestion of the meninges and increase of the cerebro-spinal fluid have occasionally been observed.

The other changes met with in various parts of the body must be regarded as secondary. These include evidences of death from asphyxia; extravasations under the serous and mucous membranes; various pulmonary lesions; epicardial and endocardial hæmorrhages; well-marked *rigor mortis*, appearing early; alterations in the muscles consisting of a dark red colour, with points of ecchymoses, or they may be pale in colour and softened, as if boiled; congestion of the liver, spleen, and kidneys; ecchymoses in the intestinal mucous membrane; distension of the bladder with urine, and congestion with ecchymoses of the mucous membrane of this organ.

PERIOD OF INCUBATION.

This is extremely variable and also difficult to estimate, as it is not possible to ascertain definitely when the bacilli gain entrance to a wound. Again, it is not uncommon for the earliest symptoms of the disease to be overlooked by the attendant, so that the case does not usually come under the notice of the practitioner until well-marked symptoms are present. In the horse the average period of incubation is usually regarded as being from four to fifteen days. Hoffmann records a case in which the disease appeared in six hours after a wound in the neck. Fröhner observed a case in which the period of incubation was forty days. In the latter case, however, an element of doubt exists, as infection may have taken place at a later date than was imagined.

The period of incubation in the horse after inoculation is from four to five days, and in sheep from two to four days, according to Schütz.

In cattle, after parturition, it may vary from five to eight days to two to three weeks.

SYMPTOMS.

Clinically we may recognise *two* types of tetanus.

One may be termed *subacute*, in which the symptoms are developed slowly, the muscular spasms and nervous excitement are not intense, the muscles of mastication are but slightly involved, and the patient is able to feed. In such cases there is usually a longer period of incubation than in the acute form, and, if treatment be adopted sufficiently early, there is a tendency towards recovery. Probably the mild cases depend on a smaller amount and a lessened virulency of the bacilli which gain entrance to a wound, or it is possible that the subjects may possess a certain degree of resistance to the full action of the toxin.

The symptoms presented are—a varying degree of stiffness in the hind-limbs, a quivering action of the tail, slight hardening of the muscles, and protrusion of the membrana nictitans when the head is suddenly raised up. The early symptoms may be so slight as to easily escape observation, and the slight stiffness in the movements of the jaws which is present may also be overlooked, as the horse is able to feed without any apparent difficulty.

In more marked cases the head is extended and the muscles of the neck are hard to the touch, while difficulty in mastication is more apparent. Any excitement or rough handling will cause protrusion of the membrana nictitans and spasms of the muscles. Still, such cases may continue to feed and run a prolonged course to a successful recovery, without any increase in the severity of the symptoms.

The *acute* form of the disease is characterised by a short period of incubation and a rapid development of the symptoms.

In this form there are variations in the development of the symptoms, some cases appear from the histories given to come on suddenly, while in others the earlier symptoms are not so severe, but soon become so. It is, however, difficult to ascertain when the first symptoms appear, as they are frequently overlooked by the attendant, or ascribed to other diseases. The characteristic symptoms are the appearance of tonic spasms of various groups of muscles; dilatation of the nostrils; the protrusion of the membrana nictitans over the eye, especially when the animal is suddenly disturbed or excited; the stiffness of the jaws; marked difficulty in mastication; the animal has a "wooden" appearance; and, in severe cases, the jaws become completely "locked," due to spasm of the muscles of mastication.

Hyperæsthesia and increased reflex irritability are manifested by excitement and marked exacerbations of the muscular spasms on the slightest noise in the vicinity, or on the attendant suddenly approaching the animal.

The muscles of the head and neck may be the first to become affected by the tetanic spasms, the disease then extending backwards; or the spasms may commence in the hind-quarters and extend forwards.

In severe and fatal cases running a rapid course the spasms affect the muscles in every region of the body simultaneously. It is said that the spasms affect first the muscles in the vicinity of the infected wound, but this we have not observed. The groups of muscles affected and the symptoms produced are as follows :

Muscles of Mastication.—Tonic spasm of these induces the condition termed *trismus* or "locked jaw." According to its degree, it varies in the effect produced from a difficulty in prehension and mastication to complete inability to perform these acts, owing to the jaws becoming locked together.

Spasm of the muscles of the head and of the facial region, also of the lingual, pharyngeal, and laryngeal regions, produces the following symptoms: "Pricking" of the ears, the tips of which approach each other; dilatation of the nostrils; retraction of the angle of the mouth; difficult deglutition; accelerated respirations; retraction of the eyes within the orbits; protrusion of the membrana nictitans.

In the condition known as *orthotonos* the extensor muscles of the neck, back, loins, and croup, are affected, so that the neck, back, and loins are rigid and in a horizontal line.

In *opisthotonos* the spasm of the spinal muscles causes a slight depression of the back and loins, the tail is elevated and twitching, the neck is drawn upwards and shows a concavity in its superior border (termed *ewe-neck*), the head is extended and the nose elevated.

Opisthotonos, with trismus, is the condition most frequently met with in cases of tetanus in the horse.

In *pleurothotonos* the trunk is bent in a spasmodic manner, either to the right or to the left side. This is seldom met with.

In *emprosthotonos*—a very rare condition—there is arching of the back.

The *abdominal muscles* are contracted, causing compression of the abdominal region, and giving the animal a "tucked-up" appearance.

Spasm of the respiratory muscles causes difficult respiration. When well marked it may induce stertorous breathing and eventually asphyxia.

The *muscles of the limbs* are spasmodically contracted, the joints can only be flexed with difficulty, and the limbs are extended outwards, supporting the body in a prop-like manner. The animal stands persistently, and if forced to move, he carries the limbs in a stilty manner. Backing and turning round are performed with extreme difficulty.

The hyperæsthesia and increased reflex irritability are prominent symptoms, more marked in high-bred, nervous horses. The slightest sound, or simply approaching or handling the animal, causes a paroxysm of spasms, during which there is a characteristic rolling and retraction of the eyes, and protrusion of the membrana nictitans, which covers the cornea to the extent of one-third or one-half.

Rigors, followed by sweating at intervals, may occur in severe cases, the latter symptom being well marked in the later stages, when the animal assumes the recumbent position and struggles violently.

The *temperature* is not elevated in ordinary cases, but in fatal cases, shortly before death, it may rise to a marked extent, even reaching 109° to 110° F. Even for some time after death it may rise still higher, and cases are recorded in which it reaches 113° F.

The *pulse* is not altered to any extent in the early stages, but during a paroxysm it becomes more frequent. It may also become hard and small in character, and in some cases it is found soft and compressible.

The *respirations* are accelerated in proportion to the severity of the case, the violence of the spasms, and the implication of the respiratory and abdominal muscles. The marked acceleration of the respirations is out of all proportion to the frequency of the pulse. The respirations are shallow in character, due to spasm of the respiratory muscles, producing a fixed condition of the ribs, and also to implication of the abdominal muscles, which compress the abdomen and restrict the movements of the diaphragm. Pulmonary and bronchial complications may ensue towards the later stages.

The *appetite* is not impaired; the animal makes efforts to feed, but prehension and mastication are interfered with, and are rendered impossible when trismus is complete. Spasm of the pharyngeal muscles interferes with deglutition, and in severe cases the swallowing even of fluids may be impossible, so that regurgitation takes place. Salivation is profuse; the saliva, mixed with mucus and with portions of semi-masticated food, accumulates in the mouth, and has a fœtid odour; it also hangs in stringy masses from the lips. Foals are unable to suck when trismus is complete.

The peristaltic action of the intestines is impaired, and this, in conjunction with the spasm of the abdominal muscles, interferes with defæcation. Owing to spasm of the sphincter vesicæ, complete retention of urine and dilatation of the bladder may be present. Micturition is also interfered with, in consequence of the difficulty of assuming the normal position for the act.

The urine may be of high specific gravity and colour, and albuminous. Occasionally priapism may be observed in the male. Consciousness is not affected until the animal assumes the recumbent position, from exhaustion, when he struggles violently in a convulsive manner. Death occurs from exhaustion, and also from excessive spasm of the respiratory muscles inducing asphyxia.

COURSE.

This depends on the rapidity with which the disease develops, and on the severity of the symptoms. With a short period of incubation, an early generalisation of the spasms and well-marked trismus, extreme nervous excitability, dyspnœa, sweating, and high temperature, a case may prove fatal in less than twenty-four hours.

In less urgent cases death may not occur before a week. We also meet with cases which live for two weeks or more, and then succumb to a complication, such as pneumonia. In the subacute form of the disease the course varies; the symptoms usually last for four to six weeks, the muscular spasms gradually abating and the muscles becoming relaxed. Œdema of the under surface of the abdomen, and of the sheath and limbs, is observed in some cases. Convalescence may take from five to six weeks, and even after this period some stiffness in the action of the hind-limbs may be observed. In some favourable cases the patient may keep up his condition and even become fatter than prior to the attack.

PROGNOSIS.

In the severe cases mentioned above recovery seldom takes place. Even in subacute cases prognosis must always be guarded, as without any warning acute symptoms may develop and a fatal result ensue.

If the patient feeds moderately well, if trismus is not well marked, and other symptoms not severe, and if, in addition, we are able to secure proper surroundings and careful nursing, we may look forward to recovery, especially when no unfavourable symptoms develop, and the case lasts for fourteen or fifteen days. Inability to swallow is usually a fatal symptom, and when the patient goes down in the stall and violent convulsions occur, death is certain to follow. Cases occur, however, in which the outlook appears most unfavourable and still recovery takes place—hence we must exercise due care in giving a definite opinion as to the probable result. In our experience, mules and jennets prove favourable subjects for tetanus, and we have seen many recoveries in these animals, even when the surroundings and attention were anything but favourable.

Symptoms of Tetanus in Cattle.—The symptoms resemble those described as occurring in the horse, but are not so severe, and evidences of reflex irritability are not so well marked. In mild cases rumination is performed with a certain amount of difficulty, while in acute cases, with trismus it is suspended; tympany of the rumen is present; prehension, mastication, and deglutition are impossible; dyspnœa, obstinate constipation, and arrest of micturition occur, and in a variable period the animal falls down and dies in violent convulsions, sometimes bellowing loudly. The tongue loses its mobility, becomes hard, is covered with thick, tenacious mucus, and firmly compressed against the palate. Owing to contraction of the oblique abdominal muscles, the flanks assume a flattened appearance, descending in a vertical manner from the lumbar transverse processes.

In cases occurring after parturition evidences of metritis may be present. The course of the acute form of the disease in cattle is generally slower than in the horse. In the subacute cases that recover evidences of improvement may appear in from two to three weeks. Very acute cases are sometimes met with which prove fatal in a few days. The mortality is estimated by authorities to be from 70 to 80 per cent.

Symptoms in Sheep.—In the sheep the disease occurs in the acute form. Opisthotonos with trismus is usually present, and there is marked elevation of the nose, also of the tail. As the disease advances, the animal assumes the recumbent posture and lies on the side, with the limbs extended, straight and rigid, and the head drawn in a backward direction. Death may occur in from two to three days, or the disease may last for a week in less acute forms.

The mortality is very high, being, according to authorities, nearly 100 per cent.

The symptoms in the goat are similar to those met with in the sheep.

Tetanus in lambs (tetanus neonatorum) usually occurs in about forty-eight hours after birth. It is characterised by a very high temperature and difficult deglutition. Death is usually rapid, occurring from either dyspnœa or starvation and exhaustion.

Symptoms in the Dog.—The disease is very rarely met with in this animal.

Wooldridge* records a case in a fox-terrier arising from an injury to the right temporal region. The symptoms observed were inability to stand; emaciation; marked hyperæsthesia; tetanic spasms, chiefly affecting the muscles of the back and right side of the neck; the head drawn up and to the right side; dilatation of the pupils, more marked in the right than in the left; trismus and difficulty in deglutition were absent.

Amongst the symptoms described by other observers we may mention tenesmus, abduction of the limbs, protrusion of the membrana nictitans, clenching of the jaws, wrinkling of the skin of the forehead, stiffness of the ears—which may be drawn towards each other or laid back—marked reflex excitability, inability to bark. The course may be rapid, or the case may last for six to ten days.

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* *Veterinary Journal,* January, 1908.

Symptoms in the Pig.—The early symptoms are usually those of trismus, the spasms spreading rapidly over the body. The animal lies on its side, with extension of the head and a rigid condition of the limbs. Champing of the jaws and frothing of the mouth have been observed. Death usually occurs early, and the mortality is very high.

DIFFERENTIAL DIAGNOSIS.

Although cases of tetanus in which the symptoms are developed offer no difficulties in diagnosis, yet in the very early stages careful observation is necessary in order to detect the presence of the disease.

The symptoms may appear at first in the hind-quarters, the head and neck not becoming involved to any extent until a later stage. In such instances the history of the case must be considered, and, on suddenly elevating the animal's head, protrusion of the membrana nictitans is usually observed. As a rule, however, cases are not often brought for treatment until the symptoms are fairly well developed—*i.e.*, when they are observed by the owner, who generally attributes them to other diseases.

Amongst the conditions and affections which might be mistaken for tetanus and require differentiation, we may mention the following:

Strychnine-Poisoning.—In this condition, which rarely occurs in the horse, the symptoms are rapidly developed, the convulsions are clonic in character, being marked by intervals of complete relaxation; while in tetanus the spasms are tonic in character, and, although modified in intensity, they are never intermittent.

Again, tetanus is very rare in the dog, while strychnine-poisoning is of frequent occurrence in this animal.

Rheumatism.—Affecting the neck; also known as *torticollis.* In this affection trismus and protrusion of the membrana nictitans are absent; also there is an absence of the characteristic spasms of tetanus, the marked hyperæsthesia and the nervous excitement.

Meningitis.—In acute meningitis paroxysms occur during which there may be a certain amount of stiffness of the jaws, and protrusion of the membrana nictitans at intervals. The convulsions are clonic in character; the patient goes down early in the disease, and consciousness is disturbed.

Generally speaking, there is a similarity between the symptoms displayed by a case of tetanus in the later stages, with the animal in the recumbent position, and a case of acute meningitis, as in both violent fits of convulsions occur. The history of the case will assist in the diagnosis.

Rabies.—This is distinguished by the history of the case, the temporary relaxation of the muscles between the violent paroxysms, the clonic character of the spasms, the alterations in the disposition of the animal, such as a tendency to attack and bite.

Laminitis.—In the early stages of very acute cases of this disease a slight similarity to tetanus in the symptoms presented might be imagined by those who lack clinical experience. Attention to the position assumed by the patient, to the marked heat and tenderness present in the feet, the inability of the animal to bear weight on one fore-foot when the other is lifted up, and the absence of stiffness in the neck and jaws and of protrusion of the membrana nictitans, should enable a differential diagnosis to be arrived at with facility.

PROPHYLAXIS.

Tetanus being such a fatal disease, we should adopt every available means to prevent it. Although admitting that it may occur in spite of all precautions, we are fully justified in stating that the use of antiseptics in surgical work has diminished the number of cases to a marked extent; the careful cleansing of wounds, and the removal of foreign bodies therefrom, are important essentials, as well as thorough irrigation with a reliable antiseptic solution when demanded by the circumstances of the case. More especially are these precautions necessary when wounds have become contaminated with earth or with excreta, or when they occur in the feet or limbs, being thus more liable to infection.

In districts where many tetanus cases have been known to occur, practitioners have found that the application of pure carbolic acid to wounds of the feet infected with earth, excreta, etc., has proved successful as a preventive measure.

It must be remembered that spores may enter the wound as well as bacilli, and, recognising the marked resistance of the former to antiseptic solutions, we think the above treatment is justified.

Cleanliness of the surroundings and of the floors is also of importance, and the liberal use of disinfectants after thorough cleansing is advisable. The litter from stalls in which a case of tetanus has been treated should not be used for healthy horses.

Practitioners or attendants who have occasion to dress a wound in a case of tetanus should on no account handle wounds in healthy horses. Thorough sterilisation of instruments by boiling, also of utensils, and the use of antiseptics, are also measures likely to prevent infection in connection with surgical operations. Carelessness, such as placing instruments or surgical dressings on the ground while dressing wounds or operating, will account for some of the cases which occur as a sequel to operations. The careful sterilisation of ligatures and suture material is also necessary. As far as possible, wounds should be protected by surgical dressings, so as to avoid contamination by dust, earth, excreta, etc.

No doubt it is difficult, especially in country practice, to have such details properly carried out; still, we should, as far as possible, endeavour to instil into the minds of attendants the importance of at least ordinary cleanliness in the handling of wounds.

The value of antitetanic serum as a preventive is now largely recognised. In " tetanus " districts, also in the case of punctured wounds—especially in nervous, highly-bred horses—and in operations, such as for herniæ, many practitioners have found the use of the serum of marked value as a preventive.* One ounce of the serum should be given by hypodermic injection at the first attendance on the case, or, in surgical cases, prior to operation. As immunisation, which is not active, only passive, is believed to last for three weeks, a second injection should be made two weeks after the first. In the case of badly contaminated wounds, it may be advisable to administer a third, or even a fourth, injection. It can only neutralise the poison when in the blood-stream; it is probably useless when the toxin has entered into combination with the nerve cells. For the prevention of tetanus in newly-born animals an antiseptic dressing should be applied to the umbilical cord. A mixture of iodine and wood-tar proves effectual and economical for this purpose.

TREATMENT.

Subacute cases of tetanus tend to recover without the employment of any medicinal treatment. All that is necessary is early cessation from work, soft, sloppy, easily-digested diet, and quiet surroundings. The animal should be removed from other horses, and placed in a loose-box, where he will not be subjected to noises or rough handling. Salines, such as sulphate of magnesia and nitrate of potassium, if readily taken in the food, are indicated in order to keep the excretory organs in a healthy condition, as the animal cannot be exercised. If, however, the appetite is fastidious, it will be advisable to dispense with the salines and to depend on suitable dieting.

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* Labat gives statistics of 703 cases of wounds, accidental and surgical, in which antitetanic serum was employed as a preventive, and in no instance did tetanus occur. Nocard's statistics show 3,088 cases (of which 2,708 were horses) of operation and accidental wounds, treated with antitetanic serum; 2,500 received the serum immediately after operations (such as castration, docking, and umbilical herniotomy), while 600 cases of punctures of the foot (from nails, etc.), treads, bites, etc., were injected in one, two, four, or more days after the injury. Only one case contracted tetanus, and in this instance (due to a prick in shoeing) the serum was not given until five days after the infliction of the injury, the attack was a mild one, and recovery was complete in twelve days. During the period of the above statistics, 314 cases of tetanus (of which 220 were in horses) were recorded by veterinary surgeons, and in these preventive treatment had not been adopted.

It is extremely doubtful whether drugs have any effect on the course of the disease. Judging from our own experience of cases treated with and without drugs, we believe that attention to hygiene and dietetics is all that is necessary.

Acute cases, in which all the symptoms develop *rapidly*, and trismus is present, generally prove fatal, and here also drugs prove of little value. If, however, the animal is able to feed, or even to drink fluid nourishment, there may be a chance of recovery.

Every variety of treatment has been adopted in cases of tetanus, including the most heroic measures and the administration of all kinds of nerve sedatives. But in spite of all efforts at treatment, the mortality is very high in acute cases. Many practitioners advise the administration of a full cathartic dose of aloes and calomel in bolus as the first step in treatment, and claim a fair proportion of successful results, if the physic can be given and if it acts promptly. Unfortunately, trismus may be so advanced that the bolus cannot be administered, and also the peristaltic action of the intestines may be so impaired that the medicine will not act, and only produces nausea.

Some advise the administration of calomel in the food until purgation ensues, but in our experience large doses of this agent may be given in this manner without causing catharsis. Eserine and arecoline are too violent in their actions to prove of service, as they increase the paroxysms, and tend to do more harm than good.

With reference to nerve sedatives, successes have been claimed from time to time from the employment of special agents, but further experience in their use has shown that their value was imaginary. Belladonna administered in the form of electuary is a favourite agent with many practitioners, who believe that it tends to lessen the muscular spasms, and to exert a favourable influence on the nervous excitement. Indian hemp, chloral, morphine, hydrocyanic acid, bromide of potassium, etc., have been advised by various practitioners.

Whatever agent is selected, it must be administered either in the food, or, if refused in this manner, it may be given in the form of electuary, hypodermic injection, or by injection *per rectum*, as drenches not only increase the spasms, but also there is the danger of the fluid entering the trachea and bronchi. We believe that when cases of acute tetanus recover, this fortunate result is to be attributed to the constitution and powers of resistance of the patient, and to careful dieting, nursing, and proper surroundings, rather than to the medicinal agents employed.

Attention to the wound is believed to be necessary in both the acute and subacute forms. If the case be seen sufficiently early, excision of the tissues in the vicinity of the wound is advised. By this procedure the formation of fresh supplies of toxin is said to be prevented by the removal of bacilli and spores from the part. If, however, a full amount of the toxin has already reached the nerve centres, causing the development of acute symptoms, then, indeed, local treatment is hardly likely to prove of benefit. In order to excise the tissues in the vicinity of the wound, without exciting the patient or increasing the spasms by the pain induced by the procedure, a local anæsthetic should be employed, such as the injection of a solution of eucaine and adrenalin around the part to be excised. The resulting wound should be dressed thoroughly with a strong solution of carbolic acid and covered with iodoform gauze. Unfortunately, in acute cases arising from wounds in the hind-feet, it is sometimes very difficult to carry out excision of the infected part, as it may be impossible to raise the leg off the ground. When this is possible, the operation may be rendered painless by injecting the local anæsthetic over the plantar nerves on each side above the fetlock. When tetanus occurs as a sequel to docking, and the case is seen sufficiently early, a fresh portion of the dock may be amputated, and the hæmorrhage checked by applying strong compound tincture of benzoin, tow, and a bandage. The actual cautery is not advisable, as it would increase the spasms and excitement.

We may state, however, that we have seen a large number of subacute cases of the disease recover without the adoption of any local treatment beyond ordinary surgical attention, while the wound is unhealed, and that, provided a case is progressing favourably and the wound cicatrised, we should hesitate before running the risk of exciting the animal by adopting a procedure the effects of which are problematical.

Antitetanic Serum.—This serum was introduced as a preventive and not as a curative agent, but it has been extensively employed for the latter purpose. Many clinicians, however, have found that it exerted no beneficial action on the course or result of the disease.

Numerous cases are recorded in which recoveries took place even when acute symptoms were present. But bearing in mind the fact that other agents are often employed in conjunction with the serum, and also that recoveries have occurred in the absence of the serum treatment, we are justified in being sceptical as to its real value. In the case of valuable horses, however, it is advisable to employ the serum, so as to give the animals every possible chance. Possibly it may neutralise the toxins that are being formed, also those present in the blood.

From experimental evidence and from a knowledge of the pathology of the disease it is quite apparent that, if the serum treatment is to prove of value, it should be employed in the early stages. The serum is usually injected in doses of 1 ounce once daily in the region of the neck, and is continued for seven to ten days, according to the progress of the case. The process should be carried out carefully and in a quiet manner, so as not to excite the animal, and rigid attention to aseptic details is necessary. The intracerebral method has been tried, but with doubtful results. Of late years American practitioners have given far larger doses of the serum than those usually employed, and claim more success by so doing, and from 4 to 6 ounces have been given daily.

The *antiseptic method* of treatment has been suggested in consequence of the results of experiments with iodine and iodide of potassium. Preparations of iodine, when injected into animals at the same time as cultures of tetanus bacilli, were found to have an antitoxic action. But to prove of value they should be used in the very early stages. Recoveries have been recorded after the employment of Lugol's solution by hypodermic injection. A combination of iodide of potassium and carbolic acid has also been advised, also solution of carbolic acid alone, but with indifferent success. The real value of these agents in tetanus has yet to be demonstrated.

Captain Dawson, A.V.C., reported two cases of tetanus in the horse treated by subcutaneous injections of a saturated solution of magnesium sulphate. One case, that originated from the operation of docking twelve days previously, appeared to be rather hopeless, as the jaws were firmly fixed and all symptoms pointed to an unfavourable prognosis. The drug was administered in doses of 40 c.c., of which 20 c.c. were injected on either side behind the shoulder. This was repeated in three hours, and also twice daily, until the twelfth day, when the dose was reduced to 20 c.c. once daily, and continued to the eighteenth day. Three doses of antitetanic serum were administered in addition to the above—viz., one on the first day, one on the third day, and one on the sixth day. After the third day the symptoms were greatly modified, and on the twelfth day the horse was able to eat grass off the ground. The animal was convalescent on the twenty-second day.

In the other case the symptoms were less severe. The doses given were 40 c.c. for five days, and 20 c.c. daily until the eighth day. The animal was convalescent on the eleventh day. In this case no antitetanic serum was given. The effect of the drug seems to be the reduction or relaxation of the tonic spasm of the muscles.*

This method is certainly deserving of further trial; it has one great advantage as compared with the employment of antitetanic serum—viz., its cheapness, as the serum proves very expensive, and so far as our experience goes, it is of little value in severe or advanced cases of the disease.

The employment of slings is a question on which opinion is divided. If the patient takes to them quietly, they are of advantage, as they obviate the danger of the animal going down from exhaustion, and struggling violently. But there are many cases in which slings produce such a degree of excitement that their employment cannot be advised. In the mild or subacute cases we have never seen the necessity for using them.

As a rule, when a case of tetanus assumes the recumbent position, violent paroxysms and convulsions occur, the animal struggling until death closes the scene. Treatment is useless in such instances, and the most humane course is to have the animal destroyed.

Finally, we may remark that the conclusion we have arrived at, after a fairly large experience in the treatment of tetanus, is that quiet surroundings and proper dieting constitute the essential therapeutical measures.

The food should consist of bran and linseed mashes, oatmeal gruel, milk, also chopped grass if it can be obtained, and provided it can be masticated. Large feeds should be avoided, as they are liable to produce derangement of the digestive organs. The feeding utensils should be kept thoroughly clean, and the food placed in such a position that the animal can partake of it without raising or dropping his head. In the treatment of tetanus it is very difficult to obtain suitable surroundings and to find attendants who are capable of carrying out our instructions with reference to quietness and careful nursing of the patient. Unfortunately, there is a prevalent idea that medicines must be administered and heroic measures carried out in order to give the patient a chance of recovery. The popular views of the nature of this disease are so grotesque that it is not surprising to find amateur treatment to be irrational and injurious in the extreme. As a rule, the owners of animals fail to recognise the nature of the disease until actual trismus is present, and the case is treated for various ailments.

The treatment of tetanus in other animals is to be conducted on similar lines to those advised for the horse.

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* *Journal of Comparative Pathology and Therapeutics*, March, 1911

TETANY.

This is a condition in which the muscles of the extremities especially are affected with tonic spasms, which may be either paroxysmal or continued. The muscles of the face and neck may also be involved, and trismus may be present. Judging from the scanty details of this affection given in veterinary literature, it would appear to be very rare in animals.

In the human subject it occurs under a variety of conditions, which include gastric and intestinal disorders, the acute infectious fevers, during pregnancy, and after removal of the thyroid gland and parathyroid bodies.

We have only met with one case of this affection in animals, which occurred in a pony mare that had recently foaled. The usual symptoms of tetanus were present, including a limited degree of trismus and protrusion of the membrana nictitans. An erroneous diagnosis of tetanus was given, but the animal recovered perfectly in two days. The following year, while again rearing a foal, exactly similar symptoms developed, and recovery took place just as quickly. The treatment adopted saw the administration of belladonna in the form of electuary.

NOTE BY EDITOR.

Cases of tetanus are recorded by human surgeons in which the disease followed operations where infection from direct sources could be excluded. Sir David Semple* pointed out that in the case of tetanus following operations an anaërobic area has been left in the wound, into which tetanus spores migrate from other regions of the body, as these spores are frequently present in the normal intestine. He also showed that "when tetanus spores are injected into a given area of a guinea-pig, and when quinine is injected into a distant area of the same animal, tetanus bacilli are to be found in the anaërobic slough produced by the quinine and nowhere else, and that a control guinea-pig which has similarly received an equal number of spores, but has *not* received any quinine, is not affected by tetanus." Colonel Smith, I.M.S.,† suggests that the spores reach the anaërobic area through the blood-current and develop into tetanus bacilli. It is interesting to note that tetanus in man has resulted from the subcutaneous administration of quinine.

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* *Lancet*, June 10, 1911.

† *Ibid.*, March 9, 1912.

ERYSIPELAS

BY E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—"St. Anthony's fire."

French : Érysipèle.
German : Rot-laufartig ; Rose ; Erysipelas.

Definition.—A specific acute contagious disease, characterised by a spreading inflammation of the skin and subcutaneous tissue, due to the entrance and development of the *Streptococcus erysipelatis vel pyogenes* in the smaller lymphatics of these structures. Toxins are formed locally, which, being absorbed, are to a large extent the cause of the marked constitutional disturbance and the fever which is present.

GENERAL REMARKS.

Erysipelas is an affection of comparatively frequent occurrence in veterinary practice. The majority of cases are met with in horses. Formerly a *traumatic* and an *idiopathic* variety of the disease were recognised, but in the present day we believe that all cases are due to the entrance of the specific micro-organism by way of a wound or abrasion of the skin, although the latter may be so slight that it is easily overlooked. As the streptococcus does not, as a rule, exist in the blood, the theory of local infection is probably the correct one.

Three forms of the disease are usually described :

1. *The Cutaneous,* in which there is a diffuse inflammation of the skin alone, the subcutaneous tissue not being involved, or only to a very slight extent. This form is rare in veterinary practice.

2. *The Cellular,* in which the subcutaneous lymphatic spaces are primarily involved. This is also of rare occurrence in animals.

3. *The Cellulo-cutaneous.*—In this both the skin and subcutaneous tissue are involved. It is also termed "phlegmonous," and is the form most commonly met with in the horse.

BACTERIOLOGY.

The *S. erysipelatis* is the specific micro-organism of erysipelas. It appears to be identical with the ordinary *S. pyogenes*, which occurs in spreading suppuration, being indistinguishable microscopically from it, and very similar in its growth. Also, inoculation experiments indicate that they are closely allied species. The different effects produced are believed to be due to the method of inoculation and to the virulence of the particular micro-organism. The frequency with which suppuration occurs in some cases also shows the close relations between the two micro-organisms. At the same time, there is a marked difference between the effects of the micro-organisms in the body.

The streptococci of erysipelas are found chiefly in the lymph spaces, and in greatest abundance in the zone of the spreading inflammation. They are also present in the lymph vessels of the uninvolved tissue, beyond the inflamed area, in which, according to investigators, an active phagocytosis occurs.

When the streptococci gain an entrance, they develop in the superficial lymph channels, and produce toxins, which become absorbed. The resulting fever and constitutional disturbance are in a great measure due to their action on the system.

The streptococci are widely distributed, and may become attached to litter, the walls of buildings in which affected animals are housed, and may also be carried by the hands or clothes of attendants, and thus infect wounds or abrasions. The vitality of the micro-organisms is not great, and it has been demonstrated that the local reaction they set up is sufficient to destroy them, even while they are spreading peripherally. They do not appear to be directly capable of infecting at any great distance from the affected animal.

Immersion for fifteen seconds in a 1 in 30 solution of carbolic acid destroys them.

ETIOLOGY.

As already mentioned, the entrance of the specific micro-organism into a wound or abrasion of the skin is the essential cause of the disease. An unprotected septic wound seems to be more liable to become infected. In very many cases no wound can be detected, but it is probable that such may be so minute as to escape observation. A cracked heel forms a common mode of entrance for the infection.

As predisposing causes, debility of the system and bad hygienic surroundings may be mentioned. But it is not uncommon to meet with cases in hunters in a hard-working condition and with good hygienic surroundings. Some horses appear to be specially susceptible to the infection. Infection may also occur from operation wounds. In our experience, wounds in the region of the head, orbit, etc., are specially liable to be followed by erysipelas, unless careful antiseptic precautions are carried out in their surgical treatment.

MORBID ANATOMY.

The lesions vary according to the severity of the case. General signs common to all septic cases may be present. Microscopic examination of a section of the skin from the affected part shows the presence of colonies of cocci, arranged in the form of chains, invading the lymphatics in the vicinity of the spreading margin. In the parts which have been recently attacked an excess of leucocytes is present. The skin over the affected part may be covered with vesicles containing dark-coloured fluid, and the subcutaneous connective tissue is infiltrated to a marked extent. Circumscribed patches of the skin lose their vitality, and are removed by sloughing, the result being the formation of unhealthy sores with rough edges. These extend deeply, and discharge a fœtid sanious fluid, which contains shreds of lymph and connective tissue.

Suppuration usually occurs, which may be accompanied by extensive sloughing of the tissues and exposure of tendons, ligaments, and even joints. The pus is thin and sanious in character, and infiltrated in the areolar tissues. There may be general evidences of pyæmic infection, partly depending on periphlebitis.

Amongst other lesions which may be met with are infarcts in the lungs, spleen, and kidneys, malignant endocarditis, septic pericarditis, and pleuritis. Occasionally the meninges may be involved; evidences of acute nephritis may also be present. Some cases in which brain symptoms have been prominent may not show lesions of the meninges on post-mortem examination.

PERIOD OF INCUBATION.

This is variable. The average may be stated as being from three to seven days.

SYMPTOMS.

If the case be seen in the early stages, rigors may be observed. The constitutional symptoms—viz., fever, pain, accelerated respirations, interference with appetite, frequency of the pulse, etc.—vary according to the severity of the case. The usual regions affected in the horse are the forearm, the metacarpal or metatarsal region, the pastern and coronet of either fore or hind limb, the hock, and the tibial region. As a rule but one of these regions is affected. The swelling appears rather suddenly, and at first resembles a mild attack of lymphangitis. But the pain and constitutional symptoms exhibited are out of all proportion to the extent of the swelling present. The effusion into the subcutaneous connective tissue is at first serous, so that the affected part pits on pressure, leaving an indentation from the fingers. This is, however, soon effaced when the pressure is removed.

Later on the effusion is increased to a marked extent, extreme tension of the skin appears, the part assumes a firm, resistant, brawny character, and is acutely painful to the touch. The animal is excessively lame in the affected limb, and may not be able to place any weight thereon. The pain may be so extreme that respirations are accelerated to a marked extent, and sweating may occur. The animal moves with great difficulty, and assumes the standing posture. The temperature rises to 104° or 105° F. The superficial veins of the skin of the affected part are dilated, and a serous exudation may be present. Vesicles are found in some cases, and when the affection is severe their contents may be of a sanguineous character.

In cases where the skin is non-pigmented, redness may be observed. In high-conditioned horses the fever is of the sthenic type, but in debilitated animals the opposite is observed. When suppuration occurs, areas of the skin lose their vitality, and are removed by sloughing, and there is a discharge of watery sanguineous pus containing shreds of lymph and the débris of broken-down tissues.

In very severe cases sloughing of a large area of skin occurs, also of the subcutaneous tissues, exposing muscles, tendons, and deep-seated structures. Suppurative arthritis may occur from the diseased processes extending to joints. The mucous membrane of the mouth and nostrils may become involved in some cases—at first petechiæ, resembling those found in purpura hæmorrhagica; later on erosions or ulcerations may appear. These lesions are, however, by no means constant, and may be absent in a large number of cases.

Wounds of the head may be followed by a very severe type of erysipelas. The disease may extend into the orbital cavities, and down the facial and pharyngeal regions. Extensive swelling of the eyelids occurs, and the appearance of the head resembles that seen in cases of purpura. We have observed meningitis to follow such cases, due to the extension of the infection to the meninges, the explanation being that the dura mater is continuous with the orbital periosteum through the foramina by which the nerves and bloodvessels enter the cranial cavity, or the infection may be conveyed by the alveolar vein.

A characteristic of erysipelas is its extension by a broad, sharply-defined, slightly raised and infiltrated margin of skin. This is often well marked in erysipelas affecting a fore or hind limb. We have met with some cases in which constitutional symptoms were not well marked, although the local manifestations of the disease were extensive.

A wound infected by erysipelas shows an unhealthy surface of a yellowish colour, and an amber-coloured discharge may be observed. No evidences of healing are present.

The *cutaneous* or *mild* form of erysipelas is seldom met with in the horse.

COURSE.

The course of erysipelas varies according to the severity of the case. In favourable cases suppuration does not occur, and the subcutaneous effusion becomes absorbed slowly, the swollen condition of the part being gradually reduced. Experience teaches us that irrational treatment exerts an important influence on the course of the disease. Thus the constant application of hot fomentations is very apt to induce suppuration and sloughing of the skin.

In some cases small circumscribed abscesses occur in the part, which discharge a purulent material, and result in the formation of unhealthy sores, which have little tendency to heal. When extensive sloughing of the skin occurs with marked destruction of the underlying tissues, we observe high fever, great constitutional disturbance, extreme pain, weak pulse, loss of appetite, debility, and loss of condition, and death may occur from exhaustion or from complications.

As already mentioned, brain symptoms may present themselves when erysipelas affects the region of the head. This is invariably a fatal complication.

Some practitioners find that relapses are of frequent occurrence in this affection, but this is not our experience.

PROGNOSIS.

This must be guarded in all cases. Even in instances where the symptoms are suggestive of a mild type of the disease at the outset, the cases may assume a serious character later on.

DIFFERENTIAL DIAGNOSIS.

The characteristic evidences of erysipelas are the spreading character of the inflammation and its mode of extension by a broad, sharply-defined, slightly raised margin of skin; also the acute pain and constitutional disturbance, and the excessive degree of lameness when a limb is involved, all of which are out of proportion to the apparent condition of the affected part.

From Purpura Hæmorrhagica it is distinguished by the above characters, and also by the history of the case. In purpura the swellings are distributed in an irregular manner over the body, and the head is usually affected early. Moreover, the swellings in purpura are not acutely painful, as in erysipelas.

From Lymphangitis.—In this affection, which generally affects a hind-limb, the local symptoms—viz., swelling and tenderness—commence in the inguinal region, and extend downwards. In erysipelas, when affecting a hind-limb, the local symptoms start in the region of the hock, metatarsal region, or coronet, and extend upwards. The pain on pressure is by no means so acute in lymphangitis, and it tends to become relieved when swelling occurs; also, sloughing of the skin and subcutaneous tissues is rarely met with.

From Malignant Œdema.—In malignant œdema the raised margin of skin found in erysipelas is not present. The tendency to early gangrene is well marked and the affected region often shows crepitation under the skin, due to the formation of gases in the subcutaneous connective tissue. In doubtful cases a bacteriological examination of the exudate will be necessary. This will decide whether the streptococcus of erysipelas or the bacillus of malignant œdema is present. In malignant œdema the wound through which infection has occurred can usually be detected.

PROPHYLAXIS.

Recognising the fact that the affection arises from the entrance of the streptococcus into a wound, it is clear that careful attention to surgical cleanliness, antiseptic precautions, and hygienic surroundings, both in surgical and ordinary wounds, are the important points with reference to prevention.

Cases of erysipelas should be isolated, and attendants having charge of such should not be allowed to dress wounds in other animals. The soiled dressings of wounds should be destroyed, and the instruments must be sterilised by boiling. The litter should not be allowed to come into contact with healthy horses. The practitioner should adopt special precautions with reference to sterilising his hands after attending a case of erysipelas, as he is liable to infect wounds when dressing them or when performing operations.

TREATMENT.

In the sthenic type of the disease a moderate dose of purgative medicine is indicated.

In asthenic cases, and in the secondary stages of all types, purgatives should not be employed; but laxative doses of sulphate of magnesia may be given in the food or drinking-water.

In the case of animals in high condition, when pain is acute and constitutional disturbance marked, the administration of aconite often proves useful. It may be combined with solution of acetate of ammonium, and repeated at intervals.

In debilitated cases aconite should not be given, but the pain may be relieved by a hypodermic injection of morphine. When weakness and debility are prominent symptoms, the judicious administration of alcoholic stimulants is necessary. The administration of tincture of iron has been advised as a specific in erysipelas, but we have never found it of any value.

Local treatment is of special importance. Some authorities advise the application of hot water in the form of fomentations. In our experience such treatment is likely to increase the tendency to suppuration and sloughing of the skin. We have found that the best local application to relieve pain and to exert a favourable influence on the course of the disease is a liberal dressing of ichthyol ointment combined with extract of belladonna. Or these agents may be mixed with glycerine, and applied thickly to the affected parts twice daily, and covered with a thick layer of cotton-wool and a soft bandage.

Thiol, in a 20 to 40 per cent. solution painted over the affected part, has also been found useful. In mild cases a lotion containing tincture of opium, belladonna, and liquor plumbi, proves of value; or the glycerinum belladonnæ (1 to 4) may be used.

When the swelling is extensive and tension well marked, free scarification is advised by some authorities, in order to relieve the tension in the skin and permit the effusion to drain off. Other practitioners condemn this treatment as likely to favour the entrance of micro-organisms and induce suppuration. We believe that neither scarification nor incisions should be carried out until there is definite evidence of suppuration beneath the skin. When such is present, free incisions should be made, so as to afford proper drainage. Hot compresses of boric lint should then be applied, but not continued too long.

When all sloughing and suppuration have ceased, dry antiseptic applications should be employed. Of these, we have found crystallised boric acid, applied in a liberal manner, to be the most useful, the parts being protected with a large dressing of antiseptic cotton-wool and a soft bandage. The unhealthy sores or ulcers which so frequently result from erysipelas, especially when affecting the coronet, fetlock, etc., should first be treated with the judicious application of hot compresses of boric lint, then stimulated to healthy action by nitrate of silver, or preferably sulphate of copper, and a dry dressing of crystallised boric acid applied. A dressing composed of iodoform, boric acid, and zinc oxide, often acts well in such cases.

A thickening of the limb is frequently met with after acute symptoms have disappeared. This is best removed by the internal administration of potassium iodide, and the local application of a liniment containing iodine.

In cases of erysipelas, when there is inability to place any weight on the affected limb, the use of slings will be necessary.

SERO-THERAPY.—Marmorek's antistreptococcic serum has been employed with a variable degree of success in the treatment of erysipelas. It is well worthy of a trial in severe cases, and may be given in similar doses to those advised for purpura hæmorrhagica (*vide* the section on Purpura Hæmorrhagica).

ACTINOMYCOSIS

By G. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.,
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Synonyms.—Ray-fungus disease ; Wooden tongue ; Lumpy jaw.

French : Actinomycose.
German : Aktinomykose ; Strahlen-Pilzkrankheit.

Definition.—A specific disease affecting cattle particularly, occasionally horses and swine, and more rarely sheep and deer ; also communicable to man. It is due to the inoculation of a vegetable parasite, known as the *Actinomyces* or Ray-fungus.

GENERAL REMARKS AND ETIOLOGY.

The definite rôle of the Actinomyces in the causation of the disease was first clearly established by Bollinger in 1877. It had been observed by him previously, and also by Perroncito, Rivolta, and Hahn. The disease has a very wide distribution, being known in almost all parts of the world. It is generally sporadic, but in the British Isles it often assumes an enzoötic character, being quite common in some districts and unknown in others. It appears to occur with greater frequency in low-lying or marshy districts. Bang and Jensen record an outbreak amongst cattle of a seaside locality in Denmark that had been fed on barley grown on reclaimed land.

The disease is not contagious in the ordinary sense of the term, since healthy animals appear to cohabit with affected ones with impunity. In an experiment conducted by Dr. Salmon, of the U.S.A. Department of Agriculture, twenty-one healthy animals were tied between animals affected with the disease, so that they were compelled to eat food contaminated with the discharges from the tumours, etc. After four months of such exposure, not one of the former showed any evidences of the disease, either during life or when slaughtered.

When several animals in the same herd are affected, it is probable that they have each contracted it from a common source, and not from one another. The disease may, however, be transmitted by experimental inoculation from animal to animal. Crookshank quotes a case in which he successfully inoculated a calf with material derived from the disease in a human being.

On account of the frequency with which actinomycosis affects the tongue, and the nature of the lesion, it is often called "wooden tongue" in this country, while in America the name "lumpy jaw" obtains. The frequency with which the disease is found about the mouth suggests that the parasite is conveyed by the food, and gains access by wounds or lacerations of the mucous membranes. It is generally believed that the organism grows on various cereals, but most abundantly on barley. The latter, of all cereals, is most likely to wound the mucous membranes; hence it is not surprising to find that barley grains or pieces of barley awns or straw have been discovered in the lesions. Wounds caused by eruption of teeth and caries of teeth also afford channels of infection. Skin wounds produced by wire fencing or by rubbing may also become infected. Gooch relates an experience in which twenty-two animals were setoned by him, the first one operated upon being the subject of a "wen" at the time. The remaining twenty-one all became affected, and Gooch concluded that they were inoculated from number one.

The disease is not a very fatal one. It generally produces its effects by mechanical interference with prehension, deglutition, or respiration. Fröhner, however, includes Actinomyces in a list of organisms which produce their effects by toxins—a view with which the writer disagrees. The majority of cases, if brought under treatment sufficiently early, can be cured, or, at any rate, improved to such an extent as to permit the animal to be profitably fed for the butcher.

Bacteriology—*The Ray-Fungus or Actinomyces.*—The organism responsible for actinomycosis belongs to the class Streptothricæ. It is a bacterium which has the power of branching in its growth. It is a pleomorphic organism occurring in three forms—viz., clubs, filaments, and spherical forms resembling cocci. Several varieties of streptothrix have been isolated having the power to produce similar lesions. That one, however, which is commonly found in actinomycosis in cattle has been called the *Streptothrix bovis*, or *Actinomyces bovis*.

If a drop of actinomycotic pus or a scraping from a freshly-cut lesion be examined on a glass slide, small granules visible to the naked eye may be observed. If these are crushed by means of another slide or cover-glass, and examined under a low power of the microscope, they will be seen to be made up of a number of club-shaped bodies, radiately arranged with their attenuated extremities towards the centre. These are considered to be a late stage in the development of the parasite. If a section or scraping of a growth at a younger stage be taken and suitably stained, the organisms will be seen as a bundle of filaments more or less irregularly arranged, branching and sending offshoots into the surrounding tissue. The filaments or threads are irregularly segmented, and consist of a body and envelope. When a branch leaves the parental cluster it may meet with greater resistance, and its growth be curtailed. It appears as though the envelope then swells at its distal extremity, and so gives it the characteristic club-shaped appearance. Single clubs can thus be seen, each with a central filament terminating about half-way up the club. Amongst the cluster of threads in the centre may also be distinguished collections of small round bodies, some of which appear refractile. They somewhat resemble micrococci, but are regarded by some as spores of the organism. Sometimes strings of " cocci " are seen continuous with typical streptothrix filaments. In very old colonies the whole of the filaments spreading from the central mass have assumed the clubbed termination, and frequently the central filaments themselves have perished, their position being occupied by a granular detritus. Some of the clubs themselves may be bifid or palmate, sometimes arranged like rosettes, and at other times in banana-like clusters. Again, in other specimens phagocytosis may be observed, large leucocytes holding the fungus in their protoplasm.

Staining.—For diagnostic purposes in old-standing cases staining is not necessary, as the colonies in pus or scrapings may be recognised unstained with a low magnification. The clubs are easily stained by Plaut's method (carbol-fuchsin followed by alcoholic solution of picric acid). The filaments and coccus-like bodies stain readily by the Gram or the Claudius method.

Cultural Characters.—The ray-fungus is not easily cultivated artificially, numerous attempts being necessary before a single positive result is obtained. It is a facultative anaërobe—*i.e.*, though it will grow in the presence of air, it can be cultivated much more readily under anaërobic conditions (Wright). It grows best in glycerine-bouillon or on glycerine-agar at or about 90° F. In the former it grows as small white granules at the bottom of the medium, and on the latter it commences after a few days as small white shining grains, which later coalesce. Old cultures become yellow and dry-looking.

MORBID ANATOMY.

Macroscopic Characters of the Lesions.—The common lesion consists of a formation of new fibrous tissue at and around the seat of inoculation of the ray-fungus, frequently with suppurating foci, which constitute small abscesses in the substance of the growth. They may point and burst, emitting pus, which feels gritty on being rubbed between the thumb and fingers. The consistence of the lesion varies. Most are firm and of the consistence of a fibroma, and greyish-white in colour; others have a soft, almost sarcomatous consistence, and are yellowish-red in colour.

On cutting into them one can usually see yellowish specks, varying from the size of a small pin-head to that of a pea, scattered through the connective-tissue mass. In those cases with pus production, abscesses of varying sizes may be seen, containing pus with macroscopic colonies and surrounded by soft granulation tissue. If a little pus or a scraping from a freshly-cut growth be spread on a piece of glass, small granules, yellowish-white and about the size of a small pin's head, may be seen. These constitute colonies of the parasite. When the tongue is affected, an indurating glossitis is set up. It commences with the formation of raised nodules, which are hard and firm to the touch. Extending inwards from these, there is a marked proliferation of fibrous connective tissue, which may extend into the muscular tissue. There is frequently ulceration of the mucous membrane, the base of the ulcers showing minute yellow spots. The increase in size of the tongue makes that organ too large for the mouth, and it is consequently protruded to a varying degree. The tongue lesion rarely suppurates. The outside of the face and the intermaxillary space are affected in a large proportion of cases, but the condition is rendered much worse when the superior or the inferior maxillary bones are invaded. Then a periostitis is followed by a rarefying ostitis. The circumference of the bone is largely increased, but its density markedly diminished, owing to the formation of spongy cavities in it. The skin over the swelling frequently ulcerates and shows granulations. The abscesses may burst and discharge the characteristic pus. This form affecting the jaw used to be erroneously called "osteosarcoma" in the old days. The pharyngeal growths are usually, but not invariably, pedunculated, forming polypi, which vary in size from a pea to a hen's egg. Sometimes the pharyngeal lesion resembles a simple superficial abscess involving the mucous membrane and submucosa. In other cases the pharyngeal lymphatic glands become affected and considerably enlarged, and may cause much trouble in diagnosis, since the only discoverable change during life is a more or less constricted pharynx without any surface irregularity. The increase in size of the glands in these cases is due to what may be described as the characteristic actinomycotic tissue, it is rarely attributable to suppuration. The respiratory apparatus may become affected with actinomycosis—viz., the larynx and trachea or the lungs. In the former case the lesions are usually polypoid, but in the lungs the lesions are either soft tumour-like growths, varying in size up to one's fist, and containing suppurating foci, or they may be practically abscess cavities. Miliary actinomycosis has also been recorded. The pleura may become implicated by extension from the lungs. Other rare seats are the liver, mammary glands, spermatic cord, and stomach.

When an actinomycotic mammary gland of a cow is cut into, the appearance closely resembles tubercular mammitis, showing either diffuse induration, with small yellow foci, or isolated nodules, with or without degenerating centres. Generalisation may occur, but is extremely rare.

Microscopic Characters of the Lesions.—Sections of an actinomycotic growth suitably stained will generally show dotted throughout it colonies of the ray-fungus similar to those mentioned as occurring in the pus. These are surrounded by round cells or leucocytes, cells of an epithelioid type, and occasionally giant cells. Passing outwards, one meets with spindle-shaped cells and then inflammatory fibrous tissue, of which the bulk of the new growth consists.

SYMPTOMS.

In Cattle.—The symptoms of actinomycosis almost entirely depend upon mechanical interference, and consequently vary with the seat of the lesions.

The lesions take the form of more or less dense fibrous new growths, some being well circumscribed at their bases, while others are diffuse, and the line of demarcation between their limits and the surrounding healthy tissues cannot be well made out. When there is a systemic disturbance, it is generally due to some complication rather than to the ray-fungus. The parasite appears not to produce any toxic material, such as is responsible in most febrile diseases for the elevation of temperature, and consequently the thermometer registers little or no alteration from the normal temperature. Respiration, the appetite, secretions and excretions, are not altered unless the growths previously referred to, impinge upon the organs concerned. Thus, when the tumour is confined to the skin and subcutis, frequently no symptoms other than the presence of the growth may be shown. The skin may be either intact, or ulcerated, granulating, and discharging pus from one or more foci. When the tongue is affected to anything more than a very slight degree, prehension is interfered with, and may be completely prevented, and the patient loses flesh and may even die of starvation. The tongue is enlarged, protruded, almost as hard as a board, and ulcerated.

The bases of the ulcers often show minute yellow spots. According to Henschel and Falk, quoted by Ostertag, the most common seat of the ulcers is at the junction of the tip and body of the tongue.

There is dribbling of saliva, and when ulceration is present the discharges are offensive in odour. When the jaw, upper or lower, is the seat of the lesions in the early stages the inconvenience appears slight; but later mastication is rendered difficult, and so nutrition is not well maintained except with crushed concentrated foods requiring less grinding. The growth here is very likely to burst, either outwardly or inwardly, the discharge feeling quite gritty. Sometimes the tumour invades the alveoli of the molar teeth, which may become loosened or displaced. Not infrequently one finds the skin and muscles of the cheek affected, and the bones not interfered with. The most varying symptoms are shown when the pharynx is affected. If the growth is superficial, it is generally pedunculated, and may cause intermittent roaring and dysphagia. If the polypus passes into the glottis, asphyxia may supervene. If, on the other hand, the offending growth passes during swallowing into the entrance of the gullet, choking occurs, with its attendant symptoms of tympany, etc. The pharyngeal lesion may take the form of a superficial abscess causing dyspnœa, dysphagia, and possibly asphyxia. If it ruptures, the pus may be swallowed, or it may escape by the nostrils, or pass into the larynx and induce broncho-pneumonia. The disease may invade the lymphatic glands in the region of the throat (retropharyngeal). The symptoms in such cases are those of roaring and difficult respiration and dysphagia, food often being returned by the nostrils, and on examination externally one may or may not appreciate an enlargement in the pharyngeal region. These cases are generally more acute than the average case. The dyspnœa develops rapidly, and unless put under urgent treatment asphyxia may occur. The lips and muffle are sometimes affected, the parts swelling diffusely to such an extent as to occlude the anterior nares and to cause oral respiration.

The skin and subcutaneous tissues are frequently inoculated with actinomyces, and the lesions produced are known in the British Isles as "wens" or "clyers." The latter term is often limited to the growth when it affects the head or pharyngeal region,* but wens may occur anywhere in the skin. Commencing as a small tumour, a wen may rapidly increase in size up to several times the size of one's fist, sometimes pedunculated, sometimes attached by means of a broad base. The surface of the skin may ulcerate and discharge a purulent material, which together with brown crusts may cover the growth. In these cases, when the surface is wiped, a red granulating area, sometimes showing yellow spots, is revealed. The writer has also met with a case in which a foreleg was affected, becoming diffusely thickened, and with a circumference of 18½ inches below the knee (elephantiasis).

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* In some parts of the British Isles, particularly in Scotland, the term "clyers" includes any similar pharyngeal enlargements such as those due to tuberculosis.

Other rare seats of the affection are the spermatic cord after castration, the udder, the lungs, the liver (Ostertag), the kidneys (Bang and Jensen), and the stomach.

Actinomycosis of the mammary gland of the cow is not a common condition, but it is probable that it exists to a greater extent than is generally believed, and is mistaken for tubercular mastitis. It may occur either in the form of firm isolated nodules, varying in size from a hazel-nut to several times that size, or in the form of a diffuse indurating mammitis. Cases of mammary actinomycosis have been recorded by Rasmussen, Phail, Maxwell, and Patterson. The latter found actinomycosis in the udders of five out of fifty cows suspected of tuberculosis.*

Actinomycosis of the Horse.—This disease is not common in the horse, but cases have been recorded affecting the submaxillary lymphatic glands, the tongue, abdominal wall, and the spermatic cord after castration. The first-mentioned is probably the most common site (McFadyean). The gland gradually increases in size, and may attain the dimensions of a cocoanut. As in the ox, it has a great tendency to burst at one or more points and discharge pus—a fact which renders diagnosis easy, since the pus can be readily demonstrated to contain the colonies of the ray-fungus. When the spermatic cord is affected, a tumour-like mass is produced adherent to the scrotal wall, and discharging the characteristic pus from several sinuses. When the tongue is affected, the organ becomes too large for the mouth, ulcerates and discharges, and may cause asphyxia.

The writer met with a case in 1911 affecting the upper lip of a horse. The lip was considerably thickened and bosselated, and was emitting pus through several small points, both on the surface of the skin and on the buccal aspect of the lip. The pus was more or less viscid, non-gritty, and occasionally streaked with blood. Botriomycosis was first suspected, but on microscopic examination of the pus the ray-fungus was readily demonstrated. The disease yielded to the local application of tincture of iodine injected into the sinuses, and the internal administration of

* In these five cases the lesions were confined to the udder, so that it is probable that infection occurred by way of the teat from the bedding.—Ed.

iodides. Markus recorded a case of actinomycosis of the rectum in a mare; the lesion occurred in the rectal wall about 4 inches in front of the anus, and produced rectal stenosis.*

Actinomycosis of the Pig and Sheep.—The most common site of this disease in the pig is the mammæ. It may also occur in the throat and the castration wounds of males and females. Infection is probably conveyed through straw. The mammary lesion usually consists of a tumour-like mass containing a purulent centre, which may communicate with the exterior and liberate the characteristic pus (a cold abscess) with colonies of the ray-fungus. As in other animals, generalisation, though rare, may occur.

In the sheep actinomycosis has been described affecting the tongue and lips (Berg) and the lungs (Grips).

DIFFERENTIAL DIAGNOSIS.

It is probable that more than one variety of streptothrix is capable of producing symptoms closely resembling those of actinomycosis. One such condition has been called *actinobacillosis* (see p. 269). In this latter disease it is said that the lymphatic glands are more likely to become affected. The affections are, however, practically indistinguishable clinically.

Tuberculosis is the condition most likely to be confounded with actinomycosis in cattle, and in very many cases it is quite impossible during life to definitely distinguish between them. Tuberculin is not of much assistance, as, although the lesion in question may be actinomycotic, yet tuberculosis in another part of the body would cause a reaction. In all cases the diagnostic feature is the discovery of the ray-fungus in the pus or in a small portion of the growth which may be removed for the purpose. The gritty condition of the pus is not diagnostic. The grittiness is not always very pronounced, and pus from a tuberculous lesion would also be somewhat gritty if any degree of calcification had occurred. Assistance in arriving at a decision may be obtained by remembering the fact that the lymphatic glands associated with the affected part are always involved in tuberculosis, but comparatively rarely in actinomycosis. The latter may also be distinguished by the improvement usually manifested when the patient is submitted to the iodine treatment.†

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* *Vide Veterinary Record*, December 2, 1911.

† Actinomycosis of the udder may easily be mistaken for tubercular mastitis during life, and even after death the lesions can only be distinguished by microscopical examination, so closely may the macroscopical appearances resemble each other in both affections.

In the horse, according to the seat of the affection, actinomycosis may be confounded with *glanders*, *botriomycosis*, and a *streptococcic infection of the tongue*. It is only likely to be confused with glanders in the early cases, for in glanders the submaxillary enlargement never attains such large dimensions, and rarely bursts. The demonstration of the ray-fungus in actinomycosis disperses any doubt. Streptococcic affections of the tongue have been erroneously diagnosed as actinomycosis. The tongue becomes *rapidly* enlarged and protrudes from the mouth, an acute glossitis being set up, whereas actinomycosis is essentially a chronic disease. The tongue may contain abscesses, with streptococci in large numbers (McFadyean). Actinomycosis of the spermatic cord may be confused with botriomycosis. The former condition, however, is very rare, and the latter very common. The only method of distinction is by the use of the microscope.

TREATMENT.

Treatment may be either medical or surgical, or both. Formerly it used to be exclusively surgical, and consisted of the extirpation of the growths where possible, or the external application or injection of caustics to the growths. Excision is still the best for pedunculated growths on the skin or in the pharynx. In the latter situation they are often difficult to reach, but may be snared with the aid of a mouth-gag and a thin metal tube and piece of stout wire. With the exceptions of those cases, by far the best results are obtained by medical treatment, potassium iodide being a specific for the disease in cattle. The dose should be regulated according to the size of the patient, and should be about ¼ drachm for each 100 pounds body-weight—that is, from 2 to 3 drachms once or twice daily. It is best given dissolved in about a pint of water and administered in the form of a drench, in order to insure the animal

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Patterson (*Journal of Meat and Milk Hygiene*, January, 1911) records a case in which a specimen of suspected tubercular mastitis was submitted to him for examination, and gives the following description: "The specimen on palpation and to the naked eye had all the appearances of being tubercular. When cut into the organ was found to contain large numbers of small cream-coloured foci, similar to tubercles, varying from the size of a pin's head to that of a split pea. The substance of the udder was also of a brownish tint, so often seen in cases of tuberculosis of that organ. On smear preparations being made in the usual way, and examined microscopically, no tubercle bacilli were found. Convinced, however, that the specimen was tubercular, and that the failure to find the bacilli was due to a small number being present (as I have often experienced), I prepared and cut sections in paraffin. On staining these sections by Ziehl-Neelsen's carbol-fuchsin, I was surprised to find the lesion was entirely due to actinomycosis."

The differential diagnosis of actinomycotic lesions of the pharynx and tubercular lesions of the same region is generally impossible during life.—Ed.

getting the whole of it.* After four or five days or a week the animal may show signs of iodism in the form of diminished appetite, catarrh of the nasal mucous membrane and conjunctiva, the fæces become dry and may be coated with mucus, the skin is covered with scurf and the hair falls off in patches. The potassium iodide must then be stopped for two or three days. During this interval 4 grains of biniodide of mercury may be given with advantage, as the growth otherwise increases somewhat when the iodide is stopped. The course of treatment varies, apparently, according to the susceptibility of the animal to iodism. Improvement is most marked in those animals showing signs of iodism, and in them, with proper intervals, a period of four to six weeks will generally see recovery complete. In other cases a longer time may be necessary, while in some cases the patient appears to be immune to the effects of iodine, and treatment fails. In favourable cases the enlargement of the tongue or the tumour gradually subsides and disappears, leaving only a small cicatrix to mark its previous existence if there has been a granulating skin wound. These good results, however, are not so well shown when the bones are invaded, as the damage to these structures is not so easily repaired. It is doubtful if the case is worth treating if the swelling of the bone much exceeds the size of a goose's egg. Biniodide of mercury is sometimes used throughout as a substitute for potassium iodide. The daily dose should be 3 or 4 grains dissolved with the assistance of 10 grains of potassium iodide, and administered as a drench in a pint of water. Although fairly good, it is slow in action, and its effects are not so generally satisfactory.

When there is ulceration or abscess formation, these conditions must be treated accordingly, by curetting and syringing, or packing the cavities with wool saturated with iodised phenol or Lugol's solution. Law suggests that where potassium iodide fails in cases with purulent complications, injections of antistreptococcic serum may be used with advantage. He also states that good results may be obtained by injecting potassium iodide solution twice a week into the tumour instead of giving it by the mouth. Some veterinary surgeons scarify the tongue and rub in biniodide of mercury ointment; others apply strong nitric acid to the wens, and claim very good results for it. The last two lines of treatment appear to be both barbarous and unnecessary.

The writer has observed in some cases that more or less acute inflammatory phenomena, such as heat, redness, and increased pain, are set up in the lesions after a few days' administration of the iodide. In some cases in human beings, also, where the specific action of the drug is much less marked, the discomfort is very great and the pain so unbearable that the drug has to be discontinued. In addition to the effects on the diseased parts, potassium iodide, when administered for long, produces emaciation and glandular atrophy. This latter point should be particularly borne in mind in the cases of males required for stud purposes, as it is said to produce sterility. These ill effects, however, can be largely avoided by regulating the doses and intervals, and seeing that the patient gets plenty of nutritious and easily digested food. Where the tongue is affected to more than a slight degree, liquid food, as milk and thin gruel, must be given, and it may be necessary to administer them by bottle. Great care, however, must be taken in drenching these animals, as inhalation pneumonia may easily occur owing to the difficulty in swallowing. Another word is necessary with reference to milch cows. By the administration of potassium iodide the milk yield is considerably reduced, and some of the iodide is excreted by the mammary gland. Consequently the milk is tainted and rendered unfit for food, and, of course, the client should be informed of this fact previous to the treatment being adopted.

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* By administering this drug in thin linseed gruel we have found it to give better results than in any other menstruum.—Ed.

MEAT INSPECTION.

As already described, the human subject is susceptible to actinomycosis as well as some of the food-producing animals, but there is no satisfactory evidence of infection of man from diseased flesh or milk. It is more probable that both obtain infection from a common source; moreover, the organism is, generally speaking, confined to the lesions. That being the case, the carcass of an animal affected with actinomycosis, provided it is otherwise healthy and well nourished, may be passed for human food with safety, the diseased part only being condemned. Local actinomycosis with emaciation would justify condemnation of the whole carcass on the latter grounds. The whole carcass should be condemned in those rare cases of generalisation.

NOTE BY EDITOR.

Actinomycosis in the Dog.—Actinomycosis has several times been encountered in the dog. Vachetta observed it in the form of a tumour affecting the jaw; Rivolta in the bloody pleural exudate, the pleural surfaces being covered with warty processes; Rabe, three cases of a purulent glandular infection, one of the cases being complicated with a purulent peritonitis; and Hartl, as a tumour in the wall of the chest, and in the anterior mediastinum.

Actinomycosis in the Cat.—Hartl isolated a fungus resembling the actinomyces in the purulent peritoneal exudate of a cat. The pus was palish yellow and resembled boiled sago.

Actinomycosis in the Bird.—Furlau, in four geese, aged three months and showing pericardial and peritoneal lesions covered by a thick gelatinous exudate, and infiltrated and friable glands found in the exudate and scraping of the glands "colonies characteristic of the actinomyces."

Actinomycosis in the Lizard.—Actinomyces have been found in nodules in the liver of the *Lacerta agilis* and *L. viridis.* Inoculations of cultures from these produced the disease in these species as well as in the *L. muralis* and the *Coluber viridflavus* (Terni and Galli-Valerio).

Actinomycosis in Man.—The first accurate description of this disease in man was given by James Israel, and later on Ponfick pointed out that the affection in man and cattle was identical. Erving collected 100 cases of human actinomycosis in America in 1902. No case of infection from animals to man of this disease has been recorded, either by milk, meat, or by inoculation. As regards the mode of infection in man, authorities believe that the causal agent is probably taken in with the food, and the site of infection in the majority of cases is in the mouth or neighbouring passages. The habit of "sucking" portions of straw or hay is regarded as a source of infection, more especially when a carious tooth is present, as the ray-fungus may be introduced by this means.

The lesions occur in the jaw in a number of cases, the symptoms presented being a swelling of one side of the face, or a chronic enlargement of the jaw, simulating sarcoma. Lesions have also been observed in the tongue, intestines, peritoneum, liver, lungs, bronchi, vertebræ, ribs, sternum, pleural cavity, cerebrum, and skin.

Subcutaneous abscesses and metastases in all parts of the body have occasionally been met with.

What is known as miliary actinomycosis occurs in the lungs, the nodules being made up of groups of the ray-fungus surrounded by granulation tissue; pulmonary abscesses have also been observed.

The treatment adopted is largely surgical, but the disease appears to be of a far more serious character in man than in cattle.

According to Osler, the average duration of the affection is ten months, and recovery is very rare. The administration of potassium iodide in doses of 40 to 60 grains daily is said to have proved curative in a number of cases. The tolerance of such large doses by man should encourage us to push the administration of this agent in those obstinate cases of the disease which we so frequently meet with in cattle. In our experience, when the disease is advanced, large doses of the drug are indicated, and often prove successful when the ordinary amounts fail. In advanced cases we find it of advantage to combine the biniodide of mercury with the potassium iodide, giving 5 grains of the former and 2 drachms of the latter, three times a day.

ACTINOBACILLOSIS

By H. GRAY, M.R.C.V.S.

Definition.—A disease of the ox, resembling in some respects that of actinomycosis, but due to a bacillus which grows in the system in a radiate manner.

History.—This disease was studied by Lignières and Spitz, who encountered it in an epizoötic form in the Argentine Republic during the summer of 1900 and 1901. It was widely spread, and attacked cattle of all ages. Horses and pigs were not affected, and sheep but rarely. It has also been encountered in France, and in the opinion of Nocard it will be found everywhere, and is confounded with actinomycosis.

Etiology.—The specific agent of the disease is a small bacillus, which, when it grows in the system, manifests itself in the form of club- or crook-like swellings. Contagion is evident in the extension of the disease, which is sometimes very easily contracted. It may, however, be often encountered in a sporadic form. The infection takes place through abrasions, chiefly of the mouth, such as are seen in foot and mouth disease, or during the process of dentition. It is a recurrent disease, one attack not giving immunity to a future one.

Bacteriology.—Microscopical examination of the pus shows granules or characteristic club-shaped tufts. The granules are composed of a single or, by reunion, of a certain number of tufts. The single granules may measure 29 to 139 μ. The club-shaped masses have an aspect identical to that of actinomycotic granules, and are easily coloured with acid stains. Examination of the pus after coloration by aniline stains or by Gram never shows filamentary forms.

The pus, collected aseptically and inseminated, does not, as a rule, give any growth; but if it is carefully rubbed up in a mortar, it always shows a culture at the end of twenty-four hours' sojourn in an incubator. The temperature of the room is not sufficient.

The first cultures are distinctly bacillary; later on they take the form of a cocco-bacillus or diplococcus; in old cultures they often show various forms. They are easily coloured by aniline stains, but are decolorised by Gram. The rods measure 1 or 2 μ in length by 0·4 μ in breadth; they are immobile, and do not form spores.

Agar is the best medium for the growth of the actinobacillus. It grows with difficulty on gelatine. Milk is never coagulated. In liquid serum the culture is uniformly distributed at the bottom of the tube.

The actinobacillus is a facultative anaërobe. It yields to heat and to desiccation. Pus preserved in hermetically sealed pipettes may be active at the end of a month.

Symptoms.—Nearly every tissue may be invaded, but the skin, especially in the vicinity of the throat, is the one most frequently attacked. In this region a tumour develops in the subcutaneous connective tissue, and may reach the size of one's fist. At the end of nine or ten days it is transformed into a large abscess, which brings about ulceration of the skin, and then discharges pus resembling thick wheat-flour paste.

Except in the jaw, one never finds yellowish granules or calcareous particles. Exuberant granulations form round the opening of the abscess cavity, and develop into greyish or scabby patches. Sometimes the abscess is situated on the sides or flanks.

The glands are commonly affected, but, as a rule, only secondarily. When the deeply-situated glands are involved, digestive or respiratory disturbance is noticed.

The tongue is often affected, and resembles that seen in actinomycosis. The actinobacillary foci are scattered in the substance of this organ. Neither in these lesions nor in the pus does one find filaments coloured by Gram.

The disease may occasionally attack the pharynx, the salivary (especially the parotid) glands, the udder, lung, and bone.

Diagnosis.—Actinobacillosis preferably attacks the skin, and rarely the jaws ; and, secondarily, the glands nearest the organ or tissues that become attacked are invaded. The pus, save that in lesions in the jaw, is never yellow in appearance. Filamentary forms are never found. The organism does not take Gram. The serum of the affected subject agglutinates the microbe in the liquid culture. These characters serve to distinguish it from *actinomycosis*.

The tufts are coloured yellow by glycerine picrocarmine, while the elements of pus take a pink tint.

Treatment.—Thomassen's treatment easily overcomes the lesions in the soft tissues, but those in the bone are not subdued. Two or three drachms of iodide of potassium should be given daily in the food for a fortnight. Solution of iodine may be injected into the tissues, in lieu of the internal administration of the iodide. Abscesses should be opened, curetted, and plugged with tampons soaked in carbolised iodine or some other antiseptic. Isolation should be enforced.

Bibliography : La Actinobacillosis, La Rivista Veterinaria, 1902, p. 77; Boletin de Agricultura y Ganaderia (Republica Argentina), 1902, p. 169; Actinobacillose, brochure, Buenos Ayres, 1902 (with plates); Recueil de Méd. Vét., 1902 ; and Nocard et Leclainche, Les Maladies Microbiennes, 1903.

BOVINE FARCY*

By H. GRAY, M.R.C.V.S.

French : Farcin du bœuf ; Farcin de France ; Arboulets.

Definition.—This is a chronic disease of the ox, characterised by a suppurative inflammation of the superficial lymphatic vessels and glands, and due to a special streptothrix, considered by Sauvageau and Radais as a hyphomycete fungus—*Oospora farcinica.*

History.—This disease was formerly rather prevalent in France, but to-day it is almost unknown. It was studied by Sorillon in 1829; by Maillet and also by Mousis in 1837, Festal in 1844, Lafosse in 1853, and Cruzel in 1869. It has been seen occasionally by Mauri in the Haute-Garonne. In 1888 Nocard made an experimental study from lesions sent from Guadeloupe, where it exists in a very grave form. It occurs in Mauritius as an enzoötic.

Bacteriology.—The streptothrix, in the form of entangled branching filaments, is found in the pus or tissues when they are stained well by Gram-Weigert, Nicolle's carbolised Gram, and better still by Ziehl, with decolorisation of the stroma by aniline hydrochlorate.

The microbe is an aërobe which grows between 30° and 40° C.

It grows well in bouillons, agar, gelatinised serum, and potato. It does not coagulate milk.

The cultures are pathogenic to the ox, sheep, and guinea-pig. The rabbit, dog, cat, horse, and ass, are refractory.

Symptoms.—It attacks cattle of all ages, and shows itself on the limbs in the form of circumscribed tumours or cords, the latter being the commoner of the two.

The cords are slightly hard and insensible, and follow the course of the superficial veins of the limbs, especially on the internal face of the metacarpal and metatarsal regions, the fore-arms and thighs, and very rarely those of the neck. The chronic inflammatory process extends to the neighbouring lymphatic glands, which become more or less swollen, but not painful. Very rarely the cords, which are of the calibre of the finger, form a circumscribed abscess. On the contrary, they frequently become pasty, and fluctuate at a certain part of their length. Sometimes this pastiness extends to five or six fingers' breadth, but it is rare for the suppurative process to extend to the skin and cause it to ulcerate. On opening the soft part of the cord and pressing out the contents, it is found that the contained material has a whitish, odourless, sebaceous appearance, which sometimes has the consistence of cream cheese. The opening into the lymphatic vessel rarely suppurates, but closes up in five or six days.

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* This article has been compiled from Nocard and Leclainche's "Les Maladies Microbiennes des Animaux."

In some instances the pressed-out cord forms new pus, which partly becomes absorbed. In other instances this pus forms small chains of encysted abscesses. Generally, induration of the lymphatics supervenes without any manifest fluctuation, and when such a cord is incised one only finds a lardaceous tissue which is very adherent to the skin or becomes confounded with it.

The circumscribed tumours are less frequent than the cords, and are found round the knee, the external faces of the fore-arms and thighs, neck, etc. Like the cords, they are slow in forming, are insensible, remain a long time without undergoing any change, and rarely suppurate. If they are incised, they are mostly found to have a lardaceous, whitish or greyish appearance, which is rarely homogeneous. Very often around a tumour of this nature one or more cords radiate, of which one end is lost in the substance of the tumour. At times the tumour contains one or more small encysted purulent foci.

The *Guadeloupe form* of bovine farcy as described by Couzin is a much more virulent type of the malady.

It seems to commence by inflammation of one of the lymphatic glands, which becomes swollen, hot, painful, and œdematous, slowly increases in size, and ends up by manifesting the characters of an indurated cold abscess, which, when opened, gives escape to a thick creamy and sometimes caseous or clotted pus. The walls of the abscess are very thick, indurated, lardaceous, and creak under the knife ; they are lined internally by a soft downy granulation. After the abscess is opened, the animal seems to return to health ; but it is not long before other tumours form and run a similar course to the first, and the animal dies in a state of marasmus.

Course.—According to Maillet, bovine farcy runs a very slow course, which may extend to a year, eighteen months, or even longer, without the general health of the animal undergoing much change. The presence of cords and swelling in the limbs does not necessarily cause lameness, but the working animal soon becomes tired. Milking cows give their usual quantity of milk; but it is very difficult to get an animal to put on fat.

Lesions.—Couzin found in every case purulent collections in the glands of the limbs and trunk. In some animals the lungs, liver, spleen, and internal glands, were crammed with nodules, the central part of which had undergone caseous or purulent changes. In the vicinity of the caseous centres a considerable number of small nodular or tuberculiform follicles were discovered, containing in the centre thick tufts of the streptothrix. Large softened nodules were found at times in the lung.

Diagnosis.—Clinically this disease is liable to be confounded with *epizoötic lymphangitis*; but this latter disease attacks horses as well as cattle, and is due to a cryptococcus, which is easily discovered in the pus. From ulcerous lymphangitis (which is encountered in the ox as well as in the horse) it is distinguished by the presence of the Preisz-Nocard bacillus in the lesions in the former disease.

It may also be confounded with *cutaneous tuberculosis*, which is not rarely encountered attacking the limbs of cattle in Great Britain. Microscopical examination of the diseased tissues and the application of the tuberculin test easily settles the differentiation.

From *cutaneous actinomycosis* it is distinguished by the presence of the actinomyces in the tumours of this malady, and by the response of the latter to the internal administration of iodide of potassium.

Treatment.—Prevention should be aimed at. The lesions may be removed by a surgical operation. Iodide of potassium is without any influence on the course of the disease.

STREPTOTHRICIC PSEUDO-TUBERCULOSIS IN THE HORSE

By H. GRAY, M.R.C.V.S.

DEAN found in a horse undergoing diphtheria immunisation, a hard nodule below and near the angle of the jaw. The swelling was about the size of a walnut, and appeared to be an enlarged indurated submaxillary gland. The animal was otherwise in good health, and did not respond to mallein or tuberculin. The nodule remained apparently stationary for about three months, then commenced to increase in size. In a week or two from this time it reached the size of a goose's egg, and a distinct fluctuation could be made out.

The pus which escaped was pale yellow in colour, and the material evacuated resembled the contents of a caseating tuberculous gland. No granules could be observed. In preparations stained by Kühne's methylene blue or Gram's method, there were observed, fairly uniformly distributed throughout the preparations, delicate filaments which suggested actinomyces. No granules consisting of interlacing filaments, nor clubs, nor rosettes, could be found. The filaments stained irregularly, giving a beaded appearance. They showed true dichotomous branching.

Lesions were produced in rabbits by inoculation. The organism somewhat resembled the *Micromyces Hoffmanni* on the one hand, and on the other the *Streptothrix Israeli.**

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* For further information on the biological characters of this organism, *vide* the Transactions of the Jenner Institute of Preventive Medicine, 1899, and the *Journal of Comparative Pathology and Therapeutics*, vol. xiii., 1900, p. 84.

For the differential characters of the latter-mentioned organisms, *vide* "Les Streptothricees," in "Technique Microbiologique et Serothérapique," par le Docteur Besson, 5e Édition, 1911.

BOTRIOMYCOSIS

By G. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.,
Professor in Charge of the Out-Patients' Clinique, Royal Veterinary College, London.

Synonyms.—Discomycosis equi.
French : Botryomycose.
German : Botryomycose; Botryomykose.

Definition.—A specific disease affecting horses, due to inoculation by a micrococcus (*Botriomyces ascoformans*), which produces a connective-tissue tumour called a "botriomycoma," with suppurating foci. According to Fröhner, the disease has on rare occasions been observed in cattle, swine, and the human being.

GENERAL REMARKS.

The organism responsible for the disease was first discovered by Bollinger in 1869, and later by Rivolta, Micellone, Jöhne, and Rabe. It is introduced into the body by inoculation, and is therefore a wound infection. The wound may be an apparently insignificant one, as a scratch inflicted by some part of the harness, such as the collar; or it may be an operation wound, as in castration or docking. The disease is very widespread and comparatively common. Fröhner records* that he operated on 400 cases in the years 1895 to 1903 in a surgical clinique of 8,000 diseased horses. These included 175 shoulder tumours, 156 fistulæ of the spermatic cord, and 75 other forms of botriomycoma. These latter included cases in various parts of the skin and in the udder of mares.

Generalisation occasionally occurs, but is comparatively rare. Cases have been recorded with secondary lesions in the lungs, spleen, liver, kidneys, and lymphatic glands. It is a sporadic affection, occurring generally in isolated cases, and showing little or no tendency to spread to other animals in the stable. Of course, if one affected animal is present, and pus containing the organisms is being discharged from the lesions, there is a chance of it being conveyed by harness and grooming utensils, and it might be spread in that way. But the development of the disease is so slow and insidious that the first case may almost be forgotten before the second one, infected from the first, shows appreciable lesions. It is so slow of development that horses castrated as yearlings, and inoculated then, frequently do not show symptoms of the disease until they are five or six years old, or even older. It is very rarely a fatal disease, except in those few cases of generalisation; but horses affected are rendered unfit for work, either by interference with some part of the harness, or, when the spermatic cord is the seat of the lesion, by interference with the gait, producing lameness.

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* "General Veterinary Surgery," Fröhner.

SYMPTOMS AND MACROSCOPIC CHARACTERS OF THE LESIONS.

The symptoms are almost entirely limited to the lesions. The latter are apparently more widespread and more frequently discharging than in actinomycosis, but as the affection does not usually interfere with prehension or mastication, the loss of condition is seldom so marked. The tumours may be single or multiple, and their size varies considerably. They may be the size of a pea or as large as one's head, or even larger. Their common sites are in the front of the shoulder (shoulder tumour), spermatic cord (scirrhous cord), the point of the elbow (an infected elbow tumour or shoe-ball), and the udder, while the same causal organism has also been found in lesions of poll-evil and fistulous withers. Wolstenholme records a case in which the lesions occurred at the lower posterior portion of the left thigh of a horse working in chain-gears. The positions of the lesions suggested that the trace-stretchers probably caused small wounds by which inoculation occurred. According to Friedberger and Fröhner, infection of castration wounds may take place through the medium of straw.

The earliest condition as affecting the shoulder not infrequently consists of a large, painful, deep-seated swelling, not very dense, and not fluctuating. This may subside after local antiphlogistic treatment, but in most cases it reappears at a later date in the form of a dense cold tumour.

The name "mycofibroma," which is sometimes applied, fairly well indicates the common characters of the older lesions wherever situated. The main substance of the new growth is dense fibrous connective tissue containing a varying number of suppurating foci, which are virtually abscesses. The skin over the enlargement may be seen to point and fluctuate, and then burst, emitting pus. The wound thus produced may then heal, and fresh abscesses form and repeat the process. Sometimes there are several openings, all communicating with purulent centres. The pus from a botriomycoma is characteristic in its appearance to the naked eye. It is sticky or mucoid, and if a little is spread on a slide a number of granules can be seen. The granules do not give a gritty feel when rubbed between the thumb and fingers, and they consist of colonies of the botriomyces or discomyces. When the shoulder is affected, the lesions frequently extend into the levator humeri muscle, and sometimes they completely surround the jugular vein. When the spermatic cord is the seat of the lesion, the new growth may be right at the end of the cord, and completely in the scrotal sac, while in some cases it extends up the cord into the inguinal canal. In these latter instances one rarely realises the extent of the condition and the difficulty of the surgical removal of the tumour until the patient is cast and a very thorough examination made. The tumours are well supplied with vessels, and are generally closely adherent by fibrous tissue to the walls of the scrotum. It is quite common for several fistulæ to run into the centre of them. A similar botriomycoma may develop on the stump of the tail after docking.

Botriomycosis of the udder of the mare is not uncommon. It is characterised by hard dense lumps or a nodular induration, with the skin usually closely adherent. Sooner or later fistulæ are developed, discharging pus in which typical colonies of the organism can be found. There is sometimes circumscribed abscess formation. Unterhössel has described a botriomycoma that weighed 35 kilogrammes, situated in the udder of a mare. The writer has met with several cases in which botriomycosis appeared to be limited to the skin and subcutaneous connective tissue of the gluteal region, and also to the region of the mane. In each of these cases the lesions were numerous, very painful, and about the size of a hazel-nut. The characteristic colonies of the organism were easily distinguished under a low magnification.

Gray has observed on several occasions botriomycosis affecting the pectoral region, an enormous tumour being produced, studded all over with fistulæ. In one of these cases the thighs were much swollen and showed several fistulæ.

When any of the botriomycomata are cut into after removal, they are found to be very dense and firm, almost tendinous in places, both as regards their hardness and their glistening appearance. In other parts they appear œdematous, and often show areas of yellowish fat. One or more cavities and channels of varying size may be seen, some very minute and others fairly large, but each containing the characteristic pus with granules.

Microscopic Characters.—Microscopic examination of sections under a low power reveals characters somewhat resembling those of actinomycosis. The mass of the growth is inflammatory fibrous tissue, with areas of fat, and varying-sized areas, which stain deeply, containing the botriomyces granules and maybe some pus-cells. These granules are best examined as obtained from pus. If one be squeezed between two thin slides, and examined under a low power, it will be seen to be almost opaque, and with an undulating margin, as though made up of a number of smaller rounded bodies packed closely together. If these are still further broken up, and stained either by methylene blue or the Gram method, they will be seen to be composed of considerable numbers of staphylococci held together by some amorphous matrix. There are no filaments and no clubs, and therefore they should be easily distinguished from those of actinomycosis. The pus also contains grape-like bunches of cocci besides those of the granules. These micrococci are indistinguishable from the *Staphylococcus pyogenes aureus.*

Cultural Characters.—The organism will grow on any of the ordinary media. On potato and on agar it produces a yellowish growth. It liquefies gelatine, and the growth becomes deposited as a yellowish precipitate. In these respects the cultures closely resemble those of *S. pyogenes aureus*, but they are said not to be so deep in colour as the latter. On microscopic examination the cultivated organisms do not reveal the two forms as found in the pus. The granules or colonies are never met with in artificial media, nor during acute stages in experimental cases inoculated with cultures. If such cases become chronic, however, pus may eventually be discharged, containing granules resembling those in naturally occurring cases.

DIFFERENTIAL DIAGNOSIS.

Although botriomycosis is fairly characteristic in its macroscopic appearances, the only certain distinction from apparently similar growths is the demonstration of the colonies of the organism. Otherwise it might be mistaken for actinomycosis, which on rare occasions affects similar situations. The same applies to tuberculosis. In each of these cases, however, the pus is frequently gritty when rubbed between the thumb and fingers, and the specific organisms are readily demonstrated. Scirrhous spermatic cords, too, are by no means all due to botriomycosis. The writer recorded a case in which a strong transfixing ligature was applied to the spermatic cord when castrating a colt with varicose spermatic veins. The ligature was not thrown off, and in three weeks a fibrous tumour as large as a man's fist had developed around the ligature at the end of the cord. This was excised, and no botriomyces could be demonstrated. In another instance a tumour 12 pounds in weight was excised by the writer from the spermatic cord of a pony. On examination it was found to be a myxofibroma, no organisms whatever being demonstrable. Numerous other examples of scirrhous cord occur in which no organisms whatever are discoverable. Elbow tumours are not usually botriomycotic in origin, but botriomycosis may be set up in them as the result of infection through a wound in this region.

TREATMENT.

In the early painful condition affecting the shoulder, resolution may occasionally be brought about by the injection of fibrolysin or of tincture of iodine into the centre of the swelling, and the application of a smart biniodide of mercury blister. In these cases the internal administration of potassium iodide may be of service.

In the diffuse superficial form with multiple suppurating points, treatment is difficult. The writer has had best results by inserting into the centre of each suppurating point a number of times on successive days the end of a grooved director, which had been previously dipped in pure tincture of iodine. In this way the medicament gently gravitates into the lesion, and further injury to the tissue is avoided.

Whenever a botriomycoma is sufficiently well circumscribed to make excision possible, it should be operated upon. The internal administration of potassium iodide has been recommended in many quarters; in fact, this drug has been spoken of almost as a specific, as it is in so many cases of actinomycosis. My experience, however, fails to support that contention, and I think surgical measures should be resorted to as early as possible. It may be that potassium iodide is of service in very early cases, and in those cases where the growths are so large or diffuse as to be inoperable, it may reduce them to an operable condition. The dose I have usually given has been 2 drachms daily or twice daily, and it is just possible that if the dose were doubled better results might be obtained. Fröhner, Malcolm, and others, have met with similar failures; while Thomassen, Seigmund, Ostertag, Hoare, and others, have reported favourably of potassium iodide. In the surgical removal of botriomycomata there is usually considerable hæmorrhage, and it is advisable to practise ligation pretty freely. I have often heard it said that all that is required is to cut off a portion of the growth, thus exposing the deeper cavities, and that the remaining growth will disappear. I strongly disagree with that practice. Although occasionally it may prove successful, it will much more frequently result in recurrence. I can recall a number of instances in which I have had to operate for botriomycoma of the spermatic cord after it had been previously treated by partial excision. There is really very little likelihood of recurrence if the obvious limits of the growth are removed. As a general wound dressing in these cases, I prefer to use solutions of iodine (½ to 1 per cent.).

With regard to botriomycoma of the udder, Fröhner says that in his experience it is a very malignant tumour, and that operative removal is liable to be followed by recurrence. For this reason he recommends total amputation of the udder, even where the disease is confined to one half of it.

NOTE BY EDITOR.

In cases of "collar" tumours of fresh development, in which there is usually a deep-seated circumscribed abscess, it is often sufficient to locate the abscess cavity by inserting a trocar and cannula; then withdraw the trocar, and cut freely into the tumour so as to expose the cavity, using the cannula as a director. After evacuating the pus, curette the cavity, and pack it with tow soaked in tincture of iodine, which may be left *in sitû* for three or four days, after which the dressing is renewed. After a few dressings the tumour often rapidly reduces, and the application of a biniodide of mercury blister completes the cure. Some practitioners fill up the cavity with the biniodide of mercury ointment, and find that this agent is preferable to tincture of iodine; but in tumours of older standing, or in those which tend to recur, excision is the preferable treatment. In many of these cases the tumour is very deep-seated and in the vicinity of important blood-vessels, so that considerable surgical skill is necessary in order to prevent serious hæmorrhage. After the removal of the tumour we always pack the cavity with antiseptic gauze, so as to avoid the risk of secondary hæmorrhage, which is not uncommon after the operation. Similar precautions are necessary after the removal of a scirrhous cord, and we may remark that in a case of this nature when the growth is large, of old standing, and has contracted extensive adhesions, the operation is a very formidable one, in consequence of the extreme vascularity of the parts; indeed, cases of fatal hæmorrhage are on record as the result of this operation. In the case of old horses with extensive scirrhous cords, we have seen good results from the internal administration of large doses of potassium iodide, and the local application of an ointment composed of the same agent with lanolin (1 to 6). We must admit, however, that in some instances there is a tendency for the growth to recur after a period of quiesence; hence, except in inoperable cases, excision is to be advised as the most radical and economical form of treatment. Obviously, the earlier the operation is carried out after the first appearance of the growth the better, as the adhesions are then far less dense and extensive, and hæmorrhage is easily controlled.

We may here remark that "collar" tumours are not always botriomycotic in character.

Botriomycosis in Cattle.—Instances of the disease in this species have been observed by Czoker and Immelman. The former found the udder affected, the lesion consisting of disseminated sclerous foci in the centre of the gland. The latter found that half the gland was converted into a fibrous tumour containing cavities in which the botriomyces were demonstrated. Günther observed botriomycotic tumours in the liver. Reali met with an instance in which there were three tumours—viz., one on the thigh, one in front of the shoulder, and another in the centre of this region. These tumours were irregular, hard, mobile and insensible, and showed fistulæ. A yellow sanious pus was discharged from these fistulæ, containing granules the size of a pin's head.

Botriomycosis in the Pig.—Wilbrandt recorded a case where, following castration, at the extremity of the spermatic cord a fluctuating tumour occurred, which was hard and insensible, and the muscles of the thigh were swollen. In the spermatic cord there was a collection of brown fluid containing small solid bodies in suspension. The muscles of the thigh were converted into a fibrous mass studded with numerous cavities, varying in size from the diameter of a pea to that of a bean. These cavities contained a granular greyish material, associated in some cases with a brown liquid. A mass of botriomyces was found in the inflamed connective tissue (Nocard and Leclainche). Czoker also recorded a case of scirrhous cord in a pig, associated with interstitial myositis of the muscles of the thigh, and due to botriomyces. Botriomycoma of the udder has also been observed in the sow.

Botriomycosis in Man.—According to Poncet and Dor (French Surgical Congress, 1897), cases of botriomycotic tumours have been observed in man, affecting the fingers, thorax, and elbows. Siethof observed conjunctivitis palpebrarum accompanied by nodular formations, in a man who had charge of a horse suffering from fistula of the spermatic cord. Other cases are recorded by several observers (Friedberger and Fröhner).

BACILLARY NECROSIS

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Synonym.—Necrobacillosis.

GENERAL REMARKS AND ETIOLOGY.

The recognition and description of the bacillus of necrosis is generally attributed to Löffler, who in 1884 investigated the condition known as "calf diphtheria," and discovered the bacillus to be the cause of that disease. The bacillus has since been shown to be responsible for a large number of morbid conditions in the domestic animals, all of which appear to be susceptible to natural infection. The affections to which it gives rise occur sporadically for the most part, but they occasionally assume clearly-defined contagious characters.

The bacillus may be demonstrated in the affected tissues by staining sections with any of the basic aniline dyes, but it does not retain the stain well, and is easily decolorised in making preparations for microscopic examination. It does not stain by Gram's method. The organism is always found in greatest numbers towards the periphery of the lesions, where it occurs in the form of long wavy filaments, 80 to 100 μ in length, and 0·75 to 1·5 μ in thickness. The filaments are irregularly segmented, intricately interwoven, and frequently grouped into bundles. A bacillary form is sometimes encountered, the rods showing great variations in length.

The bacillus is a strict anaërobe and, so far as is known, non-sporulating, its existence apart from the living host being assured by its saprophytic tendencies. It has a wide and almost ubiquitous distribution in Nature, being constantly present in soil, especially on cultivated land. Bang has demonstrated it in the cæcum of healthy pigs, and it is frequently, probably constantly, present in the alimentary canal of healthy herbivora. This wide distribution of the organism is particularly favourable to natural infection, which may take place by ingestion or by means of wounds, excoriations, or abrasions. Previous injury or the presence of pyogenic organisms apparently assist the bacillus in establishing itself in the tissues, where it produces a marked change in the original injury, transforming an otherwise simple lesion into a grave and possibly serious one. Bacillary necrosis is also met with as a complication in other diseases, such as calf septicæmia and swine fever (Bang); but, whilst secondary infection is quite common, the bacillus under certain circumstances establishes itself in the living tissues independently of previous infection, and appears to be solely responsible for the morbid condition produced.

The diseases caused by the bacillus of necrosis show a decided tendency to remain localised, the organism remaining confined to the seat of infection, but it has been shown that metastasis may occur, with secondary lesions in the lungs, pleura, or liver. Though transported by the blood in these cases, the organism cannot be demonstrated in the blood.

The lesions for which the bacillus is responsible possess one feature in common—necrosis—but their appearances and the associated symptoms present considerable variations, according to the organs or tissues attacked and the presence or absence of other organisms. They are for the most part persistent in character, and show comparatively little tendency to spontaneous recovery, but rather to a progressive enlargement by invasion of adjacent tissues. Speaking very generally, it may be said that bacillary necrosis affecting the skin and superficial tissues is of little gravity so far as the life and general health of the patient are concerned, whilst invasion of the mucous membranes, if at all extensive, or of the internal organs, gives rise to symptoms which frequently end fatally.

The following are the more important morbid conditions met with in animals for which the bacillus of necrosis has been shown to be responsible:

In the horse, certain cases of poll-evil, fistulous withers, quittor, cracked heels and other sloughing processes in the digital region, ulceration of the intestine (cæcum and colon). McFadyean and Nielsen have described multiple foci of necrosis in the lungs and accompanying pleurisy as a consequence of metastasis from primary lesions in the limbs. It would not be justifiable to assert that all cases of poll-evil, fistulous withers, quittor, and similar chronic suppurating conditions associated with necrosis of some of the deeper tissues met with in the horse, are caused by the bacillus of necrosis, but it has been placed beyond all reasonable doubt that this bacillus plays an important part in many chronic suppurating processes which previously were attributed solely to the action of pyogenic organisms.

In cattle the bacillus has been shown to be the cause of certain diphtheritic lesions of the buccal (calf diphtheria), gastric, intestinal, respiratory, and genital mucous membranes, and for necrotic processes affecting the digits ("foul in the foot"), tail, and teats. It is responsible for the multiple lesions of necrosis affecting the livers of cattle and sheep, for certain cases of navel-ill in calves and lambs, for necrosis of the skin of the lips and face and for diphtheritic ulceration of the mouth, stomach, and intestines of sheep. In the pig it has been described in diphtheritic lesions on the mucous membranes of the mouth and nose, and Bang considered it responsible for the deeper lesions of the intestine in swine fever and for necrotic areas of the lungs met with in the same disease.

It must not be supposed that the morbid conditions referred to exhaust the list of diseases in which the bacillus has been found, and in whose evolution and termination it plays an important part. They serve to show the varied situation and character of the lesions, and it is more than probable that the bacillus will be shown to be responsible for other necrotic processes occurring in animals, but in which as yet no connection with the organism has been demonstrated.

SYMPTOMS AND MORBID ANATOMY.

Affecting the mucous membranes, the lesions of bacillary necrosis present the following characters: In their earliest stages they are superficial, of a light grey colour, and are easily detached, leaving a red sore, though the natural tendency to separation is never great. More frequently the patches enlarge rapidly, extending into the deeper parts of the membrane as well as superficially, and by the coalescence of adjacent patches large areas become affected. The older lesions are of a yellowish-grey colour, firmly adherent, and the surrounding mucous membrane is swollen, red, and congested. Situated in the mouth and pharynx, such lesions give rise to profuse salivation and interference with mastication and deglutition. Affecting the respiratory mucous membrane, a cough is induced, and pieces of the necrotic tissues may be ejected, or they may be found in the fœtid discharges which escape from the nostrils. A sniffling or roaring noise indicates obstruction to the free passage of air, and broncho-pneumonia is liable to complicate the original condition. A fœtid discharge from the vulva leads to the examination of the genital passages and the detection of the lesions.

Bacillary Necrosis in Calves.—In connection with the membranous lesions, the condition first described by Dammann in 1877 under the name of "calf diphtheria" requires special mention. The terms "ulcerative" and "malignant stomatitis" of calves are also applied to the same condition. Dammann employed the term "calf diphtheria" because he believed the condition to be identical with human diphtheria; but Löffler, in the course of his investigations into the cause of human diphtheria in 1883 and 1884, devoted some attention to this condition of the calf, and demonstrated the bacillus of necrosis in the lesions. In 1893 Bang fully confirmed Löffler's conclusions, and the bacillus is now universally accepted as the cause of the affection.

The malady, which assumes definitely contagious characters, affects calves, and is confined almost entirely to those under six weeks of age. Dammann's excellent description of the clinical characters of the disease can hardly be improved upon. The affected calves lose their spirits, cease to suck, salivate profusely, and show much difficulty in swallowing. Opening of the mouth is painful; the tongue is swollen, and its movements limited. The animals are fevered, and diarrhœa frequently supervenes. Grey patches of variable dimensions, which are easily detached, cover the mucous membrane of the tongue, cheeks, palate, and fauces. They rapidly increase in thickness, forming a thick grey layer on the swollen and inflamed underlying structures, and the necrosis may extend to the subjacent bones. The cheeks, submaxillary space, and throat, are much swollen. In some cases similar lesions appear on the mucous membrane of the nose, and may extend to the larynx and trachea, giving rise to a cough and to noisy or dyspnœic respirations. Digital lesions are sometimes present, and the hoofs may be cast. The affection is very grave. A number of the calves die in the course of a few days, apparently from acute intoxication, but the majority survive for two or three weeks, complications in the form of pneumonia or enteritis usually preceding death.

The disease may be transmitted experimentally from calf to calf, and under natural conditions infection appears to be communicated by cohabitation, feeding-pails, and attendants, or by placing healthy calves in infected pens, in which the virus may retain its vitality for several months. The discharges which escape from the mouth and nose are virulent, and the fæces are also frequently infective as a consequence of the animals swallowing detached false membranes, or of the extension of the infection to the gastric and intestinal mucous membranes.

On a post-mortem of the affected calves the lesions already described may be found on the mucous membrane throughout the alimentary tract; while in the lungs, and with less frequency in the liver, necrotic areas similar to those which will be referred to shortly are of common occurrence.

Mettam has described a condition affecting Irish calves which bears a very close resemblance to this affection, and which is said to be quite common in certain parts of Munster. Young sucking calves were affected, and showed extensive necrosis of the buccal mucous membrane, with profuse salivation and loosening or falling of the teeth. Several calves were affected on the same premises, and in the mouth lesions the bacillus of necrosis was demonstrated. Moussu states that he has never seen the disease apart from omphalitis.

When the bacillus of necrosis attacks the skin and subcutaneous tissues, the affected parts and those immediately adjacent are somewhat swollen and acutely painful in the earliest stages. Some fever and general disturbance of health are commonly observed, and in the limbs an acute lameness. In a very short time the affected parts become cold and insensible, a line of demarcation appears, and the process of separation begins, accompanied by the escape of a profuse, fœtid, purulent discharge from the gap between the living and dead tissues. The elimination of the dead tissues takes place rapidly. It is usually well advanced in two or three days after the first appearance of the attack, and with its commencement the acute pain, lameness, and fever, if previously present, subside. The slough which separates varies considerably in superficial extent and depth, but, provided the elimination of the dead tissues is complete, the resulting wound heals as a rule without difficulty. Cartilage, ligament, tendon, or bone, may, however, have been attacked by the bacillus. They may be exposed when the more superficial tissues separate, and in them the bacillus may persist indefinitely. The wound then fills in part with granulation tissue, but a sinus remains, from which a purulent discharge continually escapes. The necrotic tissues in the deeper parts are constantly bathed in pus, the process extends, though more slowly than in the superficial tissues, and natural delimitation, if it occurs at all, is very slow in taking place.

Internal lesions of the liver and lung are only recognised on post-mortem examination. In the liver the more superficially situated lesions appear as raised nodules on the surface of the organ, which is usually much enlarged. There may be only one or two present, or a large number scattered irregularly throughout the liver. On incision, the affected areas are more or less spherical in outline, with irregular margins. They vary in size up to that of a walnut, and stand out clearly from the surrounding liver tissue. They are firm in consistence, and dirty brownish or greyish yellow in colour, the centre of the lesion being frequently of a lighter colour than the peripheral parts. In the more recently affected areas evidence of lobulation like that of the normal liver may be seen, but these soon disappear; and if the animal lives for any time the lesions undergo a process of softening, and are transformed into a purulent-like material of a dirty brownish-green colour, surrounded by a more or less definite capsule of newly-formed fibrous tissue.

In the lung the lesions present a similar appearance. They are rounded, clearly defined, greyish-yellow in colour, and firm in consistence. They may attain a much larger size than the liver lesions, and their number is variable. The older lesions undergo softening, and are often surrounded by a fibrous wall. In a few cases a pleurisy, for which the bacillus is also responsible, is found in association with the lung lesions, in which case the pleural cavities contain a turbid, yellowish, non-putrid fluid, with coagula floating in it and adherent to the surface of the pleura.

TREATMENT.

The internal lesions of bacillary necrosis are not amenable to treatment. The treatment of the external lesions is largely surgical. It consists of the removal of all necrotic ligament, cartilage, bone, or other tissues, and careful disinfection. It is advisable to curette the wound left after removal, especially in the case of old-standing fistulæ, since in many cases the bacillus has invaded tissues adjacent to those obviously affected, but which do not yet show marked or recognisable naked-eye change.

Similarly, the affected areas of mucous membrane should be removed when accessible, and suitable antiseptics applied. Lotions of alum, boric acid, chlorate of potassium in glycerine and water, or silver nitrate, are appropriate, and should, when practicable, be accompanied by frequent lavage of the affected membranes with dilute non-irritant antiseptics. The most satisfactory results will be obtained by early intervention before the necrosis has extended to the deeper parts of the membrane. In the form to which the name " calf diphtheria " is applied, Dammann recommended the application of 10 per cent. solutions of salicylic acid or 2 per cent. carbolic acid, after careful removal of the false membranes, together with the internal administration of ½ to 1 drachm of salicylic acid daily. The removal of the false membranes should always be accompanied by a minimum amount of injury to the underlying healthy structures. The strength of the calves must also be maintained by the administration of gruels, milk, and other nutrients, with stimulants and astringents if required. The affected calves should be completely segregated, and the sanitary arrangements of the calf-pens occupied by the healthy as well as by the sick calves should be placed on a satisfactory footing.

Obviously, infection of wounds will be prevented by the use of antiseptics and of appropriate protective dressings. Attention should be especially directed to wounds in those regions particularly exposed to infection, such as the lower portions of the limbs and the ventral aspects of the body. In the contagious affection of the calf the affected animals must be completely isolated, and calf-pens, feeding-pails, etc., subjected to a thorough process of disinfection. The opportunities of infection will be materially lessened by a general improvement in the cleanliness of calves and in the housing accommodation provided for them, which, as a general rule, leaves much to be desired from a hygienic point of view. It is beyond question that the umbilicus is, in a proportion of cases at least, the port of entry of the bacillus into the system, and with a view of preventing this and other equally serious infections, the umbilicus of all newly-born animals should receive careful attention.

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I. BACILLARY NECROSIS IN THE HORSE.

By HENRY GRAY, M.R.C.V.S.

Synonym.—Necro-bacillosis in the horse.

General Remarks.—This is a very common infection in the horse, and may occur as an enzoötic in large establishments, especially where the heavy breeds of animals are kept. It appears under certain conditions to be very contagious, being transferred from one horse to another. It is chiefly seen during the colder months of the year, more particularly when the weather is wet and cold, and when there is a great deal of mud on the roads or salt has been used to dissolve the snow. Injuries to the lower region of the limbs, especially treads caused by sharp calkins, or nails puncturing the lower surface of the feet, are often followed by necrosis of the damaged parts. The mortality is very great, and this usually arises from translation of the infection from the limbs to the lungs and other parts, or from the necessary destruction of the animal in consequence of the incurable nature of the lesions produced in the limbs.

History.—This infection, as regards modern pathology, was, in the horse, first studied by Jensen in 1895, then by Ernst, and Nielsen in 1897, McFadyean in 1900, Nocard and Leclainche in 1903, and Cuillé in 1905.

Distribution in the Animal System.—The bacillus of necrosis occurs in the pseudo-diphtheritic lesions of the croupous colitis and typhlitis of the horse, in treads, cracked heels, gangrene of the skin covering the coronary region, punctured wounds of the feet, lacerated and punctured wounds of the lower region of the limbs, lacerated or pocketed knees, and also from too active blisters or too deep firing. It may also be associated with poll-evil, fistulous withers, and, in fact, any condition in which the tendinous and fibrous or cartilaginous tissue is undergoing necrosis.

The microbe is widely distributed in Nature, and is also a normal parasite of the intestinal canal.

Symptoms.—In many instances the disease is confined to the skin covering the coronary band. In such cases it may occur as a veritable enzoötic, and is capable of being conveyed from horse to horse, especially when the coronary region is acted upon by constant wet and dirt and a low temperature. Salting the roads increases this liability. This form is mostly seen in the lighter breed of horses, especially carriage and saddle horses. In a great number of such cases the skin is found to be necrotic on the first appearance of lameness. The necrotic patch is usually circumscribed, varying in size from a two-shilling to a five-shilling piece; it is cold, and when touched seems to cause pain to the animal; it is bluish in appearance, and gives off a fœtid odour. After twenty-four hours or so it undergoes a separation from the living tissue, and if the slough is removed as soon as it has become loosened, a superficial septic wound, discharging very little fœtid greenish pus, remains. If the wound be promptly treated with some strong astringent antiseptic solution, the offensive discharge and smell are removed, and the wound commences to heal without any further complication. Such wounds as these are not serious, but if negligently treated from the first, the necrotic process is liable to extend into the deeper structures of the foot and set up serious complications, often leading to irreparable damage to the organ, or even to metastasis and death. When these superficial cases are brought to our notice at the onset we generally find the affected part swollen, painful to the touch, purplish in appearance, and discharging a slight amount of thin serous material.

Similar lesions to these arise from treads, which are generally more serious in consequence of the deeper parts involved, such lesions increasing in gravity as the central part of the front of the coronary circle is approached. Again, the necrotic lesions arising in cracked heels are similar in character, but more linear in outline, those in the centre and high in the hollow of the heel being more liable to be followed by the process spreading upwards and inwards, and often by a fatal metastasis.

Punctured wounds of the lower surface of the foot, either by nails or other foreign bodies, are not rarely followed by bacillary necrosis, even after the animal has been at work for a week or more following the injury without showing lameness. If there should be extensive sloughing of the hollow of the heels, or all round the coronary band, metastasis may supervene and cause death.

Any of these forms may also be followed by sloughing of the skin, either in patches varying from the size of half a crown, to that of the palm of the hand, or even involving all the skin below the knee. Sometimes there is accompanying this necrosis an inflammatory softening of the tendons and ligaments, with an attendant attempt at repair of the other tissues by a granulatory process. If such cases are not destroyed, they may progress until the tendons rupture, and even lead to dislocation of one of the joints in the vicinity of the lesions. Similar conditions may be encountered in pocketed or lacerated lesions of the front of the knee.

In some cases there may be no external swelling of the limb, but the process, which is also probably complicated with streptococcic or staphylococcic or both infections, works its way upwards in the skin and even in the fascia, so that when a post-mortem examination is made the whole of the external surface of the muscles of the limb is bathed with a pinkish, creamy pus. In such a case there is high fever, profuse perspiration, accelerated breathing, great restlessness bordering on delirium, and rapid emaciation, the lameness even disappearing when widespread infection has set in.

In some instances the limb may appear swollen, showing here and there an abscess. In others, though swollen, the limb exhibits patches of necrosis, which may extend to the tissues immediately surrounding the hock-joint or even to the elbow-joint. An œdematous swelling may extend to the prepuce or even to the under surface of the abdomen.

In the case of the general circulation becoming infected, it is usually announced by the sudden increase in severity of the lameness; an animal with a hitherto slight cracked heel or other lesion, undergoing an apparently favourable reparative process, is found holding the limb off the ground. There is a high temperature, the appetite is lost, the expression is anxious, and we generally observe a profuse and continuous perspiration, a rapid pulse, accelerated breathing, and extreme weakness. If the animal live long enough, the emaciation becomes profound. In consequence of the restlessness, even sometimes bordering on delirium, it is not always easy to auscultate the chest in order to discover the presence of pneumonia or pleurisy.

Lesions.—There may not be many lesions observed in the foot beyond a black or purplish necrotic area of small dimensions in the fibrous tissue, especially in the vicinity of the navicular region, even in those cases terminating fatally from metastatic lesions in the lung. In other instances the affected part or even the whole limb and under surface of the abdomen and sheath may be extensively swollen. In the centre, or at the commissures of the heel, or around the coronet, especially over the region of the lateral cartilage, raw granulating surfaces, varying in size from a shilling or half a crown to that of the palm of the hand, are found, after the necrotic skin and areolar tissue have sloughed away. In some instances following an injury to the foot, the coronary ring or the tissues in the heel may be swollen and show one or more large fistulæ, discharging a dirty greenish fœtid pus and necrotic pieces of tissue, and the pedal-joint may also be involved in the necrotic process. In others the coronary region is extremely swollen, a deep separation exists between the horny wall and the contained structures, leaving a rather wide furrow all round the coronet, resembling somewhat that appearance of the foot affected with gangrene after neurectomy. The internal structures, such as the fatty cushion, the tendons, and other similar tissues, are found swollen, highly discoloured, and undergoing necrosis. The skin over the pastern and fetlock regions, or at one or the other, or both, sides of the metatarsal, or more rarely the metacarpal, region, shows more or less extensive patchy or total loss of skin, the subjacent soft tissues and exposed tendons being swollen, soft, and often covered with yellowish or reddish granulations of various sizes, which, on being touched during life, bleed. In the swollen tissues, either below or above the hock or knee, which have not undergone complete necrotic changes, we may here and there find one or more abscesses. In rarer cases we do not find, on external inspection, much infiltration of the dermis or subdermis, but on removing the skin a layer of pinkish, creamy purulent matter, extending from the coronet to above the stifle, is discovered, the only external lesion observed during life being a pinkish granular surface, on a fair way towards reparation, in the heel.

The pleural cavity nearly always contains a more or less quantity of clear or turbid fluid, which may be of a yellowish, greenish, or reddish appearance, containing now and again coagula, and occasionally giving off a fœtid odour. The pleural covering of the internal walls of the chest, lungs, mediastinum, and diaphragm, is injected, and occasionally coated with patches of yellowish, fibrinous lymph. The lungs are usually hepatised, especially at the anterior portion, and contain several small abscesses or necrotic areas, from the size of a pea to that of an orange. In some cases there are small hæmorrhagic points and greyish areas on a pale reddish groundwork of a section of the condensed lung.

The pericardium sometimes contains a quantity of effusion of a more or less discoloured appearance.

The peritoneal cavity often contains a quantity of pinkish or reddish serous fluid.

The intestines and stomach may show at times a few stained patches on their peritoneal surface.

The liver is mostly enlarged, and has a washed appearance. The spleen appears normal. The kidneys are often congested.

Diagnosis.—This is not difficult when one takes into account that necrotic lesions or granular surfaces resulting from gangrenous sloughs are usually due to the bacillus of necrosis, which may be detected in the lesions or pus, in the form of long filaments in a more or less state of purity, or often mixed with staphylococci or streptococci. These filaments measure 80 μ to 100 μ, having thick or tapering extremities.

The bacilli also appear as rods of various lengths, and from $\frac{3}{4}$ μ to 1 μ in thickness; they may even be so short as to resemble cocci.

They are stained with ordinary aniline colours, but decolorised with Gram. They take the colouring material with more or less facility, according to origin. If they are derived from a recent abscess or a tissue undergoing necrosis, the staining is intense and regular. On the contrary, if the lesions to be examined are of some standing, the bacilli are stained with difficulty, and always show numerous clear spaces. Pure cultures of this organism are readily obtained by inoculating a rabbit with impure products, which often contain staphylococci and streptococci. In this animal small lesions soon form in the lung and liver, which often give pure cultures. The inoculations should preferably be made with a piece of necrotic tissue just undergoing necrosis inserted in a pocket made under the skin on the back of the rabbit.

When pulmonary symptoms arise during the course of a necrotic process in tissues of the feet, limbs, on the poll or withers, as in pricks, treads, gangrenous heels or coronets, poll-evil, fistulous withers, and such-like, one may fairly conclude that metastasis has taken place by the transmission of the bacillus of necrosis through the circulation or lymph channels to the lungs.

Prognosis.—When the joints are involved, or the cutaneous surface of the limbs is widely destroyed, or the tendons have undergone inflammatory softening or necrotic destruction to such an extent as to cause them to rupture, the animal will be unfitted for further service, and should therefore be destroyed. In the case of pulmonary metastasis death usually supervenes. When the necrosis is confined to small areas on the limbs, and does not extend into any important part, as the pedal, navicular, or other joint, recovery usually takes place. A slow healing, granular surface in the centre of the heel should be looked upon with a certain amount of suspicion in consequence of the frequency with which serious complications occur as the result of such lesions.

Treatment.—Any wound on the coronet or in the dangerous zone of the frog should be treated as antiseptically as possible, avoiding foot-baths and poultices, because they are very liable to favour necrosis. For those wounds on the coronets tincture of iodine is the best application.

The gangrenous patches on the coronets should be removed as early as possible, and the resulting wounds dressed with a strong solution of zinc chloride, so as to prevent the spread of the process and to render the part as aseptic as possible. After this latter object has been attained, the ordinary treatment for such lesions may be adopted.

Cracked heels should be prevented as much as possible by not clipping the hair off the limbs, and not washing with warm water and soft soap during the colder months of the year. When they arise from unpreventable causes, such as wet, chilly weather and muddy roads, the heels should be bandaged up with cotton-wool after they have been sponged out with a dilute solution of liquor plumbi subacet.

If necrosis of the heel has taken place, the reparative process of the resulting wound should be hastened by the application of biniodide of mercury ointment direct to the granulating surface. This should be alternated with zinc oxide ointment, maintained by a layer of cotton-wool and a bandage.

The application of severe blisters should be avoided in wet, chilly weather, if the animal is to be exposed, as on a grass field, as under such conditions the parts to which they are applied are liable to necrotic infection, which brings about sloughing of the tissues and sometimes the death of the animal.

In large establishments, especially those in which the heavy breed of horse is maintained, loose-boxes or stalls in which wound cases are usually placed should be thoroughly cleansed and disinfected, as recent wounds often become infected from surroundings contaminated by a previous case of the disease, and thus the malady becomes enzoötic.

It should not be forgotten that cracked heels may also be conveyed from horse to horse by the discharges from the heels.

Drainage should always be afforded in tendinous or bursal wounds, such as pocketed broken knees, suppurating poll-evil, fistulous withers, etc., even if the necrotic tissues should not be excised.

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II. BACILLARY NECROSIS IN THE DOG.

BY HENRY GRAY, M.R.C.V.S.

Synonyms.—Necro-bacillosis ; Fistulous dermatitis.

French : Dermatite phlegmoneuse et fistuleuse ; Dermatite chronique phlegmoneuse.

Definition.—A chronic phlegmonous disease of the skin, characterised by multiple abscesses, which burst and leave fistulæ, discharging a bloody serous fluid containing the bacillus of necrosis.

History.—This disease has been frequently observed, but rarely described. Attention has been called to it by Cadiot and Almy, Cadiot and Brétin, and the writer ; but the credit of pointing out its true cause and nature is due to Cuillé, who has given a very good description of it. Livesey has also recently alluded to it, but as a new disease hitherto undescribed.

Animals Affected.—It is mostly seen in the larger lymphatic breeds and in sporting dogs, and occasionally in other breeds and also in the cat. It preferably attacks adult or aged animals.

Seats of Election.—It attacks, by preference, the tail, the circum-anal skin, the feet, hock, the outer surface of the regions of the stifle, forearm and elbow, sheath, nose, and lips. In the cat, the feet and tail are attacked. In the monkey it generally affects the tail.

Etiology.—The infection gains an entrance to the tissues by inoculation, resulting from wounds or abrasions, which are frequently caused by sharp-pointed or cutting bodies, and occasionally by the *Demodex folliculorum.* In the region of the feet it enters through wounds set up by thorns, stubble, barley-grass, or other plants, and by injuries.

It is generally accompanied by other microbic infections, such as those due to streptococci and staphylococci. It may also complicate distemper and lesions of the mouth and cheeks.

Symptoms.—The disease usually commences by some swelling of the skin, which is of a purplish colour and becomes hairless. After a short period small abscesses, varying in size from a small pea to a walnut, or even to a fowl's egg, form in the subcutaneous connective tissue, and burst, and then discharge a dirty, bloody purulent fluid. The resulting wounds do not heal, but leave deep fistulous channels, which persistently discharge a thick, bloody purulent material mixed with fragments of necrotic tissue, which dries up and adheres to the surrounding skin. The skin remains hairless and very much thickened, and the fistulous openings often appear as if the skin had been punched out, giving the affected portions of the structure the appearance of a strainer or a colander. The lesions rarely extend, but usually remain stationary, without showing any inclination to heal. However, the writer* has seen many cases where the lesions have become generalised, and when the abscesses have burst or have been surgically opened, the skin has had a lacerated appearance. Cadiot and Almy have observed a case in which the disease extended from the sheath to the groin, and then to the whole of the right thigh, leading to a fatal peritonitis.

When the disease commences in the feet there is lameness, and the affected limb is carried off the ground. In the case of two or more, or even all the feet being attacked, the animal generally assumes the recumbent position, and is with great difficulty made to move. The spaces between the toes become swollen, red, and then purplish, and when touched are very painful. The skin oozes, and then several small abscesses form, burst, and discharge a bloody purulent fluid. The resulting wounds become fistulous, and discharge a thin, bloody serous fluid. They have no tendency to heal, but they often close up to break out again. After a time the swelling travels up the limb, to reach the carpus or hock, or even to extend to the groin or axilla. Along the course of this swelling small abscesses appear, which burst, and afterwards pursue a course similar to that occurring on the lower region of the limb. The chain of small abscesses, after they have burst, communicate with one another by a small channel, so that if a small probe be passed into one it will easily travel the whole length of the channel. In the digital region the probe can be passed in a similar manner for several inches, or even its whole length.

The disease has no tendency to disappear spontaneously, but will, run a long course, unless active steps be taken to combat it. It does not. as a rule, cause much disturbance of the general health of the animal, but when it becomes generalised it may produce emaciation and even death from exhaustion. In some rare cases the internal organs become secondarily invaded, leading to death, which may sometimes be quite sudden. On examination of such cases lesions have been found in various parts of the body, notably the brain, cardiac valves, lung, liver, and kidney.

Treatment.—When the disease is of recent origin and confined to a circumscribed patch, it may be advisable to excise the whole of the affected part; but when the fistulæ are deeply situated and the surrounding tissues much thickened and necrosed, the fistulæ should be slit open and the tissues curetted, then deeply cauterised with nitrate of silver or chloride of zinc, and ultimately painted from time to time with strong tincture of iodine. When the fistulæ are confined to the lower part of the limbs, they should be laid open and irrigated with strong solutions of chloride of zinc, sulphate of copper, bichloride of mercury, or peroxide of oxygen, or painted with strong tincture of iodine or pure creolin, or cauterised with nitrate of silver. All necrotic lesions should be removed with the curette. Bier's hyperæmic method overcomes many rebellious cases.

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* Hoare's "Veterinary Therapeutics," pp. 675, 697 (1906 Ed.).

For the treatment of tail lesions similar treatment should be adopted as for the limbs, care being taken to protect that organ from further injury. As a rule, however, these cases are so rebellious that the majority of practitioners amputate the tail. Death has occurred after this operation, when extensive internal lesions were present, discovered only on post-mortem examination.

The animal should not be allowed to gnaw the affected parts, as by this means infection may be carried to other regions of the body.

In the *cat* the majority of cases succumb from acute septicæmia, which results from the extensive necrotic lesions in the foot and limb. As a rule such cases are only brought to the practitioner's notice when the disease is very far advanced.

Public Health.—The bacillus of necrosis has been found in false diphtheria in the child; also in rebellious wounds in man. It is therefore advisable to treat infections in animals due to this bacillus with caution.

This bacillus has also been found in the exudate of pseudo-membranous diphtheria of the fowl.

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III. BACILLARY NECROSIS IN THE RABBIT.

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Schmorl's disease ; Necro-bacillosis.

French : Nécrobacillose du lapin ; Maladie de Schmorl ; Gangrène cutanée du lapin.

Definition.—A contagious disease of the rabbit, characterised by a progressive necrosis of the skin of the head or by circumscribed suppurations of the skin of the thighs, flanks, or abdomen, occurring as an enzoötic or epizoötic, and due to the bacillus of necrosis.

History.—This disease was described by the late Pierre Mégnin, who denominated it as an " Affection vermineuse de l'estomac du lapin de garenne, avec éruption reflexe de la face," as it was always, in his experience, associated with the presence of a great number of the *Strongylus strigosus* in the stomach and intestines. It has also been studied by Schmorl of Leipzig in 1891 ; by Mazzanti of Pisa in 1892 ; by Leclainche and Vallée in Toulouse in 1900 ; by Leclainche in Gers in 1901 ; and by Cuillé of Toulouse in 1905.

Animals Attacked.—It occurs in tame rabbits kept in confinement, as well as the wild rabbit at liberty in plantations or natural warrens. It also occurs as a spontaneous disease in the wild rat and in many wild animals kept in confinement.

Etiology.—This disease is due to the bacillus of necrosis, which gains an entrance to the system by abrasions, punctures, or wounds, especially those produced by shots, or by ingestion of hard or prickly food soiled with the saliva, discharges, or detached sloughs. It is also very probable that certain intestinal worms, as the *Strongylus strigosus*, may wound the gastro-intestinal mucous membrane, and so permit the bacillus to gain an entrance to the system. It is prevalent during the winter months of the year, and appears to be very contagious.

Period of Incubation.—Two to fourteen days.

Symptoms.—The disease manifests itself in various ways, according to the method by which the bacillus obtains an entrance into the system. For practical consideration it is advisable to subdivide them into three principal types :

Type 1.—This commences by swelling of the upper lip, nostril, and eyelids, until ultimately the whole of the skin of the face, and often the lower lip, is involved. The skin becomes at first of a purplish colour, hot and painful, and covered with pustules; after a time it assumes a brownish-black appearance, is cold and insensible. The necrotic patch gradually separates until it sloughs off, taking with it the upper lip and a part of the nose, and sometimes the lower lip, and thus leaving the incisor teeth exposed and the nasal cavities with wide openings. The resulting wound is dry, scarcely granular, shows little or no suppuration, and is surrounded by a hard, thick lardaceous tissue. In some cases a similar necrotic ulcerative process may also affect the hind-limbs and the vicinity of the external generative organs. In this condition the affected animals are almost, if not quite, unable to feed themselves, and in consequence die from starvation.

Type 2 (*Schmorl's Disease*).—This begins by the lower lip becoming swollen, purplish, and very painful. This process then gradually spreads to the under surface of the jaw and throat, to gain within a week or so the front of the chest. Constitutional symptoms do not appear before the fifth day, when pain is caused by movements of the jaw and by attempts to swallow, and the animal becomes drowsy and disinclined to move out of the corner of its hutch. Emaciation becomes profound, the breathing short and difficult, the temperature rises two or three degrees, and the animal dies in a profound comatose state. Occasionally the cheeks, floor of the abdomen, and both eyes, become involved.

Type 3.—In this form several small abscesses, varying in size from a pea to a walnut, appear on the thighs, flanks, abdomen, and other parts. The abscesses differ from those in the dog, in which the capsule is very thin, so that they rapidly become ripe, and open and discharge a bloody, purulent fluid. In the rabbit, on the contrary, the capsule is formed of a dense fibrous tissue, which has very little inclination to yield and allow the contents of the abscess to escape. They are very slow in developing, and when opened contain a thick, creamy, homogeneous white or bright green pus. Emaciation is extreme, and ends in death. The disease runs its course in from two to four weeks.

Post-Mortem Lesions.—Beyond the external lesions observed during life, we may find the glands in the vicinity of such lesions swollen and succulent. The lungs may be œdematous and contain patches of broncho-pneumonia or caseous pneumonia; the pleural and perhaps pericardial cavities may contain a bloody, turbid fluid, and the various serous membranes show ecchymotic markings. Abscesses may be found in various organs.

Prophylaxis.—This is out of the question in the case of wild rabbits. In tame rabbits the diseased should be destroyed, and the hutches thoroughly scraped, scrubbed, disinfected, and limewashed.

CONTAGIOUS PUSTULAR DERMATITIS OF SHEEP

BY E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Malignant aphtha ; Contagious dermatitis ; " Orf "; Lip and Leg ulceration of sheep ; Crusta labialis ; Ulcerative stomatitis ; Red foot ; Contagious ecthyma ; " Hair and hoof "; " Mouth and foot "; " Carbuncle of the coronary band "; Necrobacillosis of the sheep.

French : La maladie ulcéreuse des lèvres et des pattes du mouton ; Mal de bouche.

GENERAL REMARKS.

This malady has been described by various observers in Great Britain, France, Germany, Hungary, and the United States of America. The variety of synonyms must be ascribed to the different locations in which the disease may appear in the body. Thus, in some instances the lips and the limbs may be the seat of the lesions, in others the feet are attacked, in others, again, the buccal mucous membrane shows small typical ulcers. A venereal form of the disease is also described by Flook, McFadyean, and Williams.

In every form of the malady the essential cause is now believed to be the bacillus of necrosis.

Walley, in 1888,* described the disease under the heading of " Contagious Dermatitis," or " Orf," and stated that it was frequently met with in the Northern Border Counties of Scotland, and was known as " hair and hoof " when it affected the coronary structures, and as "mouth and foot " when the skin of the lips, face, and interdigital spaces was involved. His attention was drawn to the disease by Robertson, who termed it " carbuncle of the coronary band," and found that it was of common occurrence amongst sheep in Roxburghshire and the neighbouring counties.

Walley's first experience with the disease was in lambs feeding on clover ley during the autumn. He found that it was most frequently met with in sheep grazing on old pastures, and that both adults and lambs were subject to the affection. The hind-limbs were more frequently attacked than the fore, and the disease was generally conveyed to the skin of the face by the animals rubbing the mouth against the affected limbs. Not rarely the lips and feet were attacked simultaneously. Experiments to decide the contagious nature of the disease were carried out, first by scraping off a quantity of the discharge from the skin of an affected limb, placing it on cotton-wool, and securing it in contact with the unbroken surface of the interdigital skin of a healthy sheep. In about four days the characteristic lesions of the disease were produced.

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* *Journal of Comparative Pathology and Therapeutics*, vol. iii.

In other instances the discharge was applied to a breach of the surface on the skin of the coronet, and the lesions appeared on the third day, but were better marked than by the first method.

The symptoms of the disease were a varying degree of fever, which, when pronounced, induced emaciation. In the early stages the skin lesions were circumscribed, and consisted of a local inflammation, involving the skin and to some extent the subcutaneous tissue. The regions usually affected were the hind-limb, the coronet, and the lips. Generally in the two latter regions the skin was swollen, hot, and tender, and in parts devoid of pigment redness could be detected. After a few days a solution of continuity of the skin surface occurred, accompanied by an exudation of serum. A sore quickly formed, and tended to spread to any part devoid of wool with which it came in contact. In the limb the disease extended, so that the whole of its lower portion became involved, either by peripheral extension or by coalescence of discrete centres of infection. Later on the cutaneous papillæ were hypertrophied and congested, and bled easily on being scraped. A dirty grey purulent, fœtid fluid was constantly exuded, which collected on the surface of the sore, producing a condition that resembled " grease " or canker in the horse.

Small sores were also present on the skin of the leg and arm. These were covered with dry crusts. The lesions on the lips also showed these characters.

Diseases of a similar nature appear to have been known from an early period. Youatt (1837) described aphtha or thrush in the sheep, characterised by the presence of several small vesicles in the mouth, and mentioned a curious coincidence—viz., that aphtha was frequently coexistent with foot-rot, especially when the latter had run on to ulceration and fœtid discharge. Youatt drew attention to an epidemic described by Paulet that occurred amongst sheep in France in 1745. Small vesicles appeared about the nostrils and lips and various parts of the head, and the inflammation rapidly extended to the lungs by way of the trachea. In 1764 an epidemic of aggravated " thrush or aphtha decimated the Moravian flocks. Pustules appeared on the mouth, nose, and in the gullet, and dysphagia was present. The lesions appeared like bladders, and seemed to occupy the entire mouth and gullet. When the aphtha began to subside, tumours of various sizes appeared at the posterior part of one or both fore-feet. If the tumours on the foot commenced prior to the disappearance of the mouth lesions, the latter vanished rapidly, but lameness persisted and increased, and the majority of the sheep lost their feet."

In the *Veterinarian* (1836) Rawlins of Bristol reported an outbreak of aphtha. Thirty lambs were more or less affected, and presented a large fungus issuing from and around the lower gum, enveloping the teeth and protruding over the lip. When the lesion was advanced, the ewes refused to let the lambs suck—hence the latter pined—and at this stage the disease was conveyed to the udder of the ewe. The udder became swollen, and in extreme cases suppuration set in, and portions of the gland, with one or two of the teats, sloughed, and the ewe was rendered useless for stock purposes.

Williams * states that " orf " was regarded by some English and Continental authorities as a non-contagious form of foot-rot in sheep. He describes the disease as " carbuncle of the coronet," affecting particularly young sheep, especially during wet seasons and in spring and autumn. if the animals were kept on wet rough pastures. The symptoms observed were lameness, swelling of the coronet, and interdigital space ; the swellings point and rupture, leaving angry-looking ulcers which are speedily filled up with granulations that bleed readily. The swelling may extend up the limb and the ulcers may attain a large size. Similar lesions may appear on the face and head, and are probably due to contact with the affected limbs.

The malady was also recognised by Clark of Coupar-Angus.

Gresswell met with instances in which ulcers appeared simultaneously on the lips and legs of lambs.

In 1901 Berry drew attention to the disease as occurring in England, Scotland, and Wales, under the name of " contagious pustular dermatitis " (see p. 305).

Under the designation of " acute stomatitis affecting the lips and mouth of lambs," Gilruth described the disease in 1907, and observed several instances of transmission of the malady from the mouth of the lamb to the udder of the ewe. The same observer in 1908 drew attention to the affection occurring in the scrotum of castrated lambs. After the operation this region became swollen, ulcerated, and gangrenous. At the same time the disease appeared amongst the ewes, at the site of wounds due to shearing, and also at the caudal trunk after docking. Lambs, sheep one or two years old, and even old ewes, were attacked. The contagious nature of the affection was proved by inoculation experiments.

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* " Principles and Practice of Veterinary Surgery."

In the United States of America the disease was described by Melvin and Mohler, Knowles, Law, Rushworth, and others, and it was termed "lip and leg ulceration of sheep."

In 1900 McFadyean* described the affection known as "orf," and also a disease termed "malignant aphtha." In the former, lambs of any age up to a year old were attacked, and the disease was rarely seen in adult sheep. The lesions were not unlike those of aphtha, and they usually appeared on the face and legs. On the face they occurred on the skin around the nostrils and on the hairy surface of the lips, but never in the interior of the mouth. On the legs the usual seats were between the top of the hoof and the knee, or even higher, and sometimes the sensitive structures around the top of the claws were also involved, with more or less lameness as a consequence. In "malignant aphtha," ewes and young lambs were usually attacked. In lambs the lesions consisted of sores on the nose or lips, generally covered by brownish crusts or scabs. They did not occur in the interior of the mouth, but on the outside of the lips or around the nostrils, and lambs more than a few weeks old were rarely affected. In ewes the lesions developed on the teats and udder. It seems probable that malignant aphtha is only one of the forms of the disease we are discussing.

Hutyra and Marek observed in Hungary that two imported deer showed evidences of pustular stomatitis. These were placed amongst a flock of 500 sheep, and the entire lot became infected. Two deaths occurred, and at the autopsies lesions in the lungs were discovered.

In 1908 Hasenkamp described "ulcerative stomatitis" and "red foot" as occurring in Germany, due to the bacillus of necrosis, and observed secondary infections in the liver and lungs due to this bacillus. Moussu, Besnoit, Cadéac, Leclainche, and Vallée, also described the disease.

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* Annual Report for 1900 from the Principal of the Royal Veterinary College to the Royal Agricultural Society (*Journal of Comparative Pathology and Therapeutics*, vol. xiv.).

ETIOLOGY.

The bacillus of necrosis is regarded as the cause of the disease. Its entrance into the body is facilitated by wounds of the skin, mouth, and various tissues. Dry seasons are believed to favour the occurrence of the affection, as grass becomes scarce, and the animals tend to eat hard and prickly plants, which are likely to cause slight punctures of the oral mucous membrane. Any circumstances which induce fissures in the skin of the limbs, such as standing in low-lying, wet, or muddy pastures, facilitate the entrance of the bacilli. According to Rouaud, once the bacillus gains entrance to the system, it secretes toxins which exert a marked destructive effect, and induce necrotic changes in all the tissues of the body.

According to the above authority, the disease can be transmitted from one species of animal to another, and all forms of the malady can be produced according to the site of inoculation.

British authorities, however, maintain that the affection is not conveyed to cattle in direct contact with the diseased sheep.

The affection is spread by cohabitation of the diseased animals with the healthy, as the litter becomes contaminated by the bacilli, and wounds, etc., thus become infected.

LESIONS.

Rouaud* reviews the various details recorded in connection with the disease, as set forth in the report of Melvin and Mohler, Bureau of Animal Industry (Washington, U.S.A.), 1910. He is of opinion that four types of the affection should be recognised :

1. That attacking the *lips* and the *legs*, either separately or coexistent in the same animal. Lesions in the limbs predominate in winter, when snow is on the ground. Fodder containing hard, tough, spiky plants is likely to cause punctures in the oral mucous membrane, and to induce a labio-buccal type. This latter type may assume an *acute* or *chronic* form.

The *acute* form is manifested by ulceration and necrosis of the parts affected, with or without the formation of crusts. The lips and the tip of the nose may show distinct loss of substance, and the bacillus of necrosis is found in the lesions in the form of beads and filaments, on the border of the ulcers, and penetrating into the sound tissues. The lesions at first present a pustular appearance, and a marked inflammatory swelling of the affected parts is observed. The pustules dry up, and dark grey crusts are formed. The lesions become confluent, and the result is an extensive ulcerated surface covered by crusts. In severe cases there is interference with respiration and the prehension of food. On the limbs the lesions follow the same course as that described above, and lameness may be produced.

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* *Revue Vétérinaire*, January and February, 1911.

The *chronic* form shows no swelling, the lesions are not of a proliferative character, and the specific micro-organism is found in the degenerated débris in the form of bacilli or cocci.

2. The *venereal* type affects the genital organs in both sexes. It may coexist with the type already described. In the male the prepuce is more frequently attacked than the penis. The lesions commence as small pale spots on the mucous membrane, showing a yellow centre. These are formed into ulcers, which extend to the surrounding skin and become confluent, so that the entire prepuce becomes inflamed and swollen.

In the female the lips of the vulva, the perineum, and the inferior aspect of the tail, become the seats of the disease. The lesions are similar to those occurring in the male. A muco-purulent discharge issues from the vulva, and the latter is inflamed and swollen. Infection is believed to occur through contaminated litter and from the floors of infected buildings.

This type is evidently similar to the venereal disease in sheep described by Flook, McFadyean, and Williams (see p. 364).

3. The type known as "*red foot*" was observed in Germany and in the United States of America. In the majority of instances the lesions were confined to the feet, but sometimes the lips were also attacked. The disease commenced in the claw, and extended to the interdigital space, in which ulcers were formed that involved the deep structures of the foot, and a fœtid discharge was observed. Animals of both sexes and of all ages were attacked.

4. "*Mouth disease*" (*mal de bouche*) occurs amongst lambs. The buccal mucous membrane is chiefly affected, the lesions consisting of small ulcers and soft red fungoid projections of a spongy consistency, giving off an odour resembling that of Limbourg cheese.

Berry* described the disease under the heading of "Contagious Pustular Dermatitis of Sheep," and stated that it was met with from time to time in England, Scotland, and Wales, occurring more frequently "in low-lying, undrained, and wet lands, especially during or after prolonged or heavy rains, and where the herbage of such pastures is long, coarse, and inferior in quality."

Lambs or sheep under one year old are most commonly attacked, and in them the disease is more severe; but in many outbreaks sheep of all ages become affected. When infected sheep are brought into a flock of healthy animals, many of the latter will show symptoms of the disease within two weeks afterwards.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xiv.

The chief seats of the affection are the nostrils, lips, and coronets, and the lesions in uncomplicated cases generally consist of a pustular inflammatory eruption of the skin of these regions. The simple lesions commence as hard papules, varying in size from that of a small pea upwards. Pustules are formed, accompanied by much inflammatory swelling of the skin. The purulent contents of the pustules glue the hair together in small patches or tufts, and in conjunction with the proliferated epithelial cells a hard scaly crust is formed. Under this crust and around its circumference pus is found, and if the crust be removed an ulcerating surface is left, which in some instances shows unhealthy-looking granulations around its edges.

The chief seat of the lesions of the mouth is the outside of the lips, especially that of the upper lip. The pustules may extend close to the line of junction of the mucous surface and the skin, but as a rule the mucous membrane proper of the mouth is not involved. In a small number of aggravated cases, however, ulcers may be found on the mucous membrane of the lips and tongue. According to Berry, this complication is probably due to the ichorous purulent discharge, with the accumulation of effete matter around the commissures of the lips, coming into direct contact with the mucosa. In ordinary cases well-marked suppuration is not found in connection with the lesions of the lips. The pustules are generally small, rather numerous, and tend to coalesce, so that the affected part appears to be the seat of a moist eczema. Vesicles are never seen, except the parts have been dressed with irritating medicaments. In some instances pustules may be found on the skin covering the nasal bones and around the orbit; also small pustules may be observed on the skin of the cheeks.

In a few cases the pustules may occur on the skin immediately surrounding the nostrils, accompanied by acute inflammatory swelling of the parts, so that respiration may be interfered with. An abscess may occur in either the upper or lower lip as the result of large isolated pustules in these regions. In aggravated cases the lips may show various lesions, such as extensive swelling, deep fissures or cracks, ulcers, etc. The surrounding parts may be denuded of hair and epidermis, and be raw and tender.

The mucous membrane of the tongue may be implicated. The ulcers in this region are usually not extensive. They occur on the superolateral aspect of the organ which is nearest to the aggravated lesion of the lips, the usual seat being a little in front of the first molar tooth; but in severe cases they may extend to the edges and under surface of the tongue.

The first appearance of the lesion is that "of a small superficial wound or sore, in which the piece of lost mucous membrane looks as if it had been 'punched out,' and the edges of the wound—that is, the mucous surface—are not free, but closely in contact with the underlying structures of the tongue. The average size of the lesion is from that of a threepenny-piece to a shilling, circular or oval in outline, and the latter from 1 inch to 2 inches in length; but the size entirely depends upon the extent of the ulcerative and suppurative surface that the tongue is brought into contact with. In these cases mastication during rumination appears to be entirely unilateral, the sheep performing the function with caution and care to guard the sores, and the condition gives rise to a little extra salivation."*

The lesions in the feet are similar in character to those occurring in the lips, but are often more severe and aggravated. The usual seats are the anterior and lateral aspects of the coronets of either the fore or hind feet. Some of the pustules are of large size, and form abscesses in the region of the coronary band. The adjacent skin undergoes necrosis, and separation of the skin and horn results, with the formation of a suppurating wound. In rare cases, when the animal is kept under unfavourable conditions and neglected, the suppurative process extends under the horny laminæ, and the claw may be shed.

In many instances the pustules may extend up the limbs, being few in number on the metacarpal and metatarsal regions, but fairly numerous around the lower parts of the knee and hock joints, and occurring especially on the flexures of these joints. Lesions may also occur around the fetlock-joints and in the vicinity of the supernumerary digits.

Only in occasional cases are lesions found affecting the heels in the interdigital spaces, and separation of skin and horn does not occur in these regions unless from extension of pre-existing lesions affecting other parts.

On comparing this description of the foot lesions with that given of "red foot" (see p. 305), it will be seen that in the latter the deep structures of the feet are involved.

The degree of lameness present will depend on the extent of the foot lesions.

If the cases are not far advanced, and the lesions are superficial, lameness may be absent.

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* Berry, op. cit.

DIFFERENTIAL DIAGNOSIS.

The disease with which contagious pustular dermatitis may be confounded is foot and mouth disease. The distinguishing features will be found on p. 786.

PROGNOSIS.

If early treatment be adopted, and the surroundings be favourable, recovery is the rule. But in winter-time the prognosis is grave, and serious losses may occur.

According to Berry, " local treatment is attended with well-marked results, and if the sheep are taken in hand before the lesions become aggravated by dirt and the presence of accumulated discharge, they speedily recover."

Williams states that the disease is not a fatal one, but is very troublesome, and when once established, the lesions are slow in healing.

PROPHYLAXIS.

Immediate isolation of the affected animals should be carried out. All animals in contact should be closely examined, and also isolated, should any suspicious symptoms be present. Thorough disinfection of buildings, etc., should be enforced. The healthy animals should, if possible, be removed to new pastures, preferably to high, well-drained, and dry land.

TREATMENT.

The following treatment is advised by Rouaud : In benign cases the crusts should be removed, and the following dressing applied : Cresol or coal-tar, [illegible] parts ; sublimed sulphur, 10 parts ; vaseline or mutton-fat, 100 parts.

In grave cases or in the chronic forms, after removal of the crusts, caustic solutions should be very carefully applied to the affected parts only. A 10 per cent. solution of chloride of zinc is advised, or a solution of nitric acid, 1 to 7 of water.

Either of these is to be carefully applied, once only, by means of a pointed stick covered at the end with a piece of cloth. This treatment is to be followed by three dressings a week with the ointment already mentioned. The crusts should be removed by means of a small piece of wood, and not by scraping with a sharp instrument.

When a large number of animals are affected in the feet, the flock should be made to walk through a trough containing an antiseptic solution. This is to be repeated each day for a period of three weeks.

In the venereal type of the disease in the male, the necrosed lesions on the penis should be touched with one of the caustic solutions mentioned above, and solutions of potassium permanganate (1 in 500) or of peroxide of hydrogen (25 per cent.) should be injected into the prepuce daily. In the female similar injections into the vagina should be carried out.

As the bacillus of necrosis is anaërobic, every means should be adopted to expose the lesions to the action of oxygen, such as by removal of the crusts and by the application of solution of peroxide of hydrogen or permanganate of potassium.

In the treatment of the mouth lesions solutions of potassium chlorate (2 per cent.), boric acid (3 per cent.), or creolin (1 per cent.), are prescribed. German practitioners recommend the application of the following liniment to the external lesions: Creosote, 1 part; cod-liver oil, 50 parts. They also administer to each lamb, twice daily, a tablespoonful of a similar combination.

Good nourishment and proper hygienic surroundings are important adjuncts to treatment.

It is advised that animals treated and cured should not be returned to the healthy flock until after a period of at least two weeks' observation.

Berry advises that a small quantity of dry fodder be given morning and evening, such as hay, chaff, linseed and cotton cakes. Rock-salt should also be allowed. As medicinal treatment, he recommends a mixture of common salt, iron, and gentian, added to the fodder. Local treatment consists in the application of an antiseptic ointment, in order to separate the crusts when these are very hard. Thorough cleansing is then carried out, and each foot is placed in a shallow bucket or trough containing a solution of some antiseptic, such as lysol, Jeyes' Fluid, carbolic acid, Condy's Fluid, or hyposulphite of soda. He finds that one or two thorough dressings are generally sufficient, but the worst cases will require attention until improvement is observed.

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THE RELATIONSHIP BETWEEN HUMAN DIPHTHERIA AND CERTAIN DISEASES OF ANIMALS

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The belief that diphtheria of the human subject may originate from disease of the domestic animals has prevailed in some measure since human diphtheria was first recognised and acknowledged to be a specific and contagious affection. The discovery and description by Löffler in 1883 of the bacillus of human diphtheria, and the reports in the following year of his investigations into the causes of the avian affection and of ulcerative stomatitis of calves (calf diphtheria), removed much of the prevailing misconception as to the nature of these diseases and the relationship in which they stood to one another and to the human disease. The study of the epidemiology of human diphtheria and a greatly increased knowledge of the character and habits of the causal bacillus have resulted in the conclusion, universally accepted at the present day, that the principal sources of infection for the healthy human being are individuals obviously affected with diphtheria and those who are acting as the carriers of the organism of that disease without showing clinical signs of its presence. But although it is generally accepted that the infected human being is the most important source of infection for his fellows, the view still prevails that animals—more especially cats—are responsible, even though in a subsidiary degree, for the dissemination of human diphtheria. Veterinary pathologists and clinicians have consistently contested this view, and maintained that there are no facts to support it. On the other hand, the opinion has flourished in varying degrees, more particularly in medical circles, and Klein's much-debated experiments and observations have been extensively quoted in its favour.

Apart from Klein, there is extremely little to be found in the literature on the subject in support of the occurrence of diphtheria in animals. He asserted that cows and cats were the natural subjects of affections caused by the bacillus of diphtheria, and acted as sources of infection to man, the former by means of milk and the latter by contact, and it is necessary to briefly review the experiments and observations on which this opinion was based. Klein inoculated cows subcutaneously with bacilli isolated from diphtheritic membranes of human origin, and claimed that a definite infection was produced, important features of which were the appearance of a vesicular eruption on the skin of the udders and teats, from which the diphtheria bacilli could be recovered, and the elimination of the bacilli in the milk. The experimental animals died or were killed in a moribund condition.

A careful examination of Klein's reports leaves some uncertainty as to the identity of the bacilli with which the cows were inoculated, but if it be accepted that the cows were inoculated with the causal organism of human diphtheria, it cannot be accepted as having been established that the bacilli isolated from the milk and the teat lesions were those of diphtheria. Their recognition was dependant on morphological resemblance and culture in gelatine at 19° to 20° C. The morphological appearances of the *Bacilli diphtheriæ*, taken alone, are not sufficiently characteristic to permit of certain identification with them of organisms removed from the teats of cows, and abundant growth in gelatine at 20° C. is not an accepted feature of the diphtheria bacillus. A further remarkable point is that, whereas localisation to the seat of inoculation is a feature of the *B. diphtheriæ*, they were stated in these experiments to have appeared in lesions and in the secretions of a gland, both remote from the point of inoculation, and within a few days of the performance of that operation.

Löffler, commenting on these experiments, expressed the opinion that the conclusion based on them by Klein that diphtheria was a natural disease of the cow ought to be accepted only with the greatest caution. Abbot tested Klein's results by inoculating two cows with diphtheria bacilli, and found that lesions were not developed on the udders or teats, nor were diphtheria bacilli eliminated in the milk, and Ritter homologated his results. Both experimenters made a systematic bacteriological examination of the milk of the inoculated cows, and all bacilli showing the slightest morphological resemblance to the diphtheria organism were isolated, but found to differ from it in biological characters or pathogenic properties.

Milk-borne epidemics of diphtheria have been attributed from time to time to lesions of the udders and teats of cows similar to those described by Klein as occurring in his experimental animals. Klein himself records two such cases, but his note contains no reference to a bacteriological examination, and the diagnosis appears to have been based solely on the clinical and far from characteristic appearances of the teat lesions. With two exceptions, similar records of milk-borne diphtheria are identical in the absence of bacteriological support. The exceptions are the cases recorded by Deans and Todd, and that by Ashby. The former demonstrated virulent diphtheria bacilli in ulcers on the teats of two cows, and in milk drawn from their teats with all antiseptic precautions, the udder of one being normal and that of the other affected with mammitis in one quarter. They showed experimentally that the diphtheria bacilli did not cause the udder lesions, but they were unable to determine the source from which the organisms had been derived, though human contamination was suspected. Ashby recorded the isolation of diphtheria bacilli from the teat lesions of two cows on a farm, the milk from which was responsible for a milk-borne diphtheria epidemic. The bacilli were without doubt diphtheria bacilli, but, unfortunately, no experimental tests were made to determine whether the bacilli were responsible for the lesions on the cow, or were merely accidental contaminations, as in Deans and Todd's case.

It is doubtful if at the present day anyone would be prepared seriously to advance a theory that diphtheria is a disease of bovine animals. Klein's work, examined in the light of current bacteriological methods, leaves much room for question, and his results cannot be accepted, especially since other observers, working on similar lines, have failed to confirm them. Similarly, investigations of naturally acquired affections do not afford any evidence of the occurrence of diphtheria in bovines. If Ashby's case was really one of bovine diphtheria, which is extremely doubtful, it stands alone. Deans and Todd's case is important and instructive. It illustrates the passive part which cows may act in disseminating diphtheria, and emphasises the necessity of experimental tests to determine the rôle which diphtheria bacilli play in the production of animal lesions in which they may happen to be found, before the conclusion is reached that the animal affection is the result of the action of that organism.

It may be observed here that the ulcerative stomatitis of calves, to which, in his original description (1877), Dammann applied the term "calf diphtheria," because of his belief in its identity with the human infection, was shown by Löffler to bear no etiological relationship to it, and that Löffler's results were confirmed by Bang in 1893 (see p. 285).

In connection with the work already referred to, Klein conducted a series of experiments on cats, from the results of which he concluded that the cat was subject to infection with the diphtheria bacillus, and that an important feature of the resulting disease was a "characteristic enlargement and fatty degeneration of the cortex of the kidney." He also stated that by feeding with cultures of the diphtheria bacillus distributed in milk, unquestionable diphtheria could be produced in the cat. In the feeding experiments the diagnosis of diphtheria was based on the post-mortem appearances of the kidney only. The experimental cat disease bore " only a slight resemblance to human diphtheria ; the characteristic, thick, firmly adhering, whitish membranes were absent in the induced cat malady."

Embodied in Klein's report is a series of cases of ill-defined disease in cats associated with human diphtheria, and the suggestion is made that the cat disease was responsible for the human infection. In all these cases, which are recorded as instances of naturally acquired diphtheria in the cat, the identity of the cat disease with human diphtheria was a pure assumption, based on the coincident occurrence of the human and feline affections, since in all of these cases in which a bacteriological examination was made there was a complete failure to find diphtheria bacilli. In connection with his experiments on cows with diphtheria inoculations, Klein records an outbreak of disease amongst the cats in his laboratory, which he described as accidental diphtheria. He attributed infection to feeding the cats with milk from the experimental cows, which he stated were eliminating diphtheria bacilli in their milk. From what has already been said, these cows must be regarded as a questionable source of infection. The conclusion that the cat disease was diphtheria was based on the post-mortem appearances of the kidneys and on the recognition in some of the cat lesions of bacilli which, in their morphological characters, resembled the diphtheria bacilli, but whose identity was not subjected to cultural, experimental, or other test. A second identical outbreak, for which there was no known or traceable source of infection, occurred at a later date amongst the laboratory cats, and is recorded as natural diphtheria—an opinion which was based solely, as in the previous instance, on the microscopical recognition in one case only of bacilli presenting morphological resemblance to the organism of diphtheria.

It is on these facts that the prevailing belief in cat diphtheria as a source of human infection is founded, and they contain nothing which justifies the conclusion that the cat may acquire naturally a disease caused by the Klebs-Löffler bacillus. The bacteriological evidence of infection is either negative or of too limited and unsatisfactory a character to permit of the conclusion that diphtheria is a disease of the cat. The appearance of the kidney described by Klein as pathognomonic cannot be so regarded, since it is an exceedingly common condition of the cat's kidney. If the cat were susceptible to diphtheria, its close association with the human race would quickly produce evidence of the fact; and, moreover, if by contracting diphtheria cats communicated that disease to members of the human race, they would disseminate it simultaneously with greater certainty amongst the members of their own species; but such cat epidemics do not occur in association with human diphtheria. Affections presenting diphtheritic characters are met with in the cat, and occur independently of human diphtheria; but the bacteriological investigation of these affections has up to the present always resulted in a failure to demonstrate the presence of the human bacillus, and there is no evidence of any disease originating in man from this source.

Cobbett demonstrated in the nasal discharge of a horse the presence of a bacillus which possessed all the morphological, cultural, and pathogenic properties of the diphtheria bacillus. As the result of the isolation of these bacilli from the mixed organisms of the nasal discharge, Cobbett concluded that the case was one of true diphtheria, and ventured to suggest that diphtheria was a common disease of the horse, and that the horse might be found to play "a not inconsiderable part in the transmission of diphtheria"—a broad general conclusion which has not been supported by the demonstration of a similar case. Unfortunately, no test was made of the infectivity of the horse's condition to other animals of the same species, and thus the opportunity of proving whether the diphtheria bacilli were really responsible for its occurrence or had been merely grafted on to a previous infection was allowed to pass.

It thus appears that in those instances in which bacilli which without doubt were those of diphtheria have been demonstrated in naturally acquired lesions of animals, inquiry has shown that in one the bacilli were not etiologically responsible for the animal disease, and it is unfortunate that in the others the investigations were not carried to a point which permitted of a definite conclusion as to the exact relationship in which the bacilli stood to the animal affection. A statement to the effect that animals never contract a disease caused by the bacillus of human diphtheria might, therefore, be regarded as unjustified, but it can with every assurance be asserted that diphtheria bacilli have never been demonstrated to be the exciting agents in animals of a disease which had been naturally acquired. A belief to the contrary, so far as it exists at the present day, receives the support solely of those having a very limited knowledge of the diseases of animals, either from a clinical or a bacteriological standpoint, and rests upon the most uncertain and doubtful foundation.

It may, almost without fear of controversion, be asserted that the diphtheria of birds is an affection quite distinct from that of man.

Löffler came to that conclusion as the result of his earliest researches, and his views have been repeatedly confirmed. Birds are not insusceptible to infection with the Klebs-Löffler bacillus. They do not, however, develop diphtheritic lesions except as the result of intratracheal inoculation, to which birds are very resistant, and the results of which are so very uncertain that it may be regarded as beyond the region of probability for birds to develop in epidemic form a disease showing diphtheritic characters, and caused by the bacillus of human diphtheria. Nevertheless, organisms which have been regarded as identical with those of human diphtheria have been isolated in a few rare instances from avian lesions, and looked upon as their cause. With very few exceptions, these have differed from the human organism in being non-pathogenic for the guinea-pig. Ferré, however, discovered in diphtheritic birds and in the mouths and cloacæ of healthy fowls a bacillus resembling the human organism in its morphological characters, and which produced a toxin capable of causing paralysis in guinea-pigs and fowls similar to that which followed inoculation with the toxin of the human organism. Gallez also recorded the discovery of a bacillus resembling the human organism which was pathogenic for the guinea-pig, but in a very low degree, very large doses being required to produce fatal effects. On the other hand, antidiphtheritic serum has no influence on the course of the avian affection. Guèrin treated over two hundred naturally infected fowls, and obtained results in harmony with those of Harrison, the antitoxin producing no greater effect on the course of the disease than the injection of normal serum.

Clinical observations have been recorded from time to time on the simultaneous occurrence of the human and avian affections under circumstances which appeared to suggest the avian affection as the starting-point of the human diphtheria. They lend a feeble support to an opinion that the two are etiologically related, but any apparent value which they might possess in this respect is completely nullified by an entire absence of bacteriological evidence of the similarity of the coexistent affections. The almost world-wide distribution of the avian affection and the frequency of its occurrence would afford, if any etiological relationship existed between the two affections, ample evidence of the fact, and be so productive of reports of their simultaneous occurrence as to compel widespread attention, with the result that the necessary proof of their similarity and communicability would soon be forthcoming.

The work of Bordet and Fally deserves mention in this connection. After a long and careful investigation into the cause of avian diphtheria, they arrived at the conclusion that it is due to a minute organism about

$\frac{1}{5}\ \mu$ in diameter, and that it is essentially different clinically and bacteriologically from the human disease. Nuttall and Graham-Smith, after a careful résumé and review of the literature on the subject, express the opinion, with which we are in full agreement, that the investigations into this matter "point to the conclusion that true diphtheria caused by the diphtheria bacillus, if it ever occurs, is a very rare disease in birds."

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I. DIPHTHERIA OF THE CAT.

By H. GRAY, M.R.C.V.S.

Synonym.—Gangrenous or malignant sore throat.

Definition.—A contagious disease of the cat, characterised by the presence of diphtheritic membranes on the fauces, pharynx, or larynx, and due to some micro-organism yet to be determined.

Nature.—It is a very important malady from a public health point of view, as it has been alleged by Dr. Klein and others to be identical with that occurring in mankind. But the writer doubts the correctness of this view, and does not believe that the disease can be conveyed from the cat to man, for he has time out of number seen people kissing creatures so affected without becoming infected. From some lesions forwarded by the writer to Sir John McFadyean in the year 1896, this authority from cultivation experiments failed to reveal the presence of any diphtheritic bacilli in the false membranes. The latter contained a variety of organisms, but the one most abundantly present was a small bacillus which on agar formed large white colonies resembling those of the *Staphylococcus albus*. This same authority failed to transmit the disease to a young cat (six months old) by feeding it with milk to which particles of the diphtheritic membrane had been added.

Occurrence.—Although it may occur in any breed of cat, it is chiefly seen in light-coloured Persians, as the Chinchilla, Blue, Grey, and Smoke. In many instances it occurs as an enzoötic, attacking every cat in the house or in a cattery.

Mortality.—This is quite 90 per cent.

Symptoms.—The animal goes off his appetite, he shivers, has an open, lustreless coat, clear saliva flows from the angles of the mouth, there is a slight discharge from the eyes and nostrils, a croupy or dull cough, and he seems to breathe through his mouth, which is sometimes partially open, and emits a gurgling sound. After a time the discharge from the eyes and nostrils alters from a slight amount, clear colour, and thin consistence, to a profuse viscid material of a straw colour, and sometimes tinged with blood. There may be frequent sneezing and a peculiar noisy breathing, as if the throat were paralysed. The gait is staggering. At times there may be slight vomiting. The mouth and the discharges therefrom are very fœtid. Towards the end of a fatal case the animal becomes extremely weak, sits in a drooping position, seems somnolent, moans or cries when moved or touched, and rarely has sufficient energy left to offer even slight resistance to handling or dosing. In some cases, when the mouth is forcibly opened, it remains passively open. The pupils are generally dilated.

On inspection of the back of the mouth, there may be found a pinkish or port-wine coloured throat, which is swollen, and shows patches of greyish-white, yellowish, or greenish membrane on the soft palate, the fauces, the pharynx, and the larynx. These patches may run into one another, forming a continuous covering to the whole of the throat, or they may be isolated and studded here and there.

Diagnosis.—It may be mistaken for the pharyngeal form of distemper, of which it may only be a more severe type. Fish-bones, needles, and other foreign bodies in the throat, may give rise to symptoms somewhat resembling those of diphtheria. The absence of such and the presence of false membranes places the nature of the malady beyond all doubt.

Prognosis.—In fatal cases the pupils become widely dilated, there is a meaningless facial expression, the breathing becomes so noisy as to resemble something between a grunt and a moan, there is great drowsiness, which passes into a profound comatose condition, and the animal evinces now and again a delirious cry, and dies with convulsive movements of various muscles of the body.

Treatment.—This being a very contagious malady to other cats, the animal should be promptly isolated. Treatment consists in painting the affected throat with glycerine of iodine, the administration of quinine and stimulants, and painting the outside of the throat with strong tincture of iodine. Good nursing is essential. The diphtheria antitoxic serum may be tried.

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II. AVIAN DIPHTHERIA.

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Synonyms.—Diphtheritic roup ; Avian roup.

French : Diphtérie des Pigeons ; Diphtérie aviare.
German : Diphterie der Tauben ; Geflügeldiphterie.

Definition.—The term "avian diphtheria" is applied to a contagious disease of birds characterised by a diphtheritic inflammation of the mucous membranes, and affecting particularly the first portions of the alimentary and respiratory tracts.

General Remarks.—The domestic fowl and the pigeon are the most common subjects of the disease, but turkeys, ducks, geese, pheasants, and guinea-fowl, are also susceptible ; and it appears to be probable, from the large variety of birds, wild and domesticated, in which the disease has been encountered, and to which it has been experimentally inoculated, that all birds are susceptible to infection.

Avian diphtheria has a very wide distribution. It occurs in all parts of Europe, in Africa, and America, and probably few, if any, countries are free from the disease. In Great Britain it generally assumes enzoötic characters. According to Friedberger and Fröhner, it is, next to fowl cholera, the most common and the most dangerous epizoötic occurring amongst poultry in Germany. Its appearance in a poultry-run is certainly always a cause of great loss. Poultry shows and the sale and exchange of birds for breeding and other purposes are the chief means by which the disease is spread. It has frequently appeared as an epizoötic among wood-pigeons at liberty.

Etiology.—In view of the easy and extensive contamination of lesions in the mouth and pharynx of birds, and of the fact that a number of organisms, when inoculated into the buccal mucous membrane, are capable of producing lesions bearing the closest resemblance to those of avian diphtheria, it is not surprising that a variety of organisms have been isolated and have been regarded as the cause of the disease. Of these organisms, the most important is a cocco-bacillus belonging to the pasteurella group isolated by Löffler and others, by means of which they claimed to have produced the disease, and which was accepted by Nocard and Leclainche as the causal agent. Guèrin isolated a very similar organism, and described a process of sero-vaccination by which he claimed to have immunised fowls. Protozoa have also been described in the lesions, but they appear to be accidental contaminations. According to Bordet and Fally, the causal agent is not a pasteurella, but an extremely minute organism about $\frac{1}{5}\ \mu$ in diameter, which appears under the highest magnification as very small points or in slightly elongated form as slender rods. This organism cannot be identified with certainty in the lesions, owing to its scanty numbers, especially in the older lesions, its small size, and its resemblance to cellular granules. In order to isolate it, healthy birds were inoculated by passing through the membrana nictitans a thread saturated in an emulsion of false membrane removed from a natural case. The organism occurred in nearly pure culture in the lesions and lachrymal discharges of the experimental birds. Special media containing defibrinated rabbit's blood were required for its cultivation, the growth being very meagre, and appearing as little more than a slight darkening of the surface of the medium. The disease was reproduced by inoculation of the cultures. There is little room for doubt that the organism isolated by Bordet and Fally is the essential cause of the diphtheritic condition of poultry which they investigated, but it cannot be regarded as established beyond doubt that all the affections of birds which have been described under the name of "avian diphtheria" are identical. The incubative period in experimental birds is three or four days, and probably under natural conditions it does not exceed a week or ten days.

Symptoms and Lesions.—In a great many cases a number of birds are affected and the lesions well advanced before attention is directed to them. The sick birds isolate themselves and stand about with wings and feathers drooping; the combs and wattles are soft and flaccid, and of a bluish or purplish colour. The birds eat little, swallow with difficulty, offer very little resistance to handling, and hens cease to lay. In many advanced cases the respirations are obviously obstructed, the beak is constantly held open, and emaciation is well marked.

Lesions may be present simultaneously on the mucous membranes of the mouth, pharynx, nostrils, trachea, and situated on the membrana nictitans and conjunctiva, or they may be found in one only or in any combination of these positions. The mouth and pharynx are almost constantly affected, and the lesions may be easily seen on opening the beak. They appear as greyish-yellow, fairly firm, caseous-looking masses, with a rough surface which often overhangs their base. They often attain a considerable size and thickness, are more or less firmly adherent, and their detachment leaves a bleeding superficial sore. Quite commonly in advanced cases the upper part of the trachea is nearly blocked by a mass of this caseous-looking deposit. The mouth is always fœtid. An inspection of the apparently healthy birds always reveals a number with lesions in an earlier stage. These take the form of greyish unraised spots, often quite small and discrete, but which later coalesce and become covered with the thick greyish-yellow deposit.

Very similar lesions may appear on the conjunctiva and membrana nictitans; a muco-purulent discharge escapes from the eye, which may be closed, or the palpebral fissure may be blocked with greyish masses of caseous-like material.

On post-mortem examination there may sometimes be found, in addition to the lesions already described, a catarrhal condition of the bronchial and intestinal mucous membranes, a few small subserous hæmorrhages, and a soft, friable condition of the heart muscle.

The course of the disease is variable. Frequently it is chronic and slowly progressive, and birds are often affected for some time before showing obvious symptoms of illness. The majority of the affected birds succumb to the disease after a period of illness varying from a few days to several weeks. The loss is always greater amongst the young than the old birds; a number of the latter appear to possess a considerable degree of natural resistance to the affection.

The exudates which cover the mucous membranes are virulent, as well as the discharges which escape from the mouth or are coughed up, and it is highly probable that the fæces are infective in the majority of cases. Many of the eye lesions are due to inoculation by scratching, and the facilities which exist for contamination of the feet are obvious.

Treatment.—Treatment consists in the removal of the false membranes with a minimum of injury to the underlying structures, and the application of suitable antiseptics. Solutions of silver nitrate, 2 to 2½ per cent., or lysol, 2 per cent., may be used. Friedberger and Fröhner recommend corrosive sublimate in solution (1 in 500 to 1 in 1,000), or creolin 1 or 2 per cent., painted on to the mucous membrane after removal of the exudates. Turpentine has been recommended, and also liquid carbolic acid, pure and in various dilutions. The dressings should be applied once or twice daily with a brush.

From an economical point of view, however, treatment is seldom advisable, on account of the heavy mortality and the difficulty of ascertaining when recovery is complete. Birds which have apparently recovered often suffer relapses, or, owing to overlooked lesions, continue to act as sources of infection.

Prophylaxis is of greater importance. The healthy birds must be removed to fresh non-infected quarters, and the mucous membranes of the mouth and pharynx inspected systematically once daily. The detection of any suspicious appearance of the membrane should be followed at once by segregation. Once the disease is well established in a poultry-run, however, this procedure is rarely profitable, and in the great majority of instances a considerable number of birds have died or are affected before steps are taken for its recognition. Economically, the most advisable course is usually a complete clearance of the poultry and the marketing of the healthy birds. The houses and runs must be thoroughly scraped and disinfected before fresh birds are introduced, and it is advisable, owing to the difficulty of insuring adequate disinfection of all places to which the poultry have had access, to leave the premises empty for several weeks. They may be regarded as safe after one month, provided disinfection as far as practicable has been carried out. The segregation of all newly-purchased birds is a judicious procedure.

Public Health.—It is a common practice amongst poultry owners to use birds for human food in the earlier periods of the affection, after removal of the head. Although we are not aware of any evil effects arising therefrom, we are of opinion that the carcasses of affected birds should be condemned as unfit for food.

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NOTE BY EDITOR.

According to Nocard and Leclainche, cases have been recorded in which the occurrence of a pseudo-diphtheritic angina in man was clearly traced to infection from avian diphtheria. They cite the following instance, observed by Loir and Ducloux: "A child, seven years of age, living in the neighbourhood of Tunis, on a farm where avian diphtheria raged for six months, developed a grave angina, with the presence of slightly thick and slightly adherent false membranes, in which was found, by culture, a bacillus offering all the characters of that of avian diphtheria. This bacillus, inoculated under the skin of a fowl, led to the death of this bird in five days, and on post-mortem typical lesions of avian diphtheria were found, and the bacillus of this affection was demonstrated by bacteriological methods. . . . The bacterium of avian diphtheria should, therefore, take its place among the causal agents of pseudo-diphtheritic angina of man." A consideration of the above views leads us to conclude that, although avian diphtheria cannot produce human diphtheria, it may possibly cause a serious form of angina; hence suitable precautions should be taken to prevent infection from this source.

III. EPITHELIOMA CONTAGIOSUM.

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Synonyms.—Avian variola; Fowl-pox; Chicken-pox; Epitheliosis; Pigeon-pox; Canker.

Contagious epithelioma was described by Rivolta as gregarinous diphtheritis of birds, in the belief that it was caused by protozoon parasites belonging to the order of Gregarinidia; but the identification of the bodies found in the lesions as gregarines is open to grave doubt, and it has been clearly proved that they bear no etiological relationship to the disease. Like the preceding affection, contagious epithelioma attacks all the feathered inhabitants of the domestic poultry-yard, as well as most other birds. It frequently coexists with avian diphtheria.

Etiology.—Marx and Sticker, in 1902, demonstrated that the disease was due to a virus which was capable of passing through a Berkefeld filter, and their results were confirmed by Juliusberg and Hertzog. Burnet, however, obtained very varying results in his filtration experiments. He found that the virus was frequently arrested by the Berkefeld filter, although the infective material had been largely diluted with water. It has not been possible to cultivate the virus artificially, and its nature remains a matter for further investigation. It exists in the blood, internal organs, and in the fluids expressed from the lesions, and is possessed of considerable resistance to the action of heat and antiseptics.

The disease can be transmitted by rubbing juice from the lesions into an area of skin from which the feathers have been removed, or which has been lightly scarified, and the lesions then produced are confined to the part inoculated. According to Burnet, it can also be transmitted by intravenous inoculation and by feeding, and the lesions develop at the predilection seats of the disease.

Symptoms.—The disease is manifested by the appearance of wart-like nodules, varying in size to that of a pea, or slightly larger, on those parts of the skin of the head where feathers are few or absent, such as the nostrils, openings of the ears, the comb, wattles, and the eyelids, the last named being one of the most common sites. The nodules are encapsuled, firm in consistence, with a caseous centre, and usually remain discrete, but a number situated closely together form a mulberry-like mass. In some cases the contents resemble fatty tissue, except for being more vascular ; in others they are watery and contain flakes of coagulated material very much like small clots of milk. Lesions are also found on the buccal mucous membrane, the affected parts of which become covered with thick whitish-yellow exudate, in appearance resembling that of diphtheritic false membrane.

The disease, when affecting the buccal membrane, bears a very close resemblance to avian diphtheria, but differs from it in that skin lesions are never present in the latter affection. Another important point of difference is the virulence of the blood in contagious epithelioma and its avirulent character in avian diphtheria.

The affection, when limited to the skin, is comparatively benign, and spontaneous recovery is not infrequent. A greater proportion of fatalities occurs when the mucous membranes are involved, but death appears to be due in most cases to inanition or asphyxia, according to the position and extent of the lesions, rather than to any special activity of the virus.

On post-mortem examination, only the local lesions are found. There are no visceral lesions.

Treatment.—The treatment of the affection lies along lines identical with those recommended for avian diphtheria—namely, the removal of the lesions and the application of antiseptics. The use of tincture of iodine has been found very successful.

Public Health.—In every instance the carcass should be condemned as unfit for food.

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EPIZOÖTIC ABORTION IN CATTLE

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Synonyms.—Contagious abortion; Enzoötic or infectious abortion; Abortus infectiosus.

French: Avortement épizootique.
German: Seuchenhaftes Verwerfen.

In this country certain local terms are in common use—*e.g.*, "picking," "slipping," and "casting."

In human obstetrics the term *premature birth* is applied to the expulsion of a live fœtus at any age when it is capable of maintaining a separate existence; in calves this period is towards the end of the seventh month of pregnancy.

The terms *abortion* and *miscarriage* are used when a viable age has not been reached.

In cattle and in other domesticated animals these distinctions are not only unnecessary, but positively misleading.

Definition.—A specific uterine catarrh, due to *Bang's Bacillus of Cattle Abortion*, and characterised by the expulsion of the fœtus at any time from the beginning to the normal completion of gestation.

GENERAL REMARKS.

The disease is peculiar to cattle, although it is transmissible to other animals by inoculation. There are no clinical symptoms, except those immediately preceding the act of abortion, and the health of the cow is not directly impaired.

The organism reaches its habitat—*i.e.*, the pregnant uterus—through the alimentary tract and the blood-stream, or *per vaginam*; in the uterus a slow insidious catarrh is set up, resulting in the ultimate expulsion of the fœtus as a foreign body. The exudate, fœtal membranes, and fœtus, are highly infective, and the post-partum discharge from the uterus remains virulent for some time.

HISTORY.

The records of the disease are lost in antiquity, whilst its infectious nature has been recognised from very early times.

In Gen. xxxi. 38 reference is made to sheep and goats casting their young. Mascall in 1567, Lawrence in 1805, Skellet in 1808, Jonati in 1837, and Barlow in 1851, all mention the infectiousness of the malady, and the losses to which it gives rise. During the first half of the nineteenth century scientific opinion was curiously divided as to its specific nature, whereas lay opinion was largely in favour of this hypothesis.

The following significant passage occurs in the "Complete Farmer," 1807: "It is considered certainly contagious, and when it happens the abortion should be immediately buried, and the cow kept as widely apart as possible from the herd, and not receive the bull that goes with them."

St. Cyr, in 1875, stated his belief in the specific theory.

Franck, in the following year, and later Schnert and Bräuer, actually produced abortion by introducing the discharge and fœtal membranes from aborting cows into the vagina of healthy pregnant animals.

To Nocard belongs the credit of making the first scientific investigation of the disease; he established its infectious nature by a series of clinical observations, although he failed to isolate the specific organism.

In 1889 a report was published by the Highland and Agricultural Society of Scotland; whilst in England, in 1894, the Royal Agricultural Society appointed a committee to inquire into the cause of the malady.

In 1896 Bang, working in conjunction with Stribolt, had the honour of discovering the bacillus of cattle abortion. Ten years later, at Liverpool, Bang emphasised the fact that natural infection frequently occurs by way of the mouth; he also reported encouraging results in immunising non-pregnant animals with living cultures.

A Departmental Committe appointed by the Board of Agriculture in 1905 issued their first report in 1909; this was devoted to the etiology of the disease. A second report in the following year related mainly to measures of prevention. Both are replete with valuable information.

Thus Bang followed Nocard, and placed the pathology and bacteriology of the disease on a satisfactory basis; McFadyean and Stockman completed Bang's excellent work, and elucidated certain biological characters pertaining to the bacillus.

OCCURRENCE.

Epizoötic abortion is, and probably always has been, a widespread disease. Its ravages have attracted the attention of savants and laymen in every civilised country, more especially in the British Isles, France, Switzerland, Germany, Denmark, Belgium, Austria, and the North American Continent.

Whilst all mammals are said to suffer from abortion, it occurs most largely, as a contagious disease, amongst cows.

Recent researches seem to indicate that infectious abortion in mares, in ewes, and in other animals, is due to a specific organism peculiar to each species. Epizoötic abortion, on the other hand, affects cows and cows only, although it is pathogenic to the mare, ewe, goat, bitch, and guinea-pig by experimental inoculation.

No reliable data are available with regard to one species infecting another, but the chances of this occurring under ordinary conditions are probably remote.

Age.—Epizoötic abortion is essentially a disease of heifers and young cows; at the same time it may and does attack a susceptible animal of any age, and at any stage of gestation. On some farms most, if not all, of the cases have occurred in primiparæ; on others, cows of all ages have been affected; less often the disease has been confined to secundiparæ in certain years; such, at least, has been the experience of the writer.

Period of Gestation.—The majority of cases of "visible" abortion occur between the fifth and seventh month of gestation; the average period is probably between the fifth and sixth month, if a reliable estimate of the early or "invisible" abortions could be made. Some cows abort about the first or second month, and no suspicion is aroused until œstrum recurs a few days later.

Calves may be born alive towards the end of the seventh month and onwards. Seven-months calves are small and have short hair, but they are usually hardier than those born at eight months. Many also hold the opinion that cows come to their milk better at seven months than at eight months.

Amongst several hundreds of abortions which we have handled annually at isolation farms, the following figures are selected: In 300 consecutive cases, 80 per cent. were due to Bang's bacillus; average period of gestation, 6¼ months; average age, 4½ years; 8 per cent. of these aborted a second time; 2 per cent. aborted a third time; 70 per cent. of the animals retained the placenta either wholly or partially.

The dairy farms to which these animals belonged were all under the same management, and the herd was largely self-supporting; it included cows of all ages.

The *Course* of the disease is very insidious and chronic, and its existence remains unsuspected until the act is imminent. A marked feature of the malady is its extreme virulence upon its first appearance in a herd; a considerable number of the cows abort during the first year; a less number during the second year; whilst during the third year the cases are few in number, and the disease disappears spontaneously, provided no fresh animals are introduced. The great majority of dairy stocks are, however, not self-supporting; in these the later abortions are mainly confined to fresh recruits and to the heifers, and may continue for an indefinite period.

The *Percentage* varies considerably in different herds, according to the system of management and the virulence of the infection.

Fifty per cent. of abortions is common in districts in which the disease is rampant, whilst in some herds not a single calf has been reared for a year or more. We have had experience of large dairy herds in which 20 per cent. was the average figure for a number of years. Although mainly self-supporting, the disease assumed an enzoötic character in these herds, in spite of strict measures of isolation and disinfection. It is probable that the occasional introduction of fresh cows and the grazing of pregnant heifers on infected pastures accounted for the persistence of the malady.

Losses.—The economic loss is enormous, both to the individual and to the State. Many thousands of good milch cows, yielding 700 to 1,000 gallons of milk, are lost to the dairying industry every year by being fattened for the butcher. The loss to breeders is infinitely more serious, inasmuch as the source of income is more or less completely destroyed. It has been estimated that the loss in dairy cows averages £7 10s. per head; this figure is probably too low, when the indirect losses are considered.

Twenty per cent. of British dairy and breeding stocks are said to be affected, and there is little doubt that it exists in every county. Only compulsory notification, however, can determine the exact prevalence of the disease.

The losses in the case of dairy cows may be summarised as follows: (1) Diminished supply of milk; in heifers the glands may not become functionally active; cows seldom give their full supply, depending a good deal on the period of pregnancy; in all, the period of lactation is shortened, and some animals are "dry" for a long period. (2) Loss of calves at a non-viable age, or slaughter of worthless calves. (3) Temporary or permanent sterility, often the most serious factor in connection with the disease. (4) Udder disorders arising from parturition occurring whilst the glands are actively secreting, or from septic conditions in the uterus.

Indirectly, the dairy farmer is subjected to considerable inconvenience and expense in keeping up his milk-supply; it is often necessary to sell good cows, and to purchase unknown animals in the open market immediately before or after parturition. The risks attending this procedure are obvious.

The housing difficulty and extra labour are other factors which interfere with prophylaxis, and add to the cost of control.

VARIETIES OF ABORTION.—*Sporadic or Accidental Abortion* is a non-specific form of abortion which must be distinguished from the disease under consideration. Much confusion has existed in the past between the two forms, and doubtless many so-called sporadic outbreaks were in reality due to the bacillus of Bang; the latter, in all probability, is responsible for 90 per cent. of the abortions in this country.

Anything which disturbs the intimate relation which exists between the maternal and fœtal placenta may cause abortion, either accident or disease; if the latter is specific, the term *epizoötic abortion* is applied in the case of cattle.

Sporadic Abortion occurs to a limited extent in all domesticated animals; in cows it is usually due to accident or injury—any sudden impact on the gravid uterus, cows slipping and falling in the stall, fright and violent exercise (dogs), poisons, stomach and other disorders, particularly infectious diseases like foot and mouth disease. A certain number of cases are directly or indirectly due to tuberculosis.

It may follow disease of the uterus, any constitutional weakness or defect in the genital tract, or debility from any cause. Ergoted food, either grain or grasses, is said to produce abortion.

In exceptional cases great activity of the mammary glands may induce abortion by weakening the uterus and the system generally. The writer has known heavy milking cows on non-infected farms which for several years in succession (up to six or seven) failed to carry their calves the full term; these cows were seldom or never dry, and the calves were either dead at birth or died shortly afterwards. There was no discharge, nor could any abnormality of the uterus be detected by either rectal or vaginal examination, nevertheless a slight endometritis may have been present.

The "habit" of abortion without apparent cause, which is observed in the human subject, is not unknown in the lower animals, but is undoubtedly of rare occurrence.

In the simple or sporadic form single cases are the rule, although a number of abortions may occur from errors in watering and feeding and other causes. Such cases, however, disappear when the cause ceases to operate.

Epizoötic Abortion, on the other hand, is characterised by a succession of cases over an extended period, and the specific organism is easily discoverable in the fresh post-partum discharge or in the fœtal cotyledons; the macroscopic appearance of the after-birth is more or less characteristic, and the outbreak is often traceable to a previous case, a new arrival in the herd, a certain bull, or certain pastures.

While infection is the all-important factor, it has been observed that the more the milking qualities of a herd are developed by judicious breeding and management, the less resistant the genital organs become to disease of any kind, including abortion. There is little doubt that individual susceptibility is increased under an intensive system of management.

ETIOLOGY.

The causal organism is a short bacillus discovered by Bang in 1896. Nocard first isolated micrococci and bacilli, which, however, were not specific.

Bang slaughtered a cow from an infected herd showing premonitory symptoms; on opening the uterus with strict aseptic precautions, he found an abundant odourless, dirty yellow exudate between the uterus and the chorion. Cover-glass preparations stained with Löffler showed numerous small bacilli in pure culture, either free or heaped together into dense clumps, many of the latter being collected within distended cells; some of the single bacilli were as long as the tubercle bacillus, while many of the clumped bacilli were 1 μ or less, and resembled cocci; often several granules were detected in the body of the longer bacilli, and these readily took up the stain; the bacilli were immobile.

McFadyean and Stockman found certain differences in the behaviour of the bacillus towards oxygen and in its mode of growth in certain media. They were, nevertheless, dealing with the same disease.

Staining.—The bacilli are decolorised by Gram's method and its modifications, but they stain well with weak solutions of the ordinary aniline dyes—methylene blue, Löffler, or carbol-fuchsin. They are not acid-fast.

Cultural Characters.—Bang used agar-gelatine and raw serum. The English observers used agar-gelatine and special serum as the most suitable solid medium. The liquid medium consisted of equal parts of glycerine broth and special serum; the serum in each case was diluted, sterilised by heat, and rendered alkaline.

On solid media the larger colonies showed a brownish centre, greyish periphery, and dentated edge. The bacillus is essentially aërobic, although it will grow in the presence of very little oxygen; passing oxygen over the medium does not accelerate its growth. The optimum temperature is 30° to 37° C., and cultures started in the incubator grow readily at the temperature of the laboratory. Subcultures on agar slopes show a good growth in the presence of plenty of oxygen.

The chromogenic nature of the organism is shown by its growth on potato; a deep chocolate colour is developed, like the glanders bacillus. This is a marked characteristic of the abortion bacillus.

Under the microscope, clumping is well marked if the medium contains raw serum or has been sown with exudate, both of which possess the power of agglutinating the bacilli. Advantage is taken of the fact that the serum tends to lose its agglutinating power when heated.

Vitality is considerable, although the organisms are easily and quickly destroyed by weak solutions of the ordinary disinfectants. They are non-saprophytic; at the same time, if kept moist, they may live for a considerable period outside the animal body. The bacilli have been found alive after seven months in the laboratory. Bang recovered living abortion bacilli from the dark brown, putty-like exudate surrounding a mummified fœtus; the latter had been dead nine months. Their natural habitat is the pregnant uterus, but they soon disappear in the body of a non-pregnant animal.

The fact that a small percentage of cows abort a second or a third time in succession is probably due to reinfection and the absence of acquired immunity, not to persistence of the organism in the uterus.

The writer has, nevertheless, repeatedly observed abortion early in the next pregnancy, due to the animal having conceived too soon after the first attack, before the uterus has shed all the bacilli; this is likely to occur if copulation takes place on the first appearance of œstrum, about six weeks after the act of abortion; œstrum frequently appears much sooner in very early abortions.

METHODS OF INFECTION.

Natural infection occurs either by the vagina or by the mouth. Formerly the genital tract was considered the chief, if not the only, port of entrance for the bacillus, although Nocard pointed to the alimentary tract as a possible channel of infection. Less importance is now attached to infection *per vaginam;* it occurs from soiling of the gutter or bedding with the discharge from an infected cow, or from direct contact with the tail or hind parts. The curry-comb also conveys the discharge from one cow to another.

Pure cultures introduced into the vagina produced abortion in six to ten weeks; the same result followed the insertion of portions of the fœtal membranes.

Infection by the bull is of considerable importance; soiling of the penis and sheath takes place in serving an infected cow, and the virus is transmitted to other cows during the act of copulation; if conception occurs, abortion may not take place until pregnancy is advanced, by which time the part played by the bull is apt to be lost sight of. Bang, Penberthy, and other writers, have laid stress on the bull as a carrier of contagion, and many outbreaks have been clearly traced to this source. Here the bacilli reach the uterus through the mucous plug, which closes the cervix.

Infection by way of the alimentary tract was brought prominently forward by Bang in 1906; it is no doubt by far the commonest method. Food or drinking-water contaminated with the virus causes many outbreaks which admit of no other explanation. Soiled ingesta, such as fodder of any kind, grass, bedding, readily convey the disease. On infected farms discharges are conveyed by the boots and implements of attendants, apart from the careless disposal of contaminated fodder and litter; cows, of course, frequently eat their bedding.

Feeding experiments have yielded positive results in the case of the cow, sheep, and goat. Infection by way of the mouth explains the frequency with which abortion is met with in heifers served by a young clean bull just put to stud, removed from other sources of infection, and grazing on infected pastures; under these conditions, 30 to 40 per cent. may abort—*e.g.*, of 35 heifers in the writer's practice, 12 aborted; of 26 heifers, 11 aborted. In this method of infection the bacilli reach the uterus through the blood-stream.

It may be noted that abortion does not always follow experimental infection by whatever channel the virus is introduced; the introduction of living cultures into the blood-stream is, however, the surest means of conveying the disease.

The infection is usually introduced into a sound herd by the purchase of a cow which has recently aborted; the introduction of an infected pregnant animal is equally dangerous, whilst the risk attached to the use of an infected bull should not be lost sight of.

A farmer may send his cows to a neighbouring bull which has lately served an infected cow, and thus introduce the infection into his herd.

MORTALITY.

Mortality is nil. Abortion *per se* is seldom or never a direct danger to the life of the dam; its *sequelæ*, however, may be highly dangerous. Bacterial invasion of the uterus is more likely to occur after abortion than after normal parturition, owing to the catarrhal condition of the uterine mucous membrane and the frequency with which the fœtal membranes are retained. Septic metritis is often fatal, whilst a chronic catarrhal or purulent endometritis is a common sequel, which is mainly responsible for the unthriftiness or emaciation and for the sterility which are so often associated with abortion.

INCUBATIVE PERIOD.

The time which elapses between infection and the act of abortion varies greatly; it appears to depend less upon the channel of infection than upon the virulence of the organism and the stage of gestation. Individual susceptibility, or the possession of a certain degree of immunity, is also a factor. In experimentally infected animals, periods of 33, 56, and 230 days, are recorded; in ten cases the average period was 126 days (McFadyean and Stockman). In some experiments carried out by Bang an interval of six to ten weeks elapsed.

We have known cows infected during copulation abort from the fifth month onwards almost to full term. A few cows, not necessarily infected by the bull, carried their calves the full period, in spite of the presence of a typical abortion exudate teeming with clumped bacilli.

SYMPTOMS.

It follows that the disease may exist for months, during which no symptoms are shown and the general health is unaffected.

Premonitory symptoms appear a few hours to several days before the act—two or three days as a rule, exceptionally two or three weeks. They are those of approaching parturition, only less pronounced. At an early period of gestation they are absent. A cow expels a one or two months fœtus on the pasture, or behind her in the stall, in the form of a small tenacious jelly-like mass containing the embryo; this is often overlooked, and the recurrence of œstrum is the first sign which attracts attention. When pregnancy is further advanced, there is more or less œdema of the udder, filling and tenseness of the teats; if the glands are functionally active, the milk yield is usually suddenly diminished, and a few curds, or even a little blood, may be noticed. Some cows hold their milk, and the latter occasionally resembles colostrum. The vulvar labia are swollen, the mucous membrane congested, and a glairy mucus appears in increasing quantity, occasionally tinged with blood; the pelvic broad ligaments are somewhat relaxed, and uneasy movements or slight straining may occur later.

Immediately before or during the act, and for some time afterwards, a quantity of peculiar yellowish flocculent discharge is present; this is the highly infective uterine exudate which soils the tail and hind parts and the floor of the stall. This is followed by a thin yellowish, or more commonly a thick greyish, purulent discharge, the amount and appearance of which depends on the fate of the after-birth and the period of gestation.

The earlier the period, the more likely are the membranes to be voided with the fœtus. Retention is frequently seen after the fifth or sixth month. After early abortions the os uteri closes after a few hours, and the passage of a fine cannula is impossible; while even at the fifth or sixth month involution proceeds rapidly if the membranes have been expelled, and irrigation of the uterus requires to be promptly carried out.

COMPLICATIONS.

Dystokia is rare, but may occur, more especially with twins, when pregnancy is advanced. Milk fever is probably extremely rare, although we have had experience of four cases. Mastitis, retention of the placenta, and the danger of sepsis, have already been mentioned. A common sequel of abortion is a catarrhal or purulent condition of the uterine mucosa; this may occur even when the after-birth comes away normally.

MORBID ANATOMY.

Pathological changes are confined to the uterus and its contents. On opening the uterus aseptically before the act, the mucous membrane is merely in a state of catarrh, and shows little or no change to the naked eye. Under the microscope, a large number of catarrhal or shed epithelial cells and leucocytes are seen. A variable amount of typical exudate covers a portion or the whole of the mucous membrane and the chorion, partially separating the membranes from the uterus; the exudate is light yellow or yellowish-brown in colour, odourless, lumpy or flocculent, and the consistence varies from that of thin pus to soft cheese or putty; it is thick and putty-like, and dark brown or chocolate in colour, if mummification has taken place. The chorionic villi have a yellowish macerated appearance, and they are covered with exudate. The membranes have a thickened, gelatinous, or dropsical appearance, which is almost characteristic, due to subchorial œdema over a small or a large area; the œdema, however, is not always constant, and it may be so slight as to escape notice. Exceptionally, portions of the chorion are considerably thickened, indurated, and roughened, and brownish or pale green in colour; if extensive, the large tough mass is easily separated during manual removal of the placenta in the living animal. Putrefaction of the fœtus seldom or never occurs; it may be perfectly fresh or in various stages of mummification. If the latter is advanced, the membranes are closely adherent to the skin, and the whole mass is shrivelled and distorted, firm, dry, and dark brown in colour. It is said that this may occur in the short space of three months after the death of the fœtus.

The whole contents of the uterus are virulent—*i.e.*, the exudate, fœtal membranes, and the fœtus; the exudate teems with the bacilli, and is most dangerous; the chorion is infective over the cotyledons and over that portion of its surface covered with exudate; the subchorial œdema, the allantois and amnion and their contained fluids, are free of bacilli; the latter are sometimes present in the fœtal stomach, rarely in the fœtal heart blood. It should be borne in mind that the skin of the calf may be coated with exudate during and after birth, and its fæces may contain the organism.

DIAGNOSIS.

The differentiation of contagious from sporadic abortion is comparatively easy when the following factors are taken into consideration: history of the outbreak; introduction of an infected animal; use of a certain bull, or grazing on pasture on which cows have been known to abort; the succession of cases and rapid spread of the disease; character of the exudate; appearance of the after-birth; and, lastly, the exclusion of other causes.

The practitioner bases his opinion on the appearance of the after-birth and the character of the discharge which appears during or immediately after the birth of the fœtus. A microscopical examination of a smear from the fœtal cotyledons furnishes positive evidence of the existence of the disease. McFadyean and Stockman found that the membranes furnished the most reliable material for diagnosis. The characteristic clumped bacilli are, however, readily seen in the fresh exudate.

Bacteriological examination has advanced considerably of late, due to the classic experiments of the Board of Agriculture Committee. Three methods are available to detect the infection in pregnant animals and in those which have already aborted: (1) The agglutination test; (2) the "fixation of the complement" test; (3) abortin, or the reaction test. (1) and (2) are purely laboratory methods. Very encouraging results have been obtained, though they can scarcely be said to have emerged from the experimental stage. On the Continent, Holth and Grinsted have also done useful work.

The *agglutination* method depends on the power of the serum of affected animals, as compared with that of healthy ones, in agglutinating cultures of the bacillus free of serum. During the earlier experiments it was found that the serum of healthy animals never produced clumping in a higher proportion than 1 in 25, whereas the serum of infected cows possessed much higher agglutinating power, frequently 1 in 100. The strength of the serum varied, however, and definite or reliable results were not always obtained.

The "*fixation of the complement*" method is a very delicate test, and the technique is complicated. Briefly, it depends upon a substance circulating in the blood which is formed by the abortion bacilli in the uterus. The serum of the blood thereby acquires a specific affinity for abortion bacilli. This substance, called the "immune body" or "sensitiser," is also formed when an animal is injected with pure cultures. Blood for testing purposes is withdrawn from the jugular vein with an ordinary hypodermic syringe.

The *abortin or reaction* method is applicable in the field; the agent is prepared from cultures of the bacillus in the same way as tuberculin and mallein; a temperature reaction follows its hypodermic injection in the case of infected animals, beginning about the fourth hour and ending about the fourteenth. The temperature is taken every second hour from the fourth to the twelfth inclusive, and the reaction, as a rule, reaches its maximum between the sixth and the twelfth hour. A slight rise may occur in healthy animals, but a reaction over 104° F. is considered positive. Reactions occur in recently aborted animals for a certain period, also in those which have accidentally swallowed abortion bacilli, or which have been experimentally infected; on the other hand, certain bad cases of abortion have given negative results, in the same way as advanced cases of tuberculosis fail to react to a subcutaneous injection of tuberculin.

Abortin is not as yet recognised as a certain test; nevertheless, it has proved distinctly useful, and it has worked very well in some herds.

The value of the above tests is certain to be enhanced as improvements are effected in the technique.

PREVENTION AND TREATMENT.

Isolation and disinfection are indispensable as heretofore, and prompt measures should be enforced immediately the disease appears in a herd.

Test all pregnant animals, and isolate those which react and which have aborted. Those exhibiting suspicious symptoms are removed from the shed or meadow, and separate buildings and attendants are provided; on large estates isolation farms are very useful. If accommodation is lacking, animals which have aborted must perforce be placed at the lower end of the shed. Unfortunately, the traffic which takes place between diseased and healthy cows readily conveys the infection ; the carriers are the boots clothes, hands, and implements of attendants soiling the fodder, bedding, and hind-parts.

The fœtus, membranes, soiled litter, etc., should be mixed with fresh quicklime, and either burned or buried in arable or inaccessible ground ; the floor and sides of box, the tail and hind-parts of cow, the attendants' barrow, fork, and boots, must be cleansed and disinfected. If the abortion has occurred at grass, the turf should be lifted and burned, or sprinkled with lime and railed off; if in the shed, the stall and gutter are disinfected immediately, also the whole shed. Floors and channels should be flushed down with Izal or other suitable disinfectant, and the rear of the stalls may be sprinkled daily with sulphate of copper or washed down with a 4 per cent. solution of this agent. If more cases occur, the hind parts should be washed or sprayed, and the vagina of every cow syringed twice or thrice a week ; suitable pessaries may be inserted with the fingers on alternate days. If syringing causes irritation and excessive straining in young or pregnant animals, pessaries alone may be used.

The bull seldom exhibits a discharge ; if so, the prepuce is irrigated daily, and he is not put to stud for four to six weeks. Ordinarily, a weak antiseptic is injected once a week, and the long preputial hairs are clipped short. Pessaries of small diameter are useful. It is well to keep a separate bull for cows which have aborted, and to discontinue the use of a co-operative bull, if such exists.

Reverting to the *aborted animal*, under ideal conditions the uterus is irrigated frequently by the practitioner, as long as the os remains patent, especially if the membranes are retained. It should be possible to flush out the cavity at least three times at intervals of a day or two ; on the other hand, treatment may be limited to one injection, especially in early abortions, where the introduction of a small cannula is often difficult or impracticable. The after-birth is frequently retained, and its complete removal is indicated as soon as possible. Manual removal, however, is not always easy, and it is well to wait for several days if the chorionic villi are firmly adherent, the membranes friable and easily torn, or a little blood is noticed. The uterus, meantime, is irrigated freely with a mild antiseptic, and pessaries are left *in situ.*

The attendant syringes the vagina once or twice daily for two or three weeks, or longer if a discharge is present. The operation may be repeated on the first appearance of œstrum, when a little purulent discharge may be present.

The records which we have kept on abortion isolation farms show that repeated irrigation of the genital passages lessens the danger of sepsis whilst sterility or abortion in the next pregnancy is less likely to occur. There is also less chance of the infection being conveyed to healthy animals. At the same time, treatment on these lines is not always essential for the eradication of the disease. Subsequently an animal which has aborted is not returned to the herd for three to eight weeks, and three months are allowed to elapse before she is served by the bull.

It is well to treat every case of abortion as infectious, in the absence of positive evidence to the contrary.

New arrivals in a herd, about which little is known, should be isolated, and either kept under observation or tested.

The practice of ultimately selling or fattening aborted cows may be necessary in some small herds ; it depends chiefly on the necessity which may exist of keeping up the milk-supply, the period of gestation, the condition of the cow, her milking propensities, and the chances of sterility supervening. It is well to retain the aborted animal if possible, for, owing to the active immunity which is often set up, she becomes a distinct asset to the owner ; more important still, there is less need for the introduction of susceptible animals, which keep up the virulence of the infection.

The administration of carbolic acid, it may be mentioned, either *per os* or subcutaneously, is quite useless, either as a curative or a preventive. We have used it in the food in large and small doses for long periods, extending to twelve and eighteen months. Extensive trials in various herds produced no result. Carbolic acid and other vaunted " cures " are frequently used when the disease is dying out ; in this way they acquire a fictitious value.

Many *antiseptics* are employed for the genital tract ; the essentials are : no irritation, moderate price, sufficient germicidal power, and comparatively non-poisonous nature. We prefer lysol ½ to 1 per cent., or septoform ½ per cent., for the uterus and prepuce ; bacillol is effective and cheaper, but may produce irritation if the solution exceeds ¾ per cent. It is useful for handling large numbers of animals. Creolin, carbolic acid, permanganate of potash, chinosol, corrosive sublimate, Izal, and other agents, are used. Rather stronger solutions may be used for the vagina than for the uterus and the prepuce. Izal is to be preferred, as it is less likely to taint the milk than creolin or carbolic acid. It is also the best for external use in cowsheds. The genital mucosa of heifers and young cows is easily irritated, hence caution is necessary, so as to avoid the use of too strong injections.

IMMUNITY AND PREVENTIVE INOCULATION.

The *immunity* conferred by one attack may be and usually is a lasting or active one; on the other hand, it may be insufficient to protect the animal against subsequent exposure to infection.

Experimental infection in various ways has proved that an active immunity does occur, whilst experience in infected herds has shown that a large proportion of the cows abort once, and once only, although exposed to infection for long periods.

Active immunity is, however, less likely to occur in herds in which the disease has appeared for the first time. Where abortion had existed for a number of years, Bang found that one attack usually served to confer immunity; he attributed this to attenuation of the virulence of the organism.

We have had a similar experience in English dairy herds; a few instances occurred in which a cow aborted the second time in succession, but the act did not necessarily take place at a later period of pregnancy; it sometimes occurred much earlier. According to Stockman, the bacilli are probably all destroyed in four to eight weeks in the system of non-pregnant animals, and a second abortion is not due to persistence of the bacilli in the uterus.

However this may be, there is no doubt that reinfection does occur.

Inoculation.—The possibility of conferring immunity by prophylactic injections of a vaccine or a serum was suggested for the first time by Bang in 1896. Ten years later, at Liverpool, the same observer described a few inconclusive but encouraging experiments with both dead and living cultures of his bacillus. About the same time experiments on somewhat similar lines were commenced in this country on behalf of the Board of Agriculture; the Committee appointed found that "the most hopeful line of inquiry seemed to be the production of immunity by inoculation of large doses of pure cultures."

It was recognised that the first essential was to grow the bacilli freely and easily in bulk; this was soon accomplished, and preliminary experiments were undertaken in the laboratory of the Board. Dead cultures called "abortion cure" have been tried to a certain extent as a curative in pregnant animals known to be infected. Owing to the insidious nature and the prolonged course of the disease, this curative method is not likely to come into general use; nor is it recommended in animals in which pregnancy has advanced beyond the fifth month. Infection may, of course, occur at any time, and the danger period extends over most of the period of pregnancy—at least seven and a half months. On the other hand, *anti-abortion*, or large doses of virulent bacilli, was found to be harmless when injected into non-pregnant animals, while it effectually protected them against the introduction of enormous doses of the virus when the animals became pregnant.

Field trials, carefully carried out, have shown that this method of conferring immunity is likely to be followed by good results.

The technique is no doubt being modified and improved; in the earlier trials a rather large dose, amounting to 150 c.c., was injected under the skin of the neck or behind the elbow, with strict aseptic precautions. Intravenous injection may be followed by rather alarming symptoms; it is, nevertheless, the surest method of infecting an animal artificially. A period of six to eight weeks is laid down as the likely interval which should elapse between the inoculation and the time the animal goes to bull; this allows ample time for the elimination of the bacilli from the system.

In practice it is not always possible to adhere strictly to this period. If an inoculated animal proves temporarily sterile, a second injection is given when she arrives in good breeding condition. This procedure is based on the fact that vaccination is unlikely to confer immunity for an indefinite period. The method is also of service in increasing the immunity possessed by cows which have already aborted, but which have not yet conceived.

It is recommended to inoculate all non-pregnant animals, including heifers at a breeding age, the dose being the same for all.

A number of promising experiments with anti-abortion are being carried out in infected herds at the present time. It is highly probable that immunisation on these lines, perfected in all its details, has a great future before it in controlling the plague, and in finally eradicating it from our midst.

CONTROL.

It has been recognised for many years that the sale of infected cows, either pregnant or recently aborted, is mainly responsible for the spread of the disease. Herds which are self-supporting, in that they breed and rear their own stock, together with those which are able to recruit from sound herds, are most likely to keep clear of the disease.

Under ordinary commercial conditions, however, the purchaser is compelled to take unwarrantable risks by purchasing in the open market. The practice of selling cows which have recently aborted has been all too common in the past, and it is evident that private effort is incapable of dealing with the disease. State control has been advocated for some time, and the need for it has been gradually brought home to British stock-owners. Experts are of opinion that the time has arrived when the disease should be scheduled, having regard to the serious and increasing economic loss, and the ease with which a positive diagnosis can now be made. Norway is probably the only country in which legal measures are in force; notification is compulsory, and restrictions are placed on the movement of infected animals.

The Departmental Committee of the Board of Agriculture, in their second report, dated June 25, 1910, after weighing all the evidence laid before them, recommended, as a preliminary measure, that "epizoötic abortion should be included in the Diseases of Animals Act requiring—

"(1) Compulsory notification of suspected cases of the disease;
"(2) Veterinary inquiry to establish the existence of the disease on any particular premises; and
"(3) Temporary isolation and restriction on the movement of any cow that has recently aborted."

I. INFECTIOUS ABORTION IN OTHER ANIMALS.

There is little doubt that infection is responsible for most of the abortions in the domesticated animals. Recent investigations point to the existence of a specific organism peculiar to each species, which is not transmissible to other species.

By "infectious abortion" is meant that form which occurs as an independent disease due to a specific cause. It does not include the form which is commonly observed in all animals during the course of acute infectious diseases. This is known as *sporadic* or *accidental* abortion, reference to which has been made in connection with the disease in cows. Comparatively little is known at the present time regarding infectious abortion in animals other than the cow. The latter is housed for long periods, the genital organs are weakened by intensive breeding and management, and parturition occurs as a rule all the year round. The chances of infection are thereby increased.

In the *mare*, on the other hand, foaling is confined to one period of the year, and there are fewer opportunities for the spread of the disease. The etiology of infectious abortion is still obscure in sheep, goats, and pigs. A vibrio has, however, been found in ewes, which produced the disease in other ewes by experimental inoculation (McFadyean and Stockman). Further investigation is also required in the mare to establish the identity of Ostertag's streptococcus. The symptoms, treatment, and control in the various animals are very similar to those in the cow.

It is proposed to deal more particularly with the disease in the mare.

Enzoötic Abortion in Bitches.—This occasionally occurs, especially in the smaller or more delicate breeds. In some cases all the bitches in a kennel abort before the fœtuses have become covered with hair; in other instances, during the last week immediately before the term (sixty to sixty-six days) is accomplished; in still other instances, the fœtuses die towards the natural period of utero-gestation, and in consequence many bitches are lost from puerperal septicæmia.

Enzoötic Abortion in the Cat.—This is a much commoner condition in the cat than in the bitch. Every animal in a cattery may abort almost simultaneously. The etiological factor in both cases is unknown, but it seems of infectious origin. Further observation on this point is necessary. The *rabbit* also suffers from an enzoötic form of abortion.

Enzoötic Abortion in Birds.—The precipitous expulsion of the egg before the calcareous material has had time to develop, or, rather, to be deposited, occurs sometimes as an enzoötic in various avian species. Its cause is unknown, but nevertheless it is well to mention the fact, as being interesting from a comparative pathological point of view.

II. INFECTIOUS ABORTION IN MARES.

Infection has been recognised as an important factor in various parts of the world during recent years. In France, Germany, Great Britain, and in other countries, serious outbreaks have from time to time occurred; whilst in both American continents the ravages of the disease assumed alarming proportions between the years 1886 and 1892. In some States over 75 per cent. of all classes of mares are said to have aborted, and some breeders reported the loss of every foal of the season.

In 1891, in the State of Buenos Ayres, contagious abortion was recognised apparently for the first time.

Breeders suffered immense losses, and the disease spread over large tracts of country under very varied conditions.

No such extensive outbreaks have been recorded in Europe, although Guillerey* had 87 abortions amongst 97 mares. An interesting outbreak occurred in the Punjaub in a stud of 80 donkey brood mares. During the first year (1907), 29 mares aborted; in 1908, 13 aborted; in 1909, 9 aborted. The period of gestation varied from four months to full term. The clinical record points clearly to contagion, although no post-mortem examination was made. Nearly all the mares which aborted during the first year failed to abort again; only two mares aborted a second time.†

In France, Desoubry‡ had experience of the disease in a stud of pure-bred mares. In Illinois, Williams § met with numerous outbreaks resulting in the almost total loss of the foals each season. The abortions were frequently eaten by sows with impunity, whilst the cows were free from infectious abortion.

Susceptibility.—As a rule, if contagious abortion is raging amongst the cows, mares on the same premises are unaffected, and *vice versâ.* Exceptionally, abortion attributed to contagion has been observed in both species at one and the same time. This appears to have been mere coincidence, no evidence having been brought forward to show any causal relationship between the equine and bovine diseases. Bang conveyed the latter to mares by experimental inoculation, but it has never been observed naturally, whilst Ostertag's streptococcus has been found in the mare only.

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* Hutyra and Marek, "Spezielle Pathologie und Therapie der Haustiere," edit. iii., vol. i. (Seuchenhaftes Verwerfen).

† Webb, *Journ. of Comp. Path. and Therap.*, p. 289, 1909.

‡ Desoubry, *Réc. de Méd. Vét.; Journ. Comp. Path. and Therap.*, p. 154, 1909.

§ Williams, W. L., "Veterinary Obstetrics."

It will be remembered that *Bang's bacillus of cattle abortion* is transmissible experimentally to other animals; the equine disease, on the other hand, appears to be non-pathogenic to other species. Pending further bacteriological research into abortions other than bovine, no ordinary precautions should be neglected to prevent the possible spread of infection from one species to another.

Etiology.—Ostertag,[*] in 1901, claimed to have discovered the causal organism in a short streptococcus, present on the surface of the chorion, in the blood of the fœtal heart, in the fluid of the thoracic cavity, and in the stomach of still-born foals. In the body of the fœtus the streptococci were present in pure culture in seven cases; on the chorion, however, they were always associated with other bacteria, owing, no doubt, to the rapidity with which organisms gain access to the uterus through the open os. Experiments were made with the after-birth and discharges from mares which had aborted. These, together with pure cultures of the streptococcus, produced the disease in pregnant mares in which the same organism was subsequently demonstrated.

The staining reactions were similar to Bang's bacillus; the latter, however, was absent in every case of abortion in mares. *Cultures* on serum-agar showed a very scanty growth; stab cultures showed a thread-like appearance from top to bottom of the tube. In serum-bouillon a uniform cloudiness appeared after two days, and the colonies sank to the bottom of the tube forty-eight hours later.

Period of Gestation.—The majority of the cases occur between the fourth and the eighth month of pregnancy. Nevertheless, infectious abortion may attack mares, like cows, at any stage from conception to full term.

"Invisible abortion" is not uncommon during the first few weeks of gestation. Up to two months or more the abortion is seldom observed, especially out of doors, and the recurrence of œstrum is often the first sign which attracts attention. Guillerey noted abortion, usually between the fourth and the seventh month; in one-third of Kongé's[†] cases it occurred during the sixth month. Ewes are said to abort most frequently during the fourth month; sows between the eighth and the twelfth week. When pregnancy is far advanced (ninth to eleventh month), the foal is usually still-born, although perfectly fresh; if alive, it is weakly, and it may suffer from joint-ill. It seldom survives more than a few days.

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[*] *Monatshefte für praktische Tierheilkunde*, vol. xii., p. 385.

[†] Hutyra and Marek, "Spezielle Pathologie und Therapie der Haustiere," edit. iii., vol. i., p. 744.

Incubative Period.—This varies, as a rule, from ten days to three weeks. Numerous instances are recorded in which the introduction of a recently-aborted mare was followed by other cases in two to three weeks. If the mare aborted on the premises, ten days was sufficient, under favourable conditions, to transmit the infection and produce abortion in pregnant animals. A period of twenty days sometimes elapsed in the natural infection, whilst the same interval occurred in one mare after intravenous injection of pure culture (Ostertag).

It seems feasible to suppose that the comparatively short incubative period is due to the loose placental attachment in the mare.

Methods of Infection.—It is very probably the case that infection occurs most largely by ingestion, as in the cow, although vaginal infection may take place. The infection may be direct or indirect.

The food, water, bedding, and hind-parts of pregnant mares are soiled by the attendant's hands, boots, clothing, or stable utensils. The virus is most commonly conveyed by men who have handled an aborting mare during or after the act. At the same time, boxes in which an abortion has taken place convey the disease to fresh occupants.

The stallion appears to play a less important rôle even than the bull. The explanation probably lies in the fact that both the incubative period and the breeding season are of short duration. Mares which abort early are usually served again and conceive towards the end of the season, when other mares are in foal; on the other hand, those which abort at a late period are not bred from in most countries till the spring.

Six weeks appear to be ample time to allow the organisms to pass out of the system. It is exceptional for a mare to abort a second time, no matter at what period of gestation the first abortion occurs.

Symptoms and Course.—Premonitory symptoms are usually absent. A mucous discharge, especially if tinged with blood, usually points to the death of the fœtus. It may be preceded by colicky pains, and abortion soon follows. Delivery is easily and quickly effected, although dystokia may occur in advanced pregnancy, particularly in the case of twins.

A rise in temperature with catarrh of the genital tract and constitutional disturbance is said to distinguish contagious from accidental abortion; the mare is dull and listless, and fever, loss of appetite, and a dark, reddish-yellow colour of the visible mucous membranes are present for two or three days after the act. The vaginal discharge may continue for seven to ten days, or even longer. These symptoms are, however, not constant, and no signs of ill-health may be shown. In some outbreaks grave symptoms have appeared, mostly at a late period of pregnancy, when the after-birth has been retained. Septic infection occurs very rapidly under these conditions. Guillerey noticed inflammation of tendon sheaths and joints, especially the hock, and mastitis has also been described. The writer has observed arthritis of the hock and mastitis to follow abortion in cows. During the early stages of gestation the membranes are usually expelled with the fœtus, and no untoward sequelæ occur. Later on the after-birth is more likely to be retained than in normal parturition. This appears to be mainly responsible for the serious results which are apt to attend infectious abortion in mares. At the same time, the catarrhal condition of the uterus favours bacterial invasion, apart from a retained placenta.

Differential Diagnosis.—Sporadic abortion usually passes off uneventfully, and the cases are few in number. There may be the history of an accident, and it may be possible to eliminate the question of contagion. In the contagious form cases commonly follow the introduction of an infected animal; they occur in more or less rapid succession, with occasionally grave, even fatal, results. A mixed infection of the genital tract rapidly takes place; hence little useful information is likely to be obtained by making smears of the vaginal discharge. Few systematic attempts to determine the etiology of the various outbreaks have been made. Bang's method applied to the mare should yield definite and valuable information. The aseptic opening of the pregnant uterus, in which the os was still closed, would mean killing the mare before the act. The absence of premonitory symptoms and financial considerations militate against such a procedure; yet the difficulties which exist are not insurmountable.

Control and Eradication.—The broad principles of isolation and disinfection are similar to those described in the cow. In the present state of our knowledge it is well to treat every case of abortion as contagious. Some British breeders make a practice of isolating every mare which arrives from another stud, or which may have been exposed to infection of any kind *en route*. The quarantine is carried out on different premises, and it has proved very effectual, in the writer's practice, not only against abortion, but also against strangles and other scourges.

The mare should remain in the box in which she aborts, and a separate attendant should be provided, whilst thorough disinfection should be carried out. The healthy mares should be removed from the immediate neighbourhood. It is customary to irrigate the uterus once with an antiseptic in every case of abortion; if due to infection, the irrigations should be carried out daily for several days with lysol ($\frac{3}{4}$ per cent.), hydrogen peroxide, or other reliable non-irritating antiseptic. A simple gravitation apparatus acts well, and tends to prevent straining. The vagina may be syringed for ten days, or longer, if there is any discharge. It is well to allow three to six weeks to elapse before the mare is put to the horse; the longer interval is preferable—*i.e.*, about the third œstrum. The external genitals of the stallion should be examined, and treatment may be indicated on the same lines as in the bull. The writer has been asked to ascertain the mares that were pregnant in certain outbreaks in which the abortions occurred at an early period. Needless to say, familiarity with the handling of the genital organs *per rectum*, both in the pregnant and non-pregnant state, is essential for the carrying out of such work. Precautions should also be taken to prevent the operator conveying infection from one mare to another.

NOTE BY EDITOR.

In the Report of the Veterinary Director-General and Live Stock Commissioner, Department of Agriculture, Canada (1911), we find that attention is drawn to a contagious form of abortion in mares. This affection, in 1910, occurred in districts north, east, and south of Calgary, Manitoba, and along the course of the Red Deer River, and caused heavy losses to the horse-breeders in these districts. It was investigated by Mr. A. Watson, and so serious were the losses induced by the disease that the horse-breeders called a meeting at Didsbury, Alta., to discuss the measures for the suppression and prevention of the malady. As the result of the precautions and preventive measures adopted, very few cases of abortion in mares were recorded for the following year.

CONTAGIOUS GRANULAR VAGINITIS OF CATTLE

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Synonyms.—Infectious vaginal catarrh ; Colpitis granularis infectiosa ; Granular venereal disease ; Vaginitis verrucosa ; Vaginitis et metritis follicularis infectiosa.

French : Vaginite granuleuse de la vache.
German : Knötchenseuche ; Scheiden Katarrh.

Definition.—A highly infectious disease of the genital organs, characterised in the female by a swollen inflammatory condition of the vaginal mucous membrane, which is usually covered with small nodes or granules spreading upwards and forwards from the clitoris.

GENERAL REMARKS.

Youth is the chief predisposing factor, and females are more susceptible than males. Thus it follows that heifers and young cows are most largely affected, and they also prove most intractable to treatment.

The greatest importance is attached to the disease on the Continent, owing to the fact that many affected cows fail to conceive, or, having conceived, they fail to carry their calves the full term, whereby heavy losses are incurred.

On the other hand, it must be admitted that the malady, even in the acute form, has caused little, if any, damage in certain herds.

Again, the so-called pathognomonic nodules on the mucous membrane may be absent altogether or hidden in the swollen and infiltrated mucosa. They vary as to size, shape, colour, and also as to the period at which they make their appearance.

While it is possible that more than one disease has been described under this heading, it is certainly the case that atypical forms are met with.

HISTORY.

Contagious granular vaginitis has doubtless existed in most civilised countries for many years.

It attracted attention in Switzerland about the end of last century, while of late years observers have found it to be prevalent in Austria and Germany, and to a less extent in Italy, the United States, and Canada. Many writers have recorded their observations—*e.g.*, Isepponi in 1887, followed by Ehrhardt, Ostertag, Hecker, Trommsdorf, Hess, Fröhner, Raebiger, De Bruin, Ladanyi, Saccani and Tosi, and W. L. Williams.

In 1903, the Swiss Veterinary Surgeons' Society instituted an investigation and compiled certain statistics.

In 1909, Hess and Albrechtsen brought the disease prominently forward at the Ninth International Veterinary Congress at the Hague.

In this country the chief veterinary officer of the Board of Agriculture published a report on the disease in 1910, and opened the discussion on a paper on the subject read before the National Veterinary Association in the same year.

OCCURRENCE.

While it is probably the case that no country is free from the disease, it is widely distributed over the continents of Europe and North America.

In some districts not a single sound herd was found, and 60 to 90 per cent. of the animals showed more or less characteristic lesions.

In the United Kingdom several outbreaks of contagious vaginitis, attributed to this disease, have been observed. A benign granular condition of the vagina exists in many herds, affecting a large proportion of the heifers and young cows. This is probably a mild, non-virulent form of the Continental disease.

ETIOLOGY.

Ostertag claimed to have discovered the specific organism in a short streptococcus present during the first stages in the purulent discharge, and in the deeper layers of the vaginal mucous membrane.

Its causal connection with granular vaginitis does not as yet seem to have been clearly established, judging by the evidence contained in the published accounts of the disease. Blahn stated that Prowazek's bodies are invariably present in the follicular nodules; their etiological significance is, however, uncertain.

MORBID ANATOMY.

The lymph follicles in the mucosa share in the general irritation. They are swollen, and appear on the surface of the mucous membrane as firm nodules heaped up round the clitoris, and radiating upwards and forwards towards the vaginal wall. They seldom reach the os uteri or the uterine mucosa.

Thoms made sections and published microphotographs of the mucous membrane showing the swollen follicles and the cellular changes in the mucosa and submucosa.

METHODS OF INFECTION.

The infection is usually transmitted by copulation, and to a less extent by direct contact of diseased with healthy animals; also by fodder or litter soiled with vaginal or preputial discharge; the hands and clothes of attendants and the grooming utensils may also be vehicles of contagion.

Calves may be infected from the mother at birth or shortly after, and granular lesions have been observed in calves two weeks old.

The disease in the acute form is readily conveyed from animal to animal by experimental inoculation. For this purpose sterile cotton-wool swabs on wire are used, but the finger, thermometer, or vaginal speculum are quite effective in this direction.

Bulls have been known to infect cow after cow without showing any visible signs of the disease themselves.

PERIOD OF INCUBATION.

The average period of incubation is from two to six days.

SYMPTOMS AND COURSE.

The disease may assume an *acute or virulent* form and a *chronic* form. The acute form may become chronic, or the chronic form, so called, may be primary and unattended by any visible inflammatory phenomena.

In the *acute form* there is swelling of the vulvar labia; also swelling, congestion, and tenderness of the vulvo-vaginal mucous membrane, with increased mucus; pain on manipulation; a straddling gait; whisking of the tail; and arching of the back during and after micturition.

On parting the labia, the mucous membrane is red and injected, more or less streaked, and covered here and there with a greyish-white flaky pus.

A day or two later, firm rounded nodules the size of a millet-seed or thereabouts appear round the clitoris. These soon extend upwards to the roof and forwards along the lateral wall of the vagina for several inches. Exceptionally they reach the os uteri or the uterine mucosa.

They are usually arranged in parallel rows on the ridges of the mucous membrane, and form clusters round the clitoris. At first dark red in colour, they become bright red, pale, and finally blanched, fading most quickly near the upper commissure and laterally, and persisting longest at their starting-point.

Hess describes a form in which red elevated dots appear on the mucous membrane, followed by vesicles and ulcers, and associated with a tough muco-purulent exudate covering the posterior genital canal. The latter soon disappears, and numerous red granules are then seen studded over the vulvo-vaginal mucous membrane radiating from the clitoris.

Another atypical form is described in which no nodules or ulcers are present, but instead there is an acute catarrhal inflammation, with a quantity of muco-pus.

Ladanyi, again, records cases in which yellowish-white spots appear on the vaginal mucous membrane in place of the nodules.

The *chronic form* is characterised, for the most part, by the absence of discharge and irritation, and the presence of a variable number of nodules, varying in size from a small pin's head to a hemp-seed. At the upper commissure they approach close to the cutaneous margin ; inferiorly, the mucosa is smooth as far as the clitoris, and not uncommonly contiguous nodules become confluent, forming linseed-like elevations.

The granular condition may remain unsuspected for a long period, owing to the fact that it is not associated with economic loss.

In this country, at any rate, it appears to do little, if any, harm. After copulation, however, exacerbations may occur. The mucous membrane is red and angry-looking, especially round the nodules, and a slight exudate may be present in the form of whitish shreds or flakes of pus. Occasionally a little pus may be squeezed out of the clitoris by pressing it from below. Inoculation gives negative results.

Hess's observations are important here. They show that the activity of the virus disappears with the onset of chronicity.

Bulls are less severely affected as a rule. The prepuce is swollen, and shows a muco-purulent discharge, while the penis is redder than normal, and nodules may be present on the mucous membrane of the glans penis.

In acute cases there is disinclination to copulate, and the act is performed slowly and accompanied by the discharge of a little blood.

Bulls apparently healthy appear to be capable of conveying the infection from one cow to another.

DURATION.

The *acute* form lasts from four or six weeks to three months ; the mild, benign, or *chronic* form may continue indefinitely. We have observed somewhat extensive granular areas in the same animals for several years. They tend, however, to fade and gradually disappear.

In old cows the mucous membrane has normally a pronounced yellowish tint, and it is exceptional to find other than insignificant lesions. The vagina is often granule-free.

It is worthy of note that the granules are hidden during periods of physiological congestion—*e.g.*, ante- and post-partum and, to a less extent, during œstrum. They reappear later when the swelling subsides.

DIFFERENTIAL DIAGNOSIS.

The disease must be distinguished from *vesicular vaginitis*. The development of small, firm, circumscribed, and discrete elevations in the region of the clitoris, associated with acute catarrh, is characteristic of contagious granular vaginitis. In atypical cases, however, the differential diagnosis is by no means easy. The nodules may be absent or develop at a later stage, vesicles or ulcers, or both may be present, while a thick purulent exudate may line the posterior genital canal, with swelling of the vulvar labia.

These forms, associated with vesicles and slight superficial ulceration, are indistinguishable from the disease known as *vesicular vaginitis*, vesicular exanthema, or " bull-burn."

The latter is quite a benign and common disease in this country, and it resembles the granular disease in many respects. It will suffice, therefore, to point out certain important differences and resemblances.

They agree in that both are essentially venereal in character, and very contagious. The incubative periods, methods of infection, and liability to secondary and even tertiary attacks, are also practically the same.

Vesicular vaginitis, however, affects bulls more severely. The preputial discharge is more profuse, and vesicles and pustules have been noticed on the penis. The duration of the disease is decidedly shorter in both sexes; no nodules are ever seen in uncomplicated cases, and there is no chronic stage. In our experience, spontaneous recovery occurs in females in two to three weeks, and no local treatment appears to be necessary. The dirty grey purulent exudate, with which is associated a mucopurulent discharge, is sometimes mottled with blood, especially if the parts have been frequently handled; while the mucous membrane to which it is more or less adherent is swollen, intensely red and angry-looking, and bleeds readily on manipulation. The clitoris is particularly swollen, and the exudate clings to it to the last. The vesicles occur during the first stages, and, being very evanescent, are seldom observed. They are followed by very slight small pits, ulcers, or abrasions, which are quite commonly present under the purulent lining. The ulcers, however, cannot always be observed. The exudate usually quickly disappears, in about seven days, especially if no irritant treatment is adopted. A feature of the vesicular disease is that conception and natural infection may take place at one and the same time; in other words, an infected bull may impregnate a cow and inoculate her at the same time by way of the penis. We had one outbreak in which the same bull, with slight preputial swelling and discharge, served seven cows within ten days. Each cow developed the vesicular disease, and stood to the first service. Finally, in the vesicular disease constitutional symptoms are slight or absent, particularly in females, and the milk-supply is unaffected.

Reverting to the granular affection, the chronic course and the absence of ulceration serve, as a rule, to differentiate it.

Complications, however, occur if the *double infection* by any chance occurs in the same animal. We have observed the acute vesicular disease in cows showing "chronic" granular lesions. The coincidence of two venereal diseases in the human subject has also been noted. It is just possible—int his country, at any rate—that both granular and vesicular vaginitis are different phases of the same disease, and further investigation in this direction is undoubtedly required.

SEQUELÆ.

Sterility and Abortion.—Opinions are divided in regard to the effect of contagious granular vaginitis on the reproductive organs. This difference of opinion arises partly from the fact that the pathology of the disease has not yet been cleared up. It is thought to spread by contiguity from the vagina to the uterus, and even to the ovaries. On the other hand, the uterus may be infected by the penis during copulation. Nodules on the uterine mucous membrane have been noticed by Continental observers, who hold that the catarrh of the vagina extends to the uterus, and produces abortion in much the same way as Bang's bacillus, when infection occurs through the genital tract.

There is reasonable ground for believing that sterility is a common sequel. Statistics show that the pregnancy figure was very low in severely affected herds, while the number of ovarian and uterine diseases was larger than usual.

The coexistence of sterility or abortion, or both, and contagious granular vaginitis appears to be quite common. All three are widespread diseases, and one may be superimposed on the other without any causal relationship existing between them.

The elimination of Bang's bacillus is required in herds affected with both vaginitis and abortion before establishing any definite relationship between them.

It is significant to note that abortion in the United Kingdom is nearly always due to the bacillus of cattle abortion (Bang), whilst certain outbreaks of contagious granular vaginitis have occurred on the Continent with impunity so far as either sterility or abortion is concerned.

Hess, on the other hand, states that out of every 100 cases of sterility, 58 are due to the disease under consideration.

THERAPEUTICS.

Isolation and disinfection are essential in this as in other contagious diseases. Attention is particularly directed to the cleansing and disinfection of the sheds, animals, and utensils, cessation from breeding, and the segregation of the affected members of the herd.

As regards local treatment, success seems to depend less upon the form and nature of the particular medicament than upon the thoroughness, care, and time expended on carrying out the details.

As a rule, warm antiseptic irrigations should be employed once or twice daily at first, followed by powders, ointments, or pessaries, particularly the latter. These include chinosol and a proprietary preparation of mercury and iodine known by the trade name of " bissulin."

After washing out the vagina with a mild astringent and antiseptic, the pessary is introduced with the fingers, rubbed gently over the inflamed area, and then left *in situ*. Suitable pencils are also used for bulls, after clipping the preputial hairs, syringing the sheath, and washing the surrounding skin with a suitable disinfectant. The strength of the various solutions, etc., should be carefully regulated, to avoid irritation.

Six weeks' treatment is sufficient for most animals; others may require three months. The discharge ceases, the inflammation subsides, but a certain number of granules may remain for a long time with impunity.

It is advisable to keep a separate bull for infected cows, and to allow no copulation to take place during the period of treatment, as the sexual act greatly increases the existing irritation. Attention should be directed to the healthy members of the herd, and to the disinfection and isolation of new arrivals. The use of a co-operative bull is contra-indicated.

As in contagious abortion, it is well to irrigate both the vagina and prepuce at the time of copulation. The penis may also be sprayed immediately after the act with a garden syringe.

There is no State control, and no restrictions are in force in the public markets; but efforts are directed, particularly in Germany, to educate the stock-owner and to secure his co-operation in stamping out the disease over large areas. Voluntary effort is generally successful and comparatively inexpensive, when the various public bodies combine and go systematically to work. Difficulties are apt to occur, however, with reference to those herds in which no losses appear to occur from the disease.

GANGRENOUS VAGINITIS IN CATTLE

By E. WALLIS HOARE, F.R.C.V.S.

BERGMANN investigated a virulent form of vaginitis that attacked nearly all the cows on an estate in Kristianstadt (Sweden) during the autumn and winter of 1902.* It was always associated with parturition, and young cows were those generally affected. Out of 100 cows which had calved, 48 heifers and 13 of the older cows were affected, and of the latter 6 had only borne one calf and 5 two calves previously. There were forty-eight cases of the disease among the cows with their first calf, and only one case among the cows which had borne more than five calves. The affection was found to be much more virulent in primiparæ than in animals which had calved twice or more; it attained its greatest height in October and November, and by March, 1903, it had disappeared. But in the autumn of 1903 cases of the disease were again observed; fewer animals were attacked; the malady was of a milder type, and the cows which suffered most severely during the previous year were again affected. During the following year (1904) no cases were observed. An outbreak of the disease occurred simultaneously with the above on a neighbouring estate belonging to the same owner, and Bergmann observed sporadic cases every year, and also received reports of the occurrence of the malady from various parts of the country.

Etiology.—The cause of the disease was proved to be the bacillus of necrosis.

Attempts to produce the disease by introducing necrotic fragments of tissue beneath the vaginal mucous membrane of healthy cows were not successful. Bergmann examined the labia of the vulva and also the vagina of cows soon after calving, and found these structures more or less œdematous, and the mucous membranes of these regions showed small lacerated wounds with red bases; these were more marked in primiparæ, and in the inside of either labium towards the top a fairly large lacerated wound was usually present. This observer was of opinion that infection took place through the medium of these wounds, but the source of the infection was not determined. Two cows purchased from a farm where no cases of the disease existed contracted the disease, although one of these calved on the pasture, and the other was only brought into the cattle-shed when the first signs of parturition were observed.

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* Bergmann, *Fortschritte der Vet. Hyg.*, April, 1906; translation in *Journal of Comparative Pathology and Therapeutics*, September, 1906.

The infection did not appear to arise from coition, as the affected cows were served by five different bulls, and none of the latter showed any discharge from the prepuce.

One attack of the disease did not confer immunity, as even the cows that suffered from a severe type of the affection developed the malady in the following year.

Lesions and Symptoms.—After delivery the vagina and labia of the vulva showed considerable œdema, and a fœtid vaginal discharge was observed, trifling in extent at first, but gradually increased, and in from five to ten days it became profuse. Abrasions or wounds were present, especially in the upper part of the vagina; these assumed a greyish tint, gradually increased in extent, and were covered with a greyish-red or blackish-red layer of necrotic tissue. The lesions extended into the surrounding normal tissue, and there was no evidence of delimitation. When the gangrenous parts began to separate, the vaginal discharge decreased, and in from fourteen days to five weeks it ceased completely. The discharge was generally yellowish-white and viscid, but when there was an extensive gangrenous wound it became of a red colour. It contained epithelial cells, pus corpuscles, and various micro-organisms, and when most abundant the bacterial contents were numerous; but in the early stages of the disease and towards its termination the bacteria were comparatively few in number. In some cases the uterus was involved, and the discharge was then reddish in colour, thin, and very fœtid; in one instance the uterine mucosa was covered with gangrenous wounds.

Constitutional symptoms were absent, except in some cases in which the uterus was involved. When the cows recovered they again became pregnant.

Prophylaxis and Treatment.—Immediate isolation was carried out, so as to prevent possible contamination of the milk of the healthy cows by the discharge, also with a view to prevent the spread of the disease.

When the uterus was not involved, irrigations of the vagina were found to be contra-indicated, as there was a risk of introducing the infective material into the uterus. As the affection, when confined to the vagina, tends towards spontaneous recovery, and runs a typical course, Bergmann found that treatment of any kind was unnecessary, beyond cleansing the external genital organs and the root of the tail with antiseptic lotions. When, however, the uterus was involved, weak antiseptic irrigations were found necessary. It was pointed out that the uterus is not infected with the bacillus of necrosis, but with pus cocci, which develop simultaneously with the primary disease, and that, if carefully carried out, the irrigation need not be repeated more than twice. Irrigation with lukewarm water was first carried out, and when the fluid came away unstained, the antiseptic solution was then injected. Bergmann found that, unless the fluid was thoroughly removed, the irrigation was likely to exert an injurious rather than a favourable effect.

When the disease assumed an enzoötic form, it was advised that, in order to check its further spread, all wounds in the labia of the vulva and in the vagina resulting from delivery should be treated with a caustic solution, such as a 10 per cent. solution of chloride of zinc, until the superficial layers of tissue assume a white colour. In order to reach points situated in the anterior region of the vagina, a small portion of cotton-wool saturated with the solution can be applied to the affected parts by means of a long-handled forceps, or attached to the end of a thin stick.

VESICULAR EXANTHEMA OF HORSES AND CATTLE

BY E. WALLIS HOARE, F.R.C.V.S.

A PECULIAR infectious disease, affecting the generative organs of horses and cattle, and rarely sheep, goats, and pigs, is described by Continental authors. It consists of an eruption on the skin of the vulva and mucous membrane of the vagina in the female, and on the penis in the male. This eruption presents a papular, vesicular, and pustular stage, and later on develops into flat ulcers, which heal, leaving cicatrices.

The disease appears to be common in various parts of Germany, often occurs in the form of an enzoötic, and is far more frequently met with in cattle than in horses. A mild form of the malady in cattle is frequently met with in Great Britain (see p. 362).

I. VESICULAR EXANTHEMA IN HORSES.

Etiology.—The nature of the infective material has not been discovered. The virus exists in the contents of the vesicles and pustules, and in the discharge from the ulcers. French authorities regard the disease as a variety of equine variola, and, according to Peuch and Galtier, if cows and calves are inoculated with the virus, genuine cow-pox is produced. Instances are recorded in which the disease has been transmitted to man, the usual site of infection being the hands, the eruption resembling that of cow-pox, and causing swelling of the arm and axillary glands, accompanied by a rise of temperature.

The usual mode of infection in animals is by coition, but the disease can also be conveyed by other means, such as contact between an affected and a healthy mare, and by contaminated grooming utensils. According to Dayot, Hess, and others, foals may contract the disease during the act of sucking their infected dams, the eruption occurring on the lips, mouth, nasal mucous membrane, and conjunctiva.

The affection can be transmitted by experimental inoculation from horse to horse, and also to sheep, goats, and pigs.

Immunity is not conferred by one attack, and, according to Steinhoff, stallions after recovery from a primary attack may become reinfected.

The disease is most frequently observed during the spring months, this being the time at which mares are sent to the stallion. According to Friedberger and Fröhner, the number of cases of vesicular exanthema in horses occurring in Germany from the year 1886 to 1902 amounted to 3,650.

So far as we are aware, the affection has not been met with in the British Isles.

Period of Incubation.—This varies from three to six days, and in many instances may be from twelve to twenty-four hours.

Symptoms.—Two types of the disease are recognised—viz., (1) *Mild*; (2) *Severe*.

Mild Type.—In the mare the eruption occurs on the inner surface of the labia of the vulva, on the vaginal mucosa, especially in the vicinity of the clitoris, and to a less extent on the external surface of the vulva and under surface of the tail, and occasionally on the skin of the quarters.

The vulva is swollen and the vaginal mucous membrane is congested. On the latter dark red spots first appear, the size of a pin's head; these become papules, and later on vesicles and pustules. The vesicles are transparent in the early stages, then change to various tints of yellow, and alter to a brown colour; in some cases the contents are of a red colour, due to slight extravasation of blood. The pustules develop into flat ulcers with a deep red base, and discharge a yellowish glutinous fluid. Frequently two or more ulcers become confluent, and are covered by a brown scab. When healing takes place, smooth white cicatrices remain, while in skin of a dark colour round spots are left, devoid of pigment. As a rule fever and constitutional disturbance are absent, but sexual excitement, frequent attempts at micturition, vaginitis in varying degree, eczema of the surrounding parts, and rubbing of the tail, due to pruritus, may be observed. Recovery usually takes place in about fifteen days.

In the stallion the penis is swollen and the mucous membrane congested, and vesicles and pustules, forming ulcers later on, are observed. When healing occurs, cicatrices are left. Occasionally the outer surface of the sheath and the scrotum may be involved, also the inside of the thighs and under surface of the abdomen, extending to the inside of the fore-limbs, probably due to contact with infected secretions. A urethral discharge may be present in some cases, and frequent attempts at micturition may be observed. The eruption generally heals spontaneously in from three to four weeks, and sometimes in fifteen days, if the stallion is prevented from covering mares.

Severe Type.—In the mare the udder becomes swollen, and the œdema may extend along the inferior surface of the abdomen to the thoracic region; the hind-limbs may be swollen to the level of the hocks. The lymphatic glands and vessels are involved by a suppurative inflammation, and abscesses may form in the udder, in the region of the anus, and at the root of the tail. Deep ulcers may result, and in some instances the diseased process may extend to the uterus. In addition to the above phenomena, marked constitutional disturbance, high fever, a stiff gait, extensive local swellings, and emaciation, may be present. A fatal result occurs in a few cases. The course of the severe type of the disease is slow, and may last for six months or longer. Chronic vaginitis may be observed as a sequel to the affection. In the stallion swelling and abscess formation in the inguinal glands, lameness, digestive disturbances, and emaciation, may be observed.

A severe case of the disease may be mistaken for dourine, but can be differentiated from the latter by the absence of paralysis and by the fact that it can be transmitted to cattle by inoculation.

Preventive Measures.—Preventive measures consist of strict isolation of the affected animals, and instructions that such animals should not be used for breeding until perfectly cured. Attention should also be directed to disinfection of stalls and grooming utensils, etc.

Treatment.—In the mild form of the disease the employment of antiseptic and astringent lotions is indicated. In the severe cases, surgical measures may be required in addition to the above.

II. VESICULAR EXANTHEMA IN CATTLE.

Some authorities consider this affection to be similar to that occurring in the horse, while others believe that it resembles cow-pox. All agree as to its marked infectious character. The disease is conveyed by coition, and an infected bull may transmit the affection to every cow he serves. But it is generally admitted that infection can occur by other means, such as a diseased cow rubbing her hind-quarters against those of a healthy cow, or if the latter lies with her hind-quarters against troughs contaminated with the discharges, or from contact with infected litter; or the virus may be carried by the tail of an infected cow striking the vulva of a healthy cow. These modes of infection are suggested by the fact that the disease is seen in non-pregnant cows and in those far advanced in pregnancy, while it is also met with in calves of five weeks of age.

As in the affection of horses, the nature of the virus is unknown. The period of incubation is from one to six days, the maximum being ten days.

Symptoms.—In the cow the symptoms are similar to those described as occurring in the mare. The discharge is more likely to be carried to the udder in the cow in consequence of the length and movements of the tail. Eczema of the hind-quarters is frequently met with, and marked pruritus is present; the abundant and irritating discharge causes excoriation of the skin of the vulva and of the hind-limbs.

Constitutional symptoms are generally of a mild character, and abortion rarely occurs. Chronic catarrh of the vagina may result as a sequel to the disease, and sterility is frequently met with.

In the bull the penis, sheath, scrotum, and anus, may be involved. A thin yellowish muco-purulent urethral discharge may be present, which causes matting of the preputial hairs on the sheath. Frequent attempts at micturition, difficulty in serving cows, and in some instances strangury and phimosis, may be observed. In rare instances gangrene of the penis and permanent deformity of the organ have been met with.

The course of the disease is usually one or two weeks, but in severe cases it may extend to a month.

A differential diagnosis between this disease and contagious granular vaginitis may be necessary. In the latter affection there is an absence of vesicles and pustules, and the lesions consist in the presence of numerous flat, dense nodules or granular processes in the vaginal mucous membrane (see p. 352).

Prophylaxis and treatment are similar to those advised in the equine form of the disease.

III. VESICULAR VAGINITIS OR BULL-BURN.*

In Great Britain a mild form of vesicular exanthema in cattle is rather frequently met with, and is known as "vesicular vaginitis" or "bull-burn." It is readily transmitted by the hands of attendants, by grooming utensils, etc., also by actual contact and through the medium of contaminated litter, etc. In some outbreaks only those cows served by an infected bull develop the disease, and conception frequently occurs in spite of the animals contracting the disease. In other instances the infection may spread to a number of cows in a shed by means of actual contact. The disease may be conveyed by inserting swabs of cotton-wool containing the virus into the vagina, and also by the hands or even the finger when contaminated by the exudate. The degree of vaginitis varies considerably as regards intensity in different individuals in the same outbreak. The vulva and the posterior portion of the vagina may be lined by a dark grey or reddish-grey covering of fibrinous lymph, forming a more or less tough membrane, under which the mucous membrane is intensely hyperæmic, and bleeds readily. On the other hand, the exudate may be represented by flakes of lymph or muco-pus scattered over the surface of the congested and swollen mucosa. The exudate seldom persists longer than from seven to ten days, and the average duration of the disease is from two to three weeks. The clitoris is most severely attacked, and the exudate persists longer in this region, just as in the case of contagious granular vaginitis.

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* For the description of this disease we are indebted to Professor Ainsworth Wilson, F.R.C.V.S.

The secretion of milk is not interfered with, constitutional symptoms are absent, and spontaneous recovery without treatment readily occurs. In very acute cases the temperature may be elevated 1° to 2° F. for several days. No other lesions are observed. The external genitals (vulvar labia) show considerable swelling, and are painful on manipulation; switching and elevation of the tail are observed; in some cases frequent micturition occurs, accompanied by arching of the back and more or less straining for a minute or two after the act.

The vesicles in the mucous membrane are frequently unobserved; they soon rupture and assume a purulent character. The so-called ulcers that are met with are merely slight erosions or a shedding of the epithelium.

There seems little doubt but that numerous outbreaks of this disease occur, and are overlooked by the owners and attendants; hence they do not come under the observation of the veterinary surgeon.

Sterility is not a common sequel to the affection, provided the affected animals are kept from breeding for a period of six weeks.

With regard to prophylaxis, special attention should be paid to the bull. Co-operative bulls—*i.e.*, those serving cows from various districts—are more likely to develop the disease and to convey it to healthy herds.

In bulls, when first attacked, the course of the disease is more severe than in the case of cows, but when examined there may be little or no preputial discharge or swelling present.

Measures for isolation and disinfection should be carried out.

VENEREAL DISEASE IN SHEEP

By E. WALLIS HOARE, F.R.C.V.S.

Outbreaks of venereal disease among sheep in Great Britain are described by Flook and McFadyean.* These simulated in almost every respect the venereal form of contagious pustular dermatitis described at p. 305.

In Flook's cases fifty-two yearling ewes and two ram lambs were purchased and brought to a farm. Shortly afterwards one of the ram lambs developed a discharge from the sheath, and the other showed a rather extensive eruption around the mouth and nose. These two rams were separated from the yearling ewes and put with a small flock of older ewes. After the rams had been with the ewes for about a week, a number of the latter developed a peculiar affection of the vulva, and the rams were then removed.

Nine of the ewes were found to be affected, the symptoms observed being marked swelling of the vulva and the presence of raw bleeding sores on the cutaneous, as well as the mucous, surface of the labia. It was ascertained that one or two of the affected ewes had not been served by the rams in question, and it was surmised that in these the infection might have been carried by flies, which were very numerous at the time, or by the lesions on the nose of one of the rams. Constitutional symptoms and fever were absent.

One of the affected rams showed raw ulcerating sores inside the prepuce, and in the other the eruption on the upper lip appeared to be healing.

The affected animals were isolated, and the lesions were dressed with mild antiseptics, and later on iodoform ointment was applied to those on the vulva.

The outbreak investigated by McFadyean occurred in 1896,† on a farm in the county of Suffolk. Twelve ewes of the flock were affected, but as the ram which had been with the ewes was a hired one, and had been returned to its owner six days previous to the investigation, infection from this source could not be traced. According to the shepherd, this ram had access to only ten of the affected ewes.

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* *Journal of Comparative Pathology and Therapeutics*, December, 1903.

† *Ibid.*

The symptoms presented by the twelve ewes were as follows: "The labia of the vulva were intensely inflamed, swollen, tense, and painful. The lining membrane of the vulva showed inflammatory congestion. and in some cases ulcers were present either on the outside or the inside of the labia. There was a slight purulent discharge from the genital passages." Experiments were carried out by means of rubbing pledgets of sterilised cotton-wool over the lesions, so as to collect some of the discharge, and inserting one of these pledgets into the vulva of a yearling ewe, another into the vulva of a cow, and a third was introduced within the prepuce of a wether.

The pledget dropped out of the vulva of the ewe one hour after insertion, and those in the cow and wether were then removed. In the cow and ewe the results were negative. In the wether, on the third day, the prepuce was found slightly swollen around the orifice, and on the following day the swelling had increased, and a brownish crust or scab had formed on the skin close to the preputial orifice. The swelling in the prepuce increased, and a slight discharge appeared; the swelling persisted for ten days, and the small sore which was observed on the skin close to the preputial opening, continued to spread as a red ulcerating surface. On the twelfth day two small abscesses formed on the prepuce near the primary sore, and on the following day these were converted into shallow ulcers. A third abscess formed the day afterwards; the ulcers increased in extent, and were covered with brownish crusts. Healing then took place, and the lesions disappeared about two weeks afterwards.

Attempts to transmit the affection from this wether to another wether and a ewe by means of cotton-wool containing the discharge failed; also it was found impossible to isolate from the lesions an organism capable of inducing the disease.

McFadyean was of opinion that the affection could scarcely be of common occurrence among sheep, as, if otherwise, its existence would have attracted attention. Also he believed that, under favourable circumstances, the disease was one that might spread indefinitely. "But since it seems to run a comparatively rapid course, and always ends in recovery, a flock infected with it one season would probably have become clean by the following season. There may, however, be exceptions to this rule, the disease being continued from one season to the next, either by a ram or a ewe that has not made a complete recovery. At any rate, it would appear to be a not altogether needless precaution to see that newly-purchased rams are free from any signs of the disease."

VENEREAL DISEASES IN THE DOG, RABBIT, HARE, AND FOWL

By HENRY GRAY, M.R.C.V.S.

I. SPECIFIC VENEREAL DISEASES IN THE DOG.

It has often been stated that the dog is affected with a specific gonorrhœa, but on what evidence this statement is founded the writer is at a loss to comprehend, as there are no reasonable grounds to support it. What is generally mistaken for this malady is merely a catarrhal condition confined to the mucous membrane of the sheath or vaginam penis (balanitis), so commonly encountered in those animals denied sexual intercourse or suffering from an eczematous diathesis. The affection is not contagious, nor does it spread to the urethral canal. It readily disappears after repeated injections of mild antiseptic solutions of chinosol or sulphate of copper. It may, however, recur from time to time.

A profuse catarrhal discharge from the mucous membrane of the sheath is often seen during the course of distemper, when a similar discharge issues from the nostrils and conjunctival membrane. This may be transmitted like any distemper discharge, but it disappears as soon as the general infective process has run its course and recovery is obtained. It is not a specific virus of the genital canal.

Some authorities even go so far as to make the bold statement that the gonorrhœa or specific urethritis of man is conveyable to the dog, but every attempt to transmit it to this species, as well as to other animals, has failed.

We consider that there is no logical evidence yet put forward to show that the dog is the subject of a specific urethritis or gonorrhœa, either conveyable from dog to dog or from man to dog, and *vice versâ*.

II. INFECTIVE VENEREAL GRANULOMA OF THE DOG.

Synonyms.—Infective sarcoma; Lympho-sarcoma; Myxosarcoma; Vaginal warty growth; Fungating excrescence.

Definition.—A contagious disease of the canine species, affecting chiefly the mucous membrane of the vagina in the female and that of the sheath or vaginam penis in the male, and characterised by warty or cauliflower-like excrescences or growths, which are transmissible, chiefly by coition and by inoculation.

History.—This disease is mentioned by Blaine, and is stated by Hertwig, who has given a very good clinical description of it, to be contagious. It did not, however, attract much notice until 1877, when Nowinsky, a Russian scientist, made known his researches on its transmission by inoculation into the vagina and under the skin. In 1889-1890, Wehr also studied the disease. Since then numerous other investigators, including Duplay and Cazin, Geissler, Bellingham-Smith and Washbourne, Powell White, Sanfelice, Sticker and Bashford, Murray and Cramer, have devoted much attention to it.

Etiology.—S. P. Beebe and James Ewing, of the Huntingdon Fund for Cancer Research and the Department of Pathology, Cornell University Medical College, found a spirochæte in the lesions of one case, but were not successful in seven other clinical cases. In 1907 Mettam found a similar organism. Probably these spirochætes only accidentally invaded the lesions, as they are normal saprophytes of the gastro-intestinal canal of the dog.

The causal organism is, therefore, yet to be found and demonstrated. All that is known with reference to its causation is that the disease is conveyed by coitus and by inoculation. In human syphilis a spirochæte —the *Spirochæta pallida*—has been demonstrated to be the cause of that malady, for which Metchnikoff endeavoured to find a trustworthy combative serum and Ehrlich has introduced a synthetic remedy, known as "606."

The toxins of the tumours have been shown to set up acute interstitial nephritis when injected into dogs.

Geographical Distribution.—The disease occurs extensively in England, France, Germany, Russia, and the United States of America.

Susceptible Breeds.—It is chiefly seen in the British, miniature and French varieties of the bull-dog; to a less extent in the pug, and rarely in other breeds.

Sex and Age.—It attacks both sexes, especially the young and mature adult used for breeding purposes. It is rarely seen in those animals not bred from.

Mode of Transmission.—The disease is conveyed chiefly by coitus, and may occur as a veritable enzoötic in some large breeding establishments, and hardly one of the animals used for breeding escapes the infection. A mere rubbing of a portion of the infecting tumour on the abraded mucous membrane is sufficient for a successful inoculation.

Symptoms.—Although it is essentially a venereal disease, it may arise independently on other parts of the body without the generative organs becoming affected. The lesions are commonly encountered on the skin of the neck, throat, back, groin, abdomen, limbs, and between the digits. They are also witnessed as secondary nodular growths in the abdominal lymphatic glands, mesenteric glands, spleen, liver, kidney, and lung.

In the *genital organs of the bitch* the lesion generally begins as a small lumpy shotty or warty growth, usually having a sessile base, on the mucous membrane of the vagina, and this may, after a time, increase in size and multiply, the multiple growths running into one another and forming a large pinkish, purplish, or greyish-red cauliflower excrescence, which bleeds when touched, or discharges a bloody muco-purulent material that escapes from the vulva, which is, in advanced cases, generally swollen. The most usual seat of the growth is in the vicinity of the urethral opening. It may also be found at times in the vulval passage. Occasionally it may extend from the vulval passage towards the uterus, completely filling the vagina, protruding from the vulva, and causing the perineal region to bulge backwards.

In the *male* the nodular or warty growths often have a long pedicle, and are found on the penis and sheath, especially around the base, from which they may extend to other parts.

The tumours seem to grow with greater rapidity on old animals, and in those that are in a poor or anæmic condition, than they do on young dogs or those in a healthy state. The lesions may ulcerate or slough out, and where this takes place the part contracts and a scar or cicatrix remains, and the disease may in consequence spontaneously disappear. When one tumour commences to retrogress they all do so. When the disease is present, it is possible in a great number of cases for conception to take place, and to be followed by a normal parturition.

The appearance of the new growth is not preceded by any noticeable discharge, mucous, catarrhal, or sanious. Very often, when bitches are affected, they attract the attention of the opposite sex, leading one to believe they are " on heat."

Treatment.—This disease being eminently a strictly contagious one, its prevention should be aimed at by washing out the sheath of the valuable stud dog with a mild antiseptic solution, such as sulphate of copper, after an immediate service with every strange bitch. The male dog should have his genital organs examined from time to time to see if he is free from the disease.

Where the disease has not spread to inaccessible regions there is a possibility of a permanent cure, even without serious mutilation. The writer has come across many cases that were absolutely cured, without any recurrence during the remainder of the animal's life, although in the majority of instances the interval between recovery and death had extended over several years, and during that period the bitches have generally given birth to numerous progenies, which mostly have, in their turn, become parents, grandparents, etc.

There is no practical reason why the operation of sterilisation (oöphoro-hysterectomy) should be performed as a preliminary to the local treatment, and thus render a possibly curable case a valueless one from a breeder's point of view. Such an operation does not cure the disease. The writer has emphasised this point, as he knows sterilisation has been proposed and recommended, such an idea being based on the fact that sterilisation has brought about diminution of uterine fibromyomata in the human female; but this is quite a different matter, and bears no analogy to the one now under consideration.

When the disease is limited to the vagina or external genital organs, it may be permanently eradicated by thoroughly scraping away the diseased tissue by means of a Volkmann's curette, and afterwards by applying solid copper sulphate to the resulting raw surface. The crystal should be applied a few times, with intervals of two or three days, so as to destroy any diseased tissue left by chance. It may, however, be alternated with the local application of strong tincture of iodine or liquor arsenicalis (P.B.). Other caustics, as zinc chloride, iron perchloride, silver nitrate, may be used. The internal administration of arsenical compounds seems beneficial.

This treatment should be carried out thoroughly and patiently until the wound has healed up without leaving any trace of the disease. The animal should be examined from time to time, in order to see if there are any signs of a recurrence, and the same treatment should be again adopted if necessary.

During such treatment there is no necessity for the use of a general anæsthetic, a tampon saturated with cocaine and adrenalin passed up the vagina and left there for half an hour before using the curette being sufficient to efficiently blunt sensation. Instead of the tampon, a suppository containing these drugs may be used. Hæmorrhage from curettage is usually a negligible quantity. Any resulting narrowing of the vagina after cicatrisation may be overcome by the use of a uterine dilator after cocainisation. This is, however, rarely called for.

It is not known if the cat is subject to a contagious venereal disease.

III. CONTAGIOUS VENEREAL CATARRH IN THE RABBIT.

A contagious venereal catarrh affecting the genital organs of rabbits has been observed. It is transmissible from the male to the female, and *vice versâ*. It may occur as an enzoötic, taking several weeks to run its course.

It is amenable to the patient irrigation of the external genital canal of both sexes with a solution of sulphate of copper, nitrate of silver, perchloride or biniodide of mercury, or liquor plumbi.

IV. PSEUDO-VENEREAL DISEASE IN HARES.

As in the rabbit, the hare is also affected by a disease of the external genital organs. It is well known to gamekeepers and sportsmen. It occurs as a veritable epizoötic, and has been observed on various estates in Great Britain. A widespread outbreak was witnessed in Lancashire, and was partially studied by Strangeways and Chapman. The writer has observed it in Suffolk.

Occurrence.—On some estates as many as 75 per cent. of hares are affected; on others 25 per cent. This estimate is, however, only problematical.

Symptoms.—The affected animal becomes emaciated and weak. In the severe form it runs feebly, and with its hind-limbs wide apart and carried in a peculiar manner. The penis and the vagina seem to escape infection, but the lesions appear on the skin covering and surrounding the regions of the genital organs. The nose and lips are also affected. The lesions consist of a mass of granulation tissue forming around the external genital organs; this tissue is considerably raised above the surrounding parts, and is often ulcerated. Those of the nose and lips are, in the early stages, a small scab, usually on the upper lip, but sometimes on the lower, the thick fur evidently assisting in the formation of the scab. This lesion forms and spreads slowly, until in many cases a considerable portion of the lips and nose is involved, when, after a time, the ulceration increases, and the necrotic portion is replaced by soft red granulation tissue. The mucous membranes of the mouth and nose are not involved even in serious cases.

Animals kept in captivity suffer much more severely than those at liberty. If the lesions become injured, they increase in size and spread rapidly, and often extend to the bony framework. In such, small abscesses may be found in the lungs, liver, heart, muscles, or other parts.

Transmission to Other Animals.—The disease is conveyed to the rabbit and guinea-pig by contact, by inoculation, and by being in hutches contaminated by a diseased hare. In such cases typical lesions develop on the nose and lips. Young rabbits show the disease soon after birth. Lesions may appear on the top of the head and round the eyelids. In the suckling rabbit the mammary gland may be affected.

Bacteriology.—So far as the writer is aware, the microbiology of this disease has not been determined, but the clinical description somewhat resembles that of the necro-bacillosis of the rabbit (see p. 298). However, a diplococcus has been isolated, but the part it plays in the disease has not been ascertained.

V. CONTAGIOUS VENEREAL CATARRH IN THE FOWL.

Synonyms.—Cloacitis ; Vent gleet.

A contagious catarrh affecting the cloaca, and sometimes extending to the internal membrane of the lower portion of the oviduct, and also to that of the rectum, has often been observed in fowls. It is conveyed from bird to bird during coition.

It should be treated on similar lines to that of the venereal disease of rabbits. Cocaine may be added to the solution used, so as to prevent the fowl bearing down.

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EPIZOÖTIC INFECTIOUS EPIDIDYMITIS IN THE HORSE

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Contagious orchitis ; Enzoötic epididymitis.

French : Épididymite infecteuse épidémique du cheval.

This is an enzoötic disease of the entire horse which was observed in Havre by Lesueur and Lavergne, and studied by Vallée of Alfort. It attacked eight horses in succession out of a stud of thirty-four stallions.

Bacteriology.—In every affected testicle removed by castration a very small bacillus was found in the centre of the lesions of the epididymis. This bacillus, which appeared to be the specific agent of the disease, furnished pure cultures on various culture media. This organism differed entirely from any other microbe hitherto classified.

Symptoms.—The affected animals showed external signs of orchitis, with extremely serious general phenomena. At the commencement the temperature rose to 40° and even 41·5° C.

The lesions, which were generally confined to one testicle, were manifested by a tremendous swelling of the scrotum and of the sheath. After several days the initial œdema disappeared, and the testicle and its coverings formed one solid and very painful mass, and in consequence examination as to its character could only be imperfectly carried out.

The general condition rapidly improved, whilst the local signs remained stationary, preventing any working of the animals.

Mallein was used to establish the non-existence of glanders in the infected stable.

Castration seemed to be the only remedy to remove the lesion. This was carried out on six of the animals.

Lesions.—A median section of the testicle and its adherent coverings showed that the epididymis was more or less severely affected ; its canals sometimes contained a greenish-yellow, very homogeneous creamy pus. In other cases there was no longer any trace of the primary structure of the canals, and a voluminous sequestrum occupied the place of the epididymis.

In general, the testicle was absolutely indemnified. In only two cases the seminal gland was affected, very slightly in one and very severely in the other, in which the testicle contained a sequestrum the size of a child's fist, which delimited itself before the organ was removed by castration.

The vaginal sheath was constantly affected ; it showed more or less marked exudative alterations. There was, however, always a predominance of lesions in the epididymis over those in all the other appendages of the testicle.

Treatment.—Castration assured prompt and complete establishment of health.

MAMMITIS

By HUGH BEGG, M.R.C.V.S.,
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Synonyms.—Mastitis ; Garget ; Weed ; Udder-clap ; Downfall of the udder ; Mastitis catarrhalis contagiosa ; Agalaxia catarrhalis contagiosa.

French : Mammite Streptococcique de la vache.

German : Gelber Galt ; Brust drüsen-entzündung ; Enterentzündung.

Definition.—A contagious specific inflammation of one or more quarters of the mammary gland. The morbid processes may be subacute, acute, or chronic, the type of the disease, as well as its tendency to spread from animal to animal, being influenced by the propensities and virulence of the causal micro-organisms present. Rapidly contagious forms of the disease occur among cows, sheep, and goats.

GENERAL REMARKS.

An ever-increasing impetus has been given to the importance of the diseases of the udder of the cow by the awakening of the public mind to a knowledge of the dangers that lurk in the use of impure milk. The outcry for a pure and wholesome milk-supply, and more of it, is yearly becoming more general, though its accomplishment is extremely tardy. The time is more than ripe for a State-aided investigation of many of the unsolved problems that attach to diseases of the udder of cows, and the vexed question of the culpability of impure milk in causing such diseases of the human subject as septic sore throat, etc.

For quite a century mammitis has been recognised and written about as a serious disease of milch stock, but we have no record of its having been studied bacteriologically until 1875, when Dieckerhoff's article appeared in the *Veterinary Journal* with reference to an outbreak of septic mammitis which he attributed to a septic ferment. Zarn in 1877 considered the disease a contagious one, and not due to the causes usually accepted. Even prior to 1848, when Brennwald published his observations on enzoötic mammitis in Switzerland, some practitioners believed the disease to be contagious.

There is little doubt that in Great Britain, as in other countries, before the idea of contagion was mooted in regard to any form of mammitis, many dairymen had experienced outbreaks of this disease directly due to unclean habits, and ignorance of the potentialities of the first cases, with a consequent absence of any attempt at prophylaxis.

As the disease, whether it be acute, subacute, or chronic, is presumably not spread by aerial infection, but almost solely by the hands of the milkers, it may be considered virulent when it claims an average of one victim daily for a week or two. In the less virulent forms the spread of infection is much slower.

This comparative tardiness of infectivity, in the absence of bacteriological knowledge of the disease, was undoubtedly the reason why veterinarians and stock-owners were led to seek a persistent cause in dietetic errors, or some defect in the ventilation or structure of the cowshed, or the probable incapacity of one or more of the milkers, any or all of which factors, as well as many other side-issues, might easily act as contributory causes.

It is now generally admitted that the morbid processes that obtain in every case of hitherto so-called sporadic mammitis are, directly or indirectly, due to microbic action through the absorption of some infective material or some micro-organisms, and anything that lowers the vital defences of the udder favours the pathogenicity of the invading organisms, just as occurs in the case of respiratory diseases. When a state of lowered tissue-resistance results from any cause, the bacteria are comparatively unhampered in their development and attack.

The wonder is that udder disease is not more common, when we consider the apparent vulnerability of the organ and the many unfavourable influences to which it is exposed. In milch cows the udder is highly vascular and physiologically very active, secreting milk, which is a fertile medium for the cultivation of many bacteria, aërobic and anaërobic. Its delicate covering of skin is liable to various lesions, eruptive and traumatic, while the teat canals offer a comparatively easy venue for microbes.

In respect of its anatomical position, the udder, particularly when very pendulous, is liable to injury from the feet of neighbouring cows, to horn-gores when the animals are loose, and to wounds more or less extensive when jumping fences. Again, when the animal is recumbent, the organ is grievously exposed to infection through contact with excreta, suppurative discharges from the genital organs, suppurating sore feet, abscesses of the hind-limbs, etc.; and we cannot overlook the danger that arises from the filthy habit many milkers have of drawing the diseased secretion of an affected quarter direct to the ground, and leaving the cow to paddle in it, and eventually to lie down and contaminate the teats and surface of the udder with the obnoxious material.

But nothing teaches a dairyman so effectively the virtue of cleanliness in the handling of milch cows as an outbreak of acute mammitis. The labour entailed, the loss sustained, and the disorganisation of the herd and the milk-supply, impel him to do all he can to prevent a recurrence of this dread disease.

Every suppurating sore occurring in the byre must be looked upon as a menace to the udders of the cows therein.

We have occasionally seen "foul of the foot" and mammitis co-existent in the same byre, and often in the same cow, and were of opinion that the latter disease was due to infection having been carried to the udder by the hands of the milkers who dressed the affected feet.

Discharging sores of the feet, when not regularly dressed with antiseptics, contaminate the place on which the udder rests when the cow is lying down.

Similarly, we believe that every case of mammitis in the cowshed is a potential source of danger to the healthy animals when strict precautions are not taken to prevent infection.

In one serious outbreak of acute mammitis the first five cases occurred in alternate cows, which had been regularly milked by one of two milkers, whose duty it was to dress the suppurating bed-sores and feet of a cow suffering from "whitlow," which cow became the first victim of the udder trouble.

ETIOLOGY.

1. Contributory and Predisposing Causes.

Though occasionally cases of mammitis have been met with in yeld (*i.e., dry*) cows or even in virgin stock, lactation is, for the great majority of cases, practically an essential condition of the udder. For the development of the more acute forms of mammitis the state of the organ at the period of "*full flow*" is most favourable, and on this account the advent of the disease is all the more regrettable.

At that period in many deep milkers the pressure downwards of secreted milk for some time prior to each milking overcomes the teat sphincter to some extent, thus facilitating the escape of milk and the ingress of germs brought to the teats by soiled hands, litter, etc.

It is also during the first stages of lactation, when the udder is markedly congested, enlarged, and œdematous, that it is most easily injured by direct violence—*e.g.*, horn-gores, kicks, blows, etc.

Likewise, at this period the teats, etc., are most liable and susceptible to eruptive attacks, the lesions of which are a predisposition to mammitis more potent than many serious injuries, especially if the eruption involves the point of the teat, causing partial adhesion and obliteration of the opening of the duct, and necessitating forcible handling of the teat for the withdrawal of the milk. In this event, as in all other instances of stenosis of the canal, the undue compression of the teat is liable to injure the mucous membrane of the tube; bacterial invasion of the teat substance follows, then inflammatory induration, which soon extends upwards. The use of a teat siphon at this stage is a mistake, and only makes matters worse.

Other secondary causes worthy of mention are overstocking or hefting, inefficient milking, leaving teats wet with milk, exposure to colds and draughts, insanitary conditions of the cowshed generally, etc.

2. Infective or Exciting Causes.

Mammitis may be set up by various micro-organisms acting separately or conjointly, but it is worthy of note that reported outbreaks have not been due to a mixed infection, but to a single definite species.

The fact that so many cowsheds have single cases of mammitis makes one hesitate to stipulate that all cases are contagious; nevertheless, we believe that such an assertion is no more than the truth, and that any apparent falsity rests on the probability that these cases are due to organisms of such slight virulence as renders them but mildly aggressive.

In such cases it is probable that a contributory cause, such as prolonged exposure to draughts, has, by lowering the vitality of the system, lessened the functional activity of the udder, and facilitated a more rapid growth of microbes, not markedly pathogenic, that were pre-existent in the teat canal and galactophorous sinus.

When the secretion of milk has not been thus prejudiced, the repeated dilution of the culture by quantities of newly-secreted milk, regularly drawn off, serves to keep the propensities of the organisms in check for an indefinite period.

Many investigators, notably Henderson, of Edinburgh, have proved how constantly various microbes are to be found in the milk secreted by apparently healthy udders.

Experimental injection into the udder of chemical irritants can produce inflammation, or perhaps death, of the parts with which they come into contact, but if there be no accompanying or subsequent microbic invasion, no extension of disease can take place, suppuration does not occur, and the result of such an experiment has no bearing on the questions of the etiology and contagion of mammitis as we encounter it in the cowshed.

Out of a total of 444 samples of altered milk examined in 1895-1897, Zschokke found streptococci in 297 (*i.e.*, in 70 per cent.).

There are three routes by which infection of the udder may occur :

1. *By the Teat Canal.*—This is by far the most important. Experiments made by Kitt, Nocard, Franck, and Bang, proved that mammitis could unfailingly be set up by the injection into the teat of pure cultures of certain bacteria, and when Kitt merely painted the teat with a pure culture, mammitis resulted.

We believe that every experienced cattle practitioner could put on record numerous cases of mammitis directly caused by using an unclean teat siphon to facilitate the milking of a "tough" teat. It is to the competency of the teat sphincter in these cows which escape attacks of mammitis that immunity must in a great measure be attributed. In others this circular valve is less effective, while in some it is so markedly relaxed that the milk escapes spontaneously for a considerable time prior to each milking period.

The latter are the cows that are peculiarly predisposed to mammitis, for when their teats are left wet with milk, or when a drop hangs at the entrance to the canal, the pathogenic bacteria, finding these conditions favourable for their growth, pass readily through the almost patent opening to the galactophorous sinus, and set up whatever type of inflammation their virulence is capable of producing.

2. *By the Skin.*—Lesions of the integument of the cow's udder are very common, and often constitute the starting-point of infection. When the surface of the udder is inoculated in this way, a spreading lymphangitis occasionally results, involving first the superficial lymphatics of the skin and subcutaneous tissue, and later, if the case be neglected, the tissue proper of one or more quarters of the gland.

Under this heading are to be included the lesions of such specific diseases as foot and mouth disease and cow-pox, the victims of which are predisposed to what Kitt has called "lymphogenous mastitis."

3. *By the Blood-Current or Lymph-Stream.*—Such vascular infection of the udder is undoubtedly rare, if we except tubercular mastitis, in which the tubercle bacilli may have reached the udder by one of these paths in a centrifugal way.

Bacteriology.—The sources of the micro-organisms capable of setting up mammitis are manifold, since the stalls are often and variously contaminated, and there is no limit to the means by which milkers, male and female, soil their hands ; but we cannot entirely free from suspicion any microbes that find in milk a suitable propagating medium, provided they are conveyed to the teat that has a droplet of milk left at its extremity.

Whatever future investigation may reveal—and we believe it will be the means of adding considerably to the list of culpable micro-organisms —there have been up till now very few isolated in pure culture from the secretions of affected udders and proved to be specific by teat injection experiments.

The following are worthy of mention—viz. :

(1) *The Streptococcus of Nocard and Mollereau.*—This long chain streptococcus was isolated in 1884 from the udder secretions of ten cows suffering from mastitis. Kitt denominates it the *Streptococcus agalactiæ*, and Guillebeau the *Streptococcus mastiditis contagiosæ*.

Hess and Borgeaud, in studying " Gelber Galt " in Switzerland, found a streptococcus, which Kitt and Adametz demonstrated to be identical with that of Nocard and Mollereau. It was pathogenic to the cow and goat, provided it was directly introduced into the mammary gland when the latter was in a state of activity. No effects were produced by its inoculation in opposite conditions of the udder.

(2) *The Bacillus tuberculosis.*

(3) *The Bacillus Mastitidis Contagiosæ of Carré.*—In 1906, Carré conclusively proved that an outbreak of mastitis in the practice of Pierrot of Stenay was due to a microbe to which he gave the above name. It stains well with all the aniline colours, and is Gram-positive.

(4) *Guillebeau's Bacillus.*—This slightly mobile organism has been studied and described by Frendenreich. It is Gram-negative, coagulates milk with gas formation, and is supposed to belong to the coli group, most of which are credited with being capable of setting up mastitis.

(5) *Staphylococcus Mastitidis of Guillebeau and Lucet.*—This microbe is Gram-positive, and coagulates milk.

(6) *Galactococcus fulvus.*

(7) *Bacillus Lactis Aerogenes.*—In the *Veterinary Journal* of April, 1911, Gilruth and McDonald record an outbreak due to this bacillus, which had hitherto not been proved pathogenic for bovines. Of forty cows milked by a milking-machine, thirty were infected, and became dry in one or more quarters after passing through the various stages of acute mastitis.

Agar-tubes sown with the purulent secretions developed colonies, in twenty-four hours at 37° C., of short ovoid bacilli, some almost cocci, non-motile, Gram-negative, and identical with *B. lactis aerogenes*.

The authors say this organism is common in most dairies, especially those associated with piggeries, and they suggest that many sporadic cases of mammitis may be due to it, since it is pathogenic when introduced into the udder, and the infection may readily be spread by the milking-machine.

Other less studied organisms are the *Galactococcus versicolor* and the *G. albus*, both of which form chains in milk.

P. Dubois has reported an outbreak of mammitis which he was satisfied was due to the *Streptococcus conglomeratus* (Kurth).

Dr. Savage investigated thirty-six cases of mammitis from various parts of England, all more or less acute, and of all grades of severity. He found that 68 per cent. were due to streptococci, 10 per cent. (three cases doubtful) were probably due to streptococci, 16 per cent. were due to staphylococci, 3 per cent. were due to *Bacillus coli*, and 3 per cent. were due to *B. tuberculosis*.*

In the last outbreak experienced by the writer, and which has already been referred to, every case was alike in being extremely acute.

Samples of secretion from the affected quarters were submitted to Dr. J. H. Patterson, Bacteriologist for the County of Lanark. He reported the presence of staphylococci in pure culture which were identical with the *Staphylococcus pyogenes aureus*. No udder injection experiments were conducted.

Four distinct organisms have been isolated by Oreste and Marcone, and are believed to be the cause of a virulent type of mammitis of goats and ewes in Alpine regions (*vide* Contagious Agalaxia, p. 404).

From what has been published on the bacteriology of mammitis, one is forced to conclude, notwithstanding the assertions of some authors, that the production of the disease in any of its types—acute, subacute, or chronic—is not peculiarly the rôle of any particular species of microbe.

Different grades of virulence obtain among the various culpable bacilli, as well as in the multifarious forms of cocci that induce the disease, and though most of the less acute outbreaks are due to streptococci, this is not always the case; and, conversely, it can be asserted that many virulent cases of mammitis are not bacillary, but unquestionably micrococcal in origin.

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* *Journal of Comparative Pathology*, vol. xxii., p. 361: "The Bacteriology and Pathology of Mastitis in Cows; Report to Local Government Board," by W. G. Savage, M.D.

CLASSIFICATION OF MAMMITIS.

For the reasons stated above, classification on the ground of etiology alone is impracticable. The many plans which have hitherto been adopted in the classification of the disease according to its pathological anatomy are, without exception, subject to error and open to destructive criticism.

We confess our inability to distinguish clearly in the wilderness of nomenclature, and in practice have found it impossible to demonstrate exactly, either in the live animal or with the morbid specimen in front of us, how many of the terms we might not appropriately apply in a given case.

Catarrhal conditions in every case of infection by the teat precede the involution of the deeper structures, and while resolution may take place in this early stage, it is not possible when the infection deepens to specify, except in an arbitrary and artificial way, the period when the catarrhal merges into the parenchymatous or the latter into the interstitial. In an acute infection by the teat the entire structure of the quarter is implicated within a few hours.

It is, however, not difficult to determine in the early stages between a catarrhal mammitis and the primary interstitial (sometimes called "phlegmonous" or "lymphogenous") mammitis that results from infection beginning at the surface of the udder, but in this case also there is no demarcation-line between the simple interstitial and the mixed mammitis.

In all cases of mammitis there is a distinct relationship between the acuteness of the attack and the spread of the disease—the more severe the type, the quicker the infectivity, and *vice versâ*—and, however much they may vary in degree according to the type of infection present, it is characteristic that the symptoms and course are very constant all through any particular outbreak.

The study of mammitis is easiest when the disease is classified according to virulence of attack, and the following classification is deemed sufficiently comprehensive to embrace all forms of the disease met with in the cow:

I. SUBACUTE.
II. ACUTE.
III. CHRONIC.

I. SUBACUTE MAMMITIS.—Under this head are included the more benign forms of catarrhal, parenchymatous, and interstitial mammitis.

The **Period of Incubation** in the two former varies from two or three days to as many months; in the latter from two to seven days.

The pathogenicity of the causal organisms is not well pronounced, and in those cases arising from teat infection it may be that their action is limited to altering the character of the milk, which then acts as an irritant to the sensitive lining of the milk-ducts, establishing a catarrhal condition. Whatever further progress takes place is due to bacterial penetration of the impinged mucous membrane, when some degree of inflammation may be set up in the parenchyma of the gland.

Morbid Anatomy.—In catarrhal cases we find the lesions localised in the excretory apparatus—*i.e.*, the milk channels and sinus—the mucous membrane is swollen, the epithelium is shed, and there is increased vascularity, with exudation into, and infiltration of, the submucous tissue. The acini may also be involved to some extent, and at places granulations may be present, partially or completely blocking the milk channels.

In subacute interstitial cases we find the lesions consist of a perimammary lymphangitis; there is considerable effusion between the skin and the udder tissue, with some degree of exudation into, and thickening of, the interstitial tissue of the gland.

Symptoms and Course.—The symptoms do not appear very suddenly, and systemic disturbance is not well marked. The animal is only slightly indisposed; temperature not over 104° F. She ruminates, continues to take the choicest of her food, and the amount of milk secreted is not greatly reduced.

Locally in the *catarrhal* forms there is at first only a suspicion of swelling and pain, and no alteration of the milk from the quarter is apparent when it is looked at in bulk; but if a little be thrown across the back of the hand, or preferably on a dark and slightly convex surface, a few pinheads of casein can be seen.

Twelve hours later we find the pain and swelling considerably increased—they may have reached their height—and the product of the quarter is quite changed, consisting of a yellow serum containing clots.

Improvement usually begins on the third day, and recovery is often complete in seven days. The quantity of milk secreted by the quarter is often slightly prejudiced until after the next calving.

In subacute interstitial mammitis the local swelling and tenderness of the parts is more marked, the broadside of two quarters may be involved, the skin is redder in colour, but there is no alteration in the milk.

When quickly attended to, recovery is complete in two days, sometimes within twelve hours.

Prognosis is good in both cases, though the milk yield may be less than previously, and may not return to the normal amount until after the next calving.

II. Acute Mammitis.—It is but seldom that a cow suffers from what could be called "acute catarrhal" or "acute interstitial mammitis," and makes a recovery without an extension of the inflammation to the parenchyma of the gland. These are therefore usually to be looked upon as the initial stages of the graver affection, which invariably implicates the whole structure of one or more quarters within a very few hours.

The **Period of Incubation** is not more than from one to two days.

The virulent causal organisms which reach the udder via the teat canal very soon alter the character of the milk. The mucous membrane, through contact with this diseased material, suffers from an irritation that lowers its defensive power; then the bacteria can deliver an effective attack, and penetrate rapidly to the confines of the quarter.

It is extremely doubtful, notwithstanding the quick development of local symptoms, whether any species of microbe can overcome and penetrate the intact and healthy mucous membrane; but the altered secretion can prejudice its resistance, while the culture of bacteria is becoming more concentrated and powerful.

In acute mammitis commencing at the skin, the pathogenic bacteria rapidly involve all the superficial and subcutaneous lymphatics. Proceeding by the lymph-stream into the interstices, they soon invade the whole areolar tissue of the quarters affected, and finally implicate the acini and walls of the milk channels.

Morbid Anatomy.—It is rarely that one has the opportunity of examining a specimen in the early stages of the disease, thus the conditions then present can only be surmised when we study specimens in which the morbid processes are advanced. But a consideration of the early local symptoms when infection begins at the teat, indicates that an ascending sclerosis commences in the mucous membrane of the milk channels, and extends rapidly to the periphery, involving first the glandular elements, and afterwards the interstitial tissue of the quarter.

The naked-eye appearance of a section of a diseased quarter varies with the time the attack has lasted. The surface has a variegated appearance; the interstitial tissue is thickened and yellow, being filled with coagulated lymph; the acini are injected and infiltrated; and here and there we find small patches of extravasated blood and gangrenous areas of variable size.

When the progress of the disease has been rapid, the vascular disturbance is so grave, causing thrombosis, that the areas of gangrene and blood extravasation preponderate, and the cut surface is both evil-smelling and very dark in colour. In a small proportion of cases thrombosis is so general as to cause the death of the whole or major part of the quarter. The integument of such a *gangrenous* specimen is violet in colour, and easily separated and torn; and when the specimen is cut into, a dark, foul-smelling fluid exudes, tinged with escaping milk débris. The whole of the cut surface, excepting the walls of the main milk channels, presents a dark red, almost black, homogeneous appearance. When the morbid changes develop more slowly, extensive thickening of the areolar tissue prevails, and the parts become indurated and fibrous.

When such a specimen has been taken at a later stage, we find that several of the abscesses have increased in size and become confluent, and it is not uncommon to get the whole glandular structure of the quarter broken down by suppuration into a huge single abscess containing whitish-yellow pus.

Microscopical Appearances.—Sections of specimens in which the pathological changes are advanced show a complete removal of mucous membrane, and though this, even in the smallest tubes, is replaced by granulation tissue, the internal diameter of all the channels seems increased, and their walls have a solid, substantial appearance.

In some parts the adventitious granulations stretch across and block the lumen of the tubes, and though these are often quite empty and patent, we find in other places loose degenerated epithelial cells plentifully present in the lumen.

The glandular tissue is almost completely absent, while the connective tissue of the gland is enormously increased, and no doubt the hyperplasia of the septæ has helped the destruction of the lobules by the pressure of their increasing bulk.

In this proliferating connective tissue, as well as in some of the glandular lobules left, small foci of suppuration may be seen. Some of these are elongated or irregular in shape and size, indicating the coalescence of several centres, and it is in this way that abscesses having more or less fibrous walls are formed.

Microscopical inspection of a *gangrenous* specimen reveals complete destruction of all secreting cells, and nothing remains intact but the basement membrane.

Symptoms and Course.—An attack of acute mammitis is usually sudden and bewildering, and the general symptoms are almost invariably the first to meet the eye. A cow, apparently normal in health and milk yield the evening before, is found in the morning shivering violently, and straddling uneasily behind in her endeavour to relieve from pressure a violently inflamed quarter, and when a hind-quarter is attacked the uneasiness is very marked.

Much constitutional disturbance prevails. The thermometer registers 3° to 5° F. of fever, the pulse may be full and throbbing, but in some cases is small and feeble, counting upwards of 100 per minute; and with hair on end and an occasional inclination of the head towards the painful organ, she presents a picture of acute suffering and resentfulness of interference. For a day or two she takes no notice of food, though she will occasionally take a few mouthfuls of water. Rumination is entirely suspended. There may be slight tympanites, and an abnormal state of the bowels, constipation or diarrhœa. The ears, horns, and extremities, are alternately hot and cold ; she stands persistently, and if, wearied out, she seeks the recumbent position, the discomfort of lying on the painful organ soon causes her to get on to her legs again.

Local Symptoms.—When infection begins at the teat, and the case is noticed early, it is seen that the base of the teat is first diffusely indurated, then in a few hours the whole organ is engorged, red in appearance, hard and extremely painful when handled, and the skin is fixed. The teat is so stiff and inflexible that any available fluid can only with difficulty be withdrawn. The first strippings usually consist of from 3 to 4 ounces of a yellowish fluid, resembling stale beer, with no distinctive odour, and some flocculi are found suspended in it. Later on the secretion becomes more grumous and altered in colour, from blood-staining and the presence of pus cells and epithelial débris, and the odour is offensive. When centrifuged, there is a heavy deposit below a quantity of clear yellow supernatant fluid.

After two days the violence of the symptoms, systemic and local, usually begins to subside, and the product of the quarter becomes more and more pus-like in appearance and consistence, and if the teat remains effective the quantity procurable is increased.

Occasionally, however, it becomes impossible to draw off the secretion, the teat induration does not yield to manipulation, local stasis of circulation (thrombosis) occurs, then a change of colour to violet-red, and later to a greenish-black hue, with coldness of the parts and peeling of the skin when the teat is handled, indicates that an ascending gangrene, which soon implicates the whole quarter, has set in.

Local premonitory signs of gangrene are stiffening, change in colour, and coldness of the teat, which yields, when drawn, a thin stream of dark red, evil-smelling fluid.

While these changes are proceeding, the general symptoms become more grave: breathing is short and jerky, the pulse more frequent and almost imperceptible, and the temperature falls to subnormal.

Hitherto the animal has not been able to lie down for more than a minute or two, but now prostration is extreme, and in the absence of pain in the diseased quarter she goes off her legs, and soon dies intoxicated without a struggle. Cases are on record of animals having withstood gangrene of one quarter, the healthy tissue having managed to throw off the mortified part before lethal intoxication had occurred.

Death usually supervenes within thirty-six hours after the teat has become livid.

But this untoward sequel is rather rare, and the cow usually regains a fair measure of health and usefulness—even though abscesses form and burst externally in the diseased quarter—provided no spread of infection to the other quarters takes place.

Still, the latter frequently obtains, and the progress of the disease in the other quarters would indicate that infection had occurred directly from one quarter to another, due to the incompetence of the intervening partition, and now the cow becomes a total wreck.

It sometimes happens, in the event of the teat becoming plugged, or so indurated that the udder contents cannot be removed, that the entire quarter becomes a huge abscess, and when this is evacuated by puncture or ablation of the teat, its walls collapse like an empty bladder.

In a large percentage of cases that are properly treated the symptoms gradually decline, and the quarter, while yielding quantities of thick creamy discharge, continues to lose bulk, till after two or three months it becomes almost completely atrophied, and useless for all time.

Prognosis is unfavourable. We have never seen any restoration of function worthy of the name in a quarter that has run the gauntlet of acute mammitis.

The general symptoms of acute mammitis set up by inoculation of the skin of the udder are similar to those already described. Locally one or more quarters may be affected. There is a rapid infiltration of the subcutaneous tissue, producing a marked œdema, which pits on pressure. The parts are extremely tender on manipulation, and the adjacent limb is abducted. The skin is very tense, red, and hot, and the teat is much swollen. Occasionally the lymphangitis is not confined to the udder, but extends forwards on to the abdomen, and backwards, involving the perineum. Though the function of the quarter is in considerable abeyance, the secretion is not at first markedly altered; but this occurs as soon as the interstitial inflammation, by extension, is complicated with parenchymatous mammitis. Superficial abscesses occasionally form, and patches of thickened gangrenous skin may be thrown off, while, as in the mixed mammitis already referred to, there may be deep-seated abscess formation.

Prognosis in such cases is grave, though we have occasionally had resolution in the course of ten days in those instances where the inflammation did not exceed the bounds of the interstitial tissue.

Complications and Sequelæ.—We have already referred to gangrene, atrophy, and abscess formation. The first is the gravest complication, and the last the most common.

Pyæmia is a frequent complication while pus formation is proceeding in the quarter, and the *arthritic changes* (probably streptococcal) which occur are in every particular analogous to those which accompany some cases of suppurative metritis. Its onset is usually heralded by the patient's disinclination to rise, and when she is forced to her feet it is found that she is lame in one or both hind-limbs, and there may be also simultaneous lameness of both fore-limbs.

We have never witnessed paraplegia as a sequel, and think it likely that the paralysis recorded by some authors may only have been apparent, and the inability to rise really due to synovitis of the hocks, etc.

The seats of the lesions are constant—viz., the hocks, hind- and fore-fetlocks.

There is a diffuse inflammatory swelling on the inner aspect of one, and very soon of both hocks, and this proceeds till the hollow of the joint resembles a painful "bog-spavin," which causes the animal to paddle uneasily, and seek again the recumbent position. The fetlock lesions in all the limbs are usually confined to the postero-superior aspect of the joint, and in trying to find relief the animal attempts to stand on her toes. With suitable treatment the pain in the joints soon subsides, leaving an amount of diffuse thickening that gradually becomes absorbed. Neglect of these sequelæ leads to the formation of grievous bed-sores, the result of decubitus. Deep-seated abscesses of the limbs, etc., often necessitate the slaughter of the animal. Septicæmia, ending in death, is rare, unless after the advent of gangrene of the affected quarter of the udder.

III. Chronic Mammitis.—Though many cases of acute mammitis gradually reach a chronic stage, we prefer to limit the above term to that type of the disease in which the invasion of the quarter by the microbes (usually streptococci) and their effects on the tissues are so slow that a careless milker, handling the teats only, and that in deficient light, fails to detect the presence of multiple small nodules in the udder substance.

The **Period of Incubation** may extend over some months.

Morbid Anatomy.—A specimen obtained from a long-standing case feels irregularly indurated and lumpy. On section, it is fibrous, and presents a greyish-white appearance. There is much thickening of the interstitial tissue, but, owing to atrophy of the acini, the quarter is not increased, but may be much lessened in bulk. The sclerosis is nodular, and a catarrhal condition of the lining of the milk channels is apparent. Sections examined microscopically show the sclerotic condition clearly, and, when stained, reveal among the cellular débris the responsible streptococci. Occasionally slowly progressive abscess formation may be observed.

Microscopical Appearances.—These do not greatly differ from those of some cases of acute mammitis.

Desquamation of epithelium prevails in the less advanced parts, but in many places the basement membrane of the mucous coat is also removed, and well-formed granulations are projecting into the channels.

But the outstanding feature of udder tissue in which the pathological changes have been slow is extreme thickness of the trabeculæ, constituting a well-marked sclerosis.

Symptoms and Course.—The attack is so slow that there is practically no systemic disturbance. Immediately above the teat one or more nodules usually appear, and gradually increase in size, while in other cases the sclerotic foci are so numerous throughout the tissue that the quarter, when newly milked, feels like a bag of pebbles held together, yet separated, by a compressible stroma.

The milk, though a little less in quantity, appears to be at first normal in quality, but it does not keep, and the complaints which farmers receive from wholesale dairymen are often the result of mixing with the rest this apparently good milk.

As the nodules increase in size or become confluent, the morbid process becomes more catarrhal, the secretion is palpably unfit for use, being yellowish in colour and containing a few clots. If a little is centrifuged or allowed to settle in a test-tube, it yields an abnormal deposit of leucocytes, etc.; and when a stained smear is examined microscopically, it is found to be teeming with streptococci.

Exacerbations of the morbid processes now and again occur, accompanied by some constitutional disturbance, and during the height of the attack engorgement of the quarter (not so formidable as in acute cases) proceeds, with conglomeration of the nodules, till the quarter is indurated throughout. In this event abscesses may now slowly form, and if these communicate with the milk channels, the discharge can usually be removed by the teat, considerably lessening the bulk of the quarter; but if the line of least resistance is towards the skin, several succeeding abscesses may burst externally. In this way much of the udder tissue may be destroyed, and frequently the remainder is so much sclerosed that its secreting power is lost, and a varying degree of atrophy succeeds the exhaustion of the suppurative process.

In other outbreaks we get, instead of multiple nodules, a chronically progressive sclerotic induration of the parts, associated with dull pain and slow but radical alteration of the secretion. This type ends in more or less complete atrophy of the quarter, though occasionally the destruction of the glandular tissue is not absolute; and after the next calving the functional activity of some of the secreting cells is restored, and the quarter yields some little quantity of apparently normal milk.

The history of these outbreaks *in re* contagion is not so convincing as that of acute mammitis, but it is not uncommon to find 10 per cent. of a herd so affected, the outbreak lasting over a period of from one to two years.

Prognosis is unfavourable, for, though the patient does not suffer much in body, the disease is not amenable to treatment, and the complete loss of the quarter is almost a foregone conclusion.

DIFFERENTIAL DIAGNOSIS OF SUBACUTE, ACUTE, AND CHRONIC MAMMITIS.

The history of each outbreak will indicate the virulence of the organismal activity. To determine the potentialities of the first case, centrifuge a sample of the secretion, make smears from the deposit, stain, and examine microscopically. Sow some of the deposit on suitable media, retransplant the colonies if necessary, and test their pathogenicity by injecting an emulsion of them into the teat of a goat, sheep, or cow. If by injecting a guinea-pig subcutaneously with the culture we find it to be highly pathogenic, we may conclude that rigorous measures should at once be adopted.

PROPHYLAXIS.

Prophylaxis is of paramount importance. When dairymen fully realise the microbic origin of all cases of mammitis, they will less grudgingly adopt those preventive measures that are calculated to give them immunity from udder disease in the cowshed.

Every cowshed should be thoroughly disinfected at least twice a year, special attention being given to the hind-part of stalls. Avoid spilling of milk, and never at any time strip a diseased quarter direct to the ground. If anything of this kind occurs, scald and cleanse the place, then disinfect.

Clean overalls should be worn by milkers, to prevent soiling of hands through contact with dirty clothing.

Before each milking the teats and under surface of the udder of every milch cow should be swabbed with a cloth steeped in a mild antiseptic solution, and then dried. This measure protects the cows, makes the teats nicer to handle, and, if the other hind-parts of the cow are cleanly, renders the milk practically free from dirt deposit, so that it keeps well.

Milkers should have short finger-nails, and their hands should be well washed and disinfected before commencing, and as often as necessary during milking.

After milking, the teats, etc., should be dried with a clean cloth, and if any danger threatens in the byre, repeat the use of the antiseptic swab before drying.

When milking is over, the back part of every stall should, if necessary, be cleaned down, to remove effectively spilt milk or other discharges.

When possible, no dressing of sores on animals in the byre or out of it should be done by any milker. If a milker must perform this duty, the hands should be steeped in some antiseptic solution, washed with soap and warm water, and steeped again in the solution before handling a teat.

When the nature and incidence of the first few cases betray the operation of virulent microbes, the owner would be well advised to slaughter the victims, unless he is prepared to isolate them completely, and tell off a special nurse, who should not be allowed to handle the other stock. Then the main byre should be disinfected, especially the vacated stalls, and any litter there burned.

If the flooring of such stalls consists of pervious material, it should be dug up and new material put down.

As most of the udder and teat eruptions met with are infective between the cows, and predispose the tissue proper of the udder to disease, the affected animals should be quarantined under the conditions referred to above, and the parts treated with proper antiseptic dressings.

If for any reason the victim of mammitis or teat eruption is retained in the main byre, it should be attended to last, by one milker only, who should in the first instance empty the healthy quarters ; then the secretion from the diseased quarter should be drawn into a dish containing an antiseptic solution, and afterwards burned or buried.

Newly-bought animals should be quarantined till their udders are proved to be sound.

Avoid the use of infected teat-tubes, probes, or bougies. All such instruments should be made of material that will stand boiling or steeping in some reliable antiseptic. After use they must be well washed and wrapped in boric lint, and before using again should be boiled for five minutes, the point of the teat being disinfected before introducing any instrument.

As mammitis due to the use of an infected teat-siphon in the process of injecting either fluid or air into the udder not uncommonly occurs as a sequel to milk fever, care should be taken to sterilise the instrument prior to introducing it into the teats; also, when the udder is fully distended with air, precautions should be adopted to prevent undue pressure on the organ by attending to the position in which the cow lies, as bruising of the udder may act as a predisposing cause of mammitis (see Milk Fever, Vol. II.).

While we are not prepared to say that thorough milking of cows to the last drop, though commendable on the score of economy, is of great importance as a preventive of mammitis, we may assume its prophylactic importance when a quarter is the seat of latent incubation. For its accomplishment an expert milker should "after" (*i.e.*, finish the milking of) every cow, when he could take note of anything that was amiss, and attend to it.

The cautious dairyman will always eschew the buying of a three-teated cow, unless he is convinced that the deficient quarter is absolutely quiescent.

TREATMENT OF MAMMITIS.

Medical treatment is of signal service in subacute, but of little avail in acute mammitis. The animal should be comfortably and securely clothed, and, if attended in the shivering stage, should receive a diffusible stimulant of 10 ounces of whisky in water. Afterwards 3-ounce doses of sulphate of magnesia and 2 drachms of nitrate of potash in water may be given alternately every four hours; also antifebrin, 2 drachms placed on the back of the tongue with a dry spoon, and washed over with a little water. A daily dose of 1¼ drachms of 40 per cent. formaldehyde, well diluted with water, is to be recommended, or 1 drachm in 4 ounces of sterile water may be given intravenously.

Opiates and aconite are of service when pain is severe. The hydragogue action of a full dose of Epsom salt in robust animals is beneficial, and seems to lessen the force of the attack.

Locally.—Hot fomentations, gentle and prolonged friction, with mildly stimulating or anodyne liniments, with repeated stripping of the teat to remove retained secretion, are calculated to modify an acute attack, and at once start the reparative process in subacute cases.

We can recommend an embrocation in which some extract of belladonna has been rubbed down, or glycerinum belladonnæ, or Thompson's liniment, composed of belladonna, soap liniment, and glycerine. Remedies in common use by dairymen are soft soap and whisky, mustard and water, brine, treacle, etc.

Poultices of linseed or hops, etc., are useful, but they are difficult to apply, and instead of these we recommend the employment of the modern substitute for poultices—viz., a combination of kaolin and glycerine. A thick layer of this cataplasm should be spread over the quarters affected, over this put a layer of cotton-wool, and cover with a special suspensory bandage tied over the patient's back to support the weight of the udder. The cataplasm is to be renewed every twelve hours.

We have seldom derived benefit from the use of teat-tubes in mammitis, and disaster is the rule when attendants employ them.

Later on ointments of iodine or iodide of potassium may be applied, or tincture of iodine; and when acute pain and redness of the skin have subsided, and the quarter remains hard and indurated, a mild blister of biniodide of mercury may be used.

When gangrene supervenes, ablation of the quarter should be attempted before septic intoxication has occurred.

When it is no longer possible to remove accumulated pus or clotted material by the teat, the latter should be amputated at the root with scalpel or scissors. This gives perfect drainage, which may be assisted by copious and repeated injections of mild antiseptic solutions and by the employment of massage.

Abscesses pointing externally should be carefully opened, so as to avoid excessive hæmorrhage, and the cavity daily swabbed out with antiseptics. Antiseptic injections are held in great repute by some practitioners, but though the treatment is scientifically correct, we have found them impracticable in the first stages of acute mammitis, when the cow cannot tolerate the manipulation necessary to dislodge imprisoned milk débris, and the teat is liable to become indurated and useless if over-manipulated. When their use is admissible, the results are often excellent, provided great care is taken to thoroughly remove the contents of the quarter before injecting. The best injection is a 4 per cent. solution of boric acid at body temperature. The quarter is to be filled, but not too tightly, and should be manipulated during the operation. It may be repeated every second or third day, if considered necessary, but precautions must be taken to secure perfect asepsis.

When septic arthritic complications appear, a smart blister should at once be applied to the affected joints, in order to prevent decubitus, but the blistered surfaces must be suitably covered.

In the case of the hind-limbs, it is important to prevent such a calamity as contact of the teats with the blister; in the case of the fore-limbs, we must keep the cow from licking the blistered surface. These measures having been taken, she should be turned loose into a box; if this is not available, a string link must be put in her neck-binding, if this is an iron chain, and the dung channel levelled up. Both measures are precautionary against hanging, which is a not uncommon result of an awkward fall.

We have repeatedly known calves to thrive when put to cows having one or two quarters in a chronic catarrhal condition, and we have observed the salutary effect of this treatment on the udder. But we know of a healthy calf, which in an experiment was put to one of fifteen cows that had got over the painful stage of acute mammitis, and it lay sickly after each meal. It appeared to avoid the diseased quarter to a great extent, and after ten days showed arthritic complications, and had to be destroyed.

TUBERCULAR MAMMITIS.

At p. 136 the reader will find a full description of this type of mammary disease, which is so important from the public health point of view. Perhaps I shall be excused if I give here a short supplement of my experience as an inspector of dairy herds in Lanarkshire: 1·1 per 1,000 of Lanarkshire cattle were detected in 1910 as suffering from mammary tuberculosis; nine-tenths of these cases occurred in the hind-quarters, and in 90 per cent. only one quarter was affected.

Contrary to the experience of other writers, I have not always encountered the lesion in the upper part of the hind-quarter. Other favourite seats are, just behind the teat, the tumour causing the quarter to sag, or immediately in front of the teat, and extending backwards over its base. I consider the outstanding features of tubercular mammitis to be the occurrence of a single lesion, with well-defined though often irregular borders, and the absence of pain and of any direct adhesion of the tumour to the skin of the udder.

The appearance of the secretion is not reliable, though experienced bacteriologists can give fairly correct predictions from the naked-eye appearance of the deposit when the sample is centrifuged.

The absence of any history of injury or of an attack of mammitis is helpful.

Age Incidence.—It is seldom seen in animals less than five years old, though I have encountered it in a virgin heifer killed in fat condition, once in a three-year-old milch cow, and twice in four-year-olds. I have noticed that the tubercular quarter of a yeld (*i.e.*, a dry) cow usually contains a few ounces of yellow secretion, though the other quarters are quite dry. This yields a heavy deposit, and smears made from it, when stained, invariably show tubercle bacilli.

With the sanction of Dr. J. T. Wilson, County Medical Officer for Lanarkshire, and in co-operation with Dr. J. H. Patterson, County Bacteriologist, who had previously been interested in the question, some little work was done to determine whether fore, middle, or after milk was richest in tubercle bacilli.

Mr. James F. Taylor, M.R.C.V.S. (who assists me in dairy-herd inspection), and I revisited four cows condemned for tubercular mammitis, whose milk samples were positive on the smears. We procured multiple consecutive samples from each cow, and the results of Dr. Patterson's examination are set forth in the following extract from his section of the Annual Report of the Medical Officer of Health for 1910:

> **Tubercular Disease of the Udder of Cows**—*Milk.*—During the year 227 samples of milk from 225 cows with suspected disease of the udder were examined, 53 of which gave positive results, giving an average of 23·5 per cent. From the beginning of February, special attention has been given to the examination of smear preparations, in order that an immediate report might be given, instead of waiting three weeks for the animal inoculation test. Out of 44 specimens so examined, 31 were found positive. The milk in each case was stopped within two days of the taking of sample.
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> It might here be mentioned in connection with the examination of smear preparations that an elaborate and complicated process of staining is not necessary, as a good result does not depend so much on the process of staining as the time spent on the examination of the film. The method now adopted is the ordinary Ziehl-Neelsen, the film, after decoloration, being well washed in absolute alcohol to clear it up.
>
> All the samples giving negative smears were subjected to the animal inoculation test.
>
> Twenty-seven samples of milk were obtained from four of the positive cows, for the purpose of ascertaining whether the "fore," "mid," or "strippings" milk contained most bacilli; also whether the bacilli were more numerous in the cream than in the deposit. The samples were taken by the County Veterinary Surgeon.
>
> In each case the whole of the milk from the affected quarter was drawn off in separate consecutive samples, the last one always containing the last drop of milk procurable.
>
> Each sample, when received, was centrifugalised for fifteen minutes, and smear preparations made with one platinum loopful of the cream and of the deposit. The loopful was then thoroughly mixed on the slide and spread over an area the breadth of the slide and 1 inch in length. The films were stained by Ziehl-Neelsen's method. The count, which, of course, was an approximate one, was carried out by running the $\frac{1}{12}$ inch oil-immersion lens straight across the centre of the film, counting the bacilli in each field, and multiplying the total by 12 (the film being an inch in length and the lens used $\frac{1}{12}$ inch). Where the bacilli were few in number, the whole film was examined and counted. The results obtained were as follows:

Cow No. I.:

Sample.	Cream.	Deposit.
1.	not done.	negative.
2.	,,	,,
3.	,,	8 bac.
4.	,,	14 ,,
5.	,,	negative.
6.	,,	6 bac.
7.	,,	2 ,,
8.	,,	8 ,,

Cow No. II.:

1. Slight cream, ¼ inch deposit	7 bac.	660 bac.
2. ,, ,,	8 ,,	912 ,,
3. ,, ,,	3 ,,	1,380 ,,
4. ,, ,,	5 ,,	3,816 ,,
5. Very slight cream, ¼ inch deposit	5 ,,	2,196 ,,
6. ,, ,, ,,	4 ,,	636 ,,
7. ,, ,, ,,	2 ,,	1,104 ,,
8. Very watery. Thin layer of cream, 1 inch pus-like deposit	0 ,,	396 ,,

Cow No. III.:

1. Watery, slight cream, ½ inch pus-like deposit	—	168 bac.
2. ,, ,, ,,	—	276 ,,
3. ,, ,, ¼ inch ,,	—	192 ,,
4. ,, ,, ,,	—	252 ,,

(Milk from quarter only amounted to about 2 ounces.)

Cow No. IV.:

1. Watery, slight cream, ¼ inch deposit,	120 bac.	5,172 bac.
2. ,, ,, ,,	1,020 ,,	6,804 ,,
3. ,, ,, ,,	1 212 ,,	5 916 ,,
4. ,, ,, ⅛ inch ,,	1,584 ,,	14,460 ,,
5. ,, very slight cream, ⅛ inch deposit	364 ,,	10,908 ,,
6. ,, heavy cream, $\frac{1}{16}$ inch deposit	2,532 ,,	5,316 ,,
7. ,, ,, ¼ inch blood-stained deposit	1,824 ,,	4,824 ,,

It will be seen from the results obtained from Cows I., II., and IV., that the "mid" milk was richest in bacilli, and would evidently be the proper part to take when such a milk was to be examined, more especially by smear preparation. No reliance could be placed on the samples taken from Cow No. III., as the milk was too small in amount.

From the samples taken from Cows II., III., and IV., it was clearly shown that the deposit is the proper part of the specimen to take for the examination, it containing by far the greater number of tubercle bacilli.

ACTINOMYCOTIC MAMMITIS.

Numerous investigators have shown that the udder of the cow is not infrequently attacked by actinomycosis. Its occurrence in the pig has been well recognised by many Continental writers, and both Bang and Rasmussen are credited with seeing fifty-two cases in three months. Presumably both saw the same series.

In the cow it is probably more common than is generally supposed. We have often had reason to suspect actinomycosis when samples of milk taken were declared negative for tubercle. Dr. J. H. Patterson, County Bacteriologist for Lanarkshire, records that out of fifty specimens of udders examined for tubercle, the lesions in five cases were due to actinomyces. It is, therefore, very likely that a fair percentage of udders suspected of tubercle, but which prove negative, are the seat of actinomycosis. Dr. Patterson has also on a few occasions found in milk samples, elements suggestive of actinomyces.

These results indicate a real danger to the human subject. It would be well if more udders affected with nodular induration were submitted to microscopical examination.

Lesions.—When manipulated, the affected quarter cannot easily be differentiated from tubercular infection, but often there are many lesions in the quarter, whereas in tubercular mammitis a single lesion is the rule. Section of the organ reveals many yellow-coloured foci, and the surface has a brownish tint.

Microscopical examination of ordinary smears often shows fragments of club-shaped elements, but the fungus is readily demonstrated when sections cut in paraffin are stained with Ziehl-Neelsen's carbol-fuchsin.

BOVINE MAMMITIS AND PUBLIC HEALTH.

We need not do more than mention the undeniably great danger to the consumer which exists in milk containing tubercle bacilli. Naturally, its disease-producing potentialities are greatest to the infant, the child, and the fever-stricken patient with lesions of the digestive mucosa, for all of whom milk is the staple diet.

In the present state of our knowledge it is not possible to estimate to what extent, if any, public health is influenced by the occurrence in cows of mammitis other than tubercular mammitis. Many outbreaks of " septic sore throat " in man have been laid at the door of the cow on no stronger evidence than that swabs from the patients' throats have furnished the bacteriologist with micrococci similar to those that were plentifully found in the secretion of the diseased udder of one or more members of the indicted herd.

The circumstantial evidence often produced does not bear close investigation, and while we think that medical officers of health, in carrying out their responsible duties, are entitled to put out of the market for a time any supply of milk that can in the remotest degree be suspected of causing an outbreak of disease, we know of no case in which it has been clearly proved that the cows were culpable from the occurrence of mammitis in the herd.

Nevertheless, when we consider the many species of bacteria that are found in apparently healthy udders, and are capable of demonstration in the milk, and, further, that a great many micro-organisms can under favourable circumstances attack the vitiated mucous membrane of the udder, we must admit the possibility of danger to the throat as well as to the digestive organs following the use of milk that is grossly contaminated with such bacteria. Such milk does not keep well, and no dairyman would knowingly contaminate his general supply by adding to it material that in a few hours would render the supply palpably unfit for use. But the carelessness of some milkers, the want of proper light in the byre, and the great haste which characterises the early-morning milking of the cows in some herds, all contribute to the possibility of a milk-supply being contaminated by the admixture of the abnormal secretion of a quarter affected with incipient mammitis, or it may be of a little material from a suppurating quarter.

The apparently normal secretion of unaffected quarters in a case of mammitis is sometimes abnormally rich in deposit and bacteria. We think a dairyman might reasonably be called upon to withhold such milk, boil it, and use it for some other purpose than human consumption. Perhaps, when such an order is made general, the producer may successfully plead for compensation.

A meat inspector must be very careful in adjudicating on carcasses of animals which have recently suffered from an attack of mammitis. In judging the merits of the carcass he must be very much guided by the state of the udder. Unless the animal is killed within twelve hours of an acute attack, she is not likely to be in such a condition that her carcass will pass inspection. It is only when the disease has entirely subsided, and the animal has for some time regained her general health, that the carcass is fit for market.

Carcasses must be seized when the animal has been killed in a feverish or sickly condition of more than a few hours' standing, when the udder appearance indicates advanced mammitis or any tendency to gangrene, or when there is evidence of any degree of septicæmia.

"In June, 1889, in Cotta, in Saxony, 136 persons, of whom 4 died, were affected with meat-poisoning after eating the flesh of a cow which was slaughtered on account of having suffered from a serious attack of mammitis. This flesh was poisonous both in the raw and the cooked state." At the present time the rough-and-ready methods of inspection of such carcasses in our British abattoirs leave much to be desired.

We must entirely agree with Professor E. J. McWeeney, of Dublin, who, in an excellent paper on "Meat-Poisoning: its Nature, Causation, and Prevention," published in vol. i. of the *Journal of Meat and Milk Hygiene*, and read at the Dublin Health Congress in August last, urges the pressing need of a more scientific modern practice of bacteriology in the abattoir. In this paper are described methods of determining within a few hours the presence in flesh of the special bacilli that are notorious in causing meat-poisoning.

GANGRENOUS MAMMITIS OF EWES.

This disease has been observed in French flocks for over half a century, and it is occasionally seen in Germany and in Italy. French shepherds and others have called it "mal de pis," "araignée," and "cru," and in those districts where milch ewes are kept it claims many victims every year, and the affection usually appears soon after lambing. The disease also occurs in England, and is generally termed "black udder," from the blackish or purplish appearance of the affected glands. Gilruth studied the affection in New Zealand (*Veterinary Journal*, 1910, p. 648).

Etiology.—The cause is a very small micrococcus discovered in 1875 by Rivolta, and isolated and thoroughly studied by Nocard in 1887. It stains readily with any of the aniline dyes, especially by the methods of Gram and Weigert, and has no tendency to form chains. It may occur singly or in colonies, and can be cultured in broth, milk, or on potato or gelatine. Infection occurs by the teat canal or by means of a skin wound. Bridré thinks this micro-organism occurs normally in the mammary gland, and that mammitis only declares itself after a lesion of the interior of the gland has occurred. This enables the microbe to invade the tissues. According to Besson, the goat and laboratory animals are not receptive to the micrococcus.

Morbid Anatomy.—The skin is infiltrated, the peri-mammary connective tissue is œdematous, and is followed by emphysema and gangrene. The red serosity which escapes when the part is incised abounds in micrococci, and the tissues have a parboiled appearance.

Diagnosis.—The symptoms are so striking that the practitioner has no difficulty in diagnosing the disease.

Prognosis is unfavourable. The disease is almost invariably fatal, unless the affected part is timeously amputated.

Symptoms and Course.—The general and local symptoms of acute mammitis are developed simultaneously. The affected part, usually one gland only, is hot, painful, and engorged, and the secretion changes to yellow and then red, as in gangrenous mammitis of cows. In a short time dark violet-coloured patches appear and extend, and the gland becomes cold, insensible, and less bulky.

Symptoms of acute septicæmia soon appear, the animal goes off its legs, grinds the teeth, shivers, and dies in from two to three days. Before death the temperature falls considerably below the normal.

In very exceptional cases localised gangrenous patches are shed, leaving unhealthy, slow-healing ulcers; and though the animals may survive, they cannot nurse their young, and are so very weakly that they do not readily fatten for the butcher.

Treatment.—Rigid prophylaxis should be carried out. The affected animals should be isolated, and the fold disinfected.

Surgical treatment, consisting of complete ablation of the affected part, is the only measure worth trying. This in the hands of Esser and Moussu has been very successful. The diseased part is simply dissected away, bloodvessels are ligatured, and though a large wound results, it heals kindly under antiseptic dressings. Bridré immunised ewes by inoculating them with a vaccine prepared from a culture in bouillon. attenuated by three to four months' sojourn in a stove raised to 37° C. The dose given was 0·5 c.c. Immunisation was obtained at the end of a fortnight. The same authority found that hyperimmunised sheep possess a serum having preventive properties.

GANGRENOUS MAMMITIS OF GOATS.

Gangrenous mammitis of goats is similar in almost every respect to that described as occurring in ewes. According to Nocard and Leclainche, the mammitis in the goat observed by Mathis and afterwards by Leblanc and Moussu is identical with that of the sheep.

PURULENT MAMMITIS OF GOATS.

Purulent mammitis of goats has been observed by Leblanc, Gillibert, and Bossi-Virginio. Like the gangrenous form, it usually occurs soon after kidding. The slow developing attack results in engorgement of the gland, which is painful, hot, and red. Constitutional symptoms are well marked, the milk changes in character, and soon becomes purulent and evil-smelling. Eventually abscesses form, and burst externally, discharging large quantities of pus.

The disease is supposed to be due to a diplococcus which has a tendency to chain formation.

Treatment consists in the opening of the abscesses and swabbing out the cavities with antiseptic and astringent solutions.

MAMMITIS IN MARES.

The udder of the mare is comparatively seldom the seat of disease, and cases of mammitis in this animal are generally sporadic. Few practitioners have had much experience of equine mammitis, and, consequently, very little has been written regarding it. J. F. Healy reported, at a meeting of the Irish Central Veterinary Association,* that during the months of June, July, and August, he had encountered twenty-three cases of mammitis in mares within an area of ten miles.

The disease may set in a few days before foaling, indicating the probability of some pre-existent, latent infectivity in the gland, or it may occur at any time during the lactation period, provided the organ is subjected to such ulterior conditions as are calculated to lead to microbic invasion of it.

But the udder of the mare is immune from many circumstances that predispose the cow to mammary disease—*e.g.*, milker's hands, soiled stalls, etc.—nor do her teats and udder rest on the ground, as in the case of the cow when she is recumbent.

Occasionally cases are met with in young virgin mares, also in brood mares after their foals have been weaned, and the disease has been observed in pregnant mares a short time previous to foaling.

When mammitis occurs in the mare, the causal explanation is often to be found in some diseased condition of the foal—*e.g.*, strangles, stomatitis, or it may be a suppurating sore on the face or other part of its body that can come into contact with the mammæ.

Bermbach records two cases that occurred in mares suckling foals affected with strangles.

From the mammary secretions of a mare suffering from mastitis Lucet isolated an organism which he called the *Streptococcus mastitidis equi*, and this, when injected subcutaneously in the horse, caused abscesses.

Parent and Schutz have also isolated a streptococcus from the pus of a case of equine mammitis.

Symptoms and Course.—The mare is ill at ease, she resents the attentions of the foal, and, when moved, exhibits a stiffness or lameness simulating that of a gelding suffering from scirrhous cord. She refuses food, rigors may be present, the pulse is full and bounding, and the temperature 103° to 105° F.

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* *Veterinary Record*, January 24, 1903.

Locally there is swelling of the affected part, which is hot, shining, tense, and extremely painful. One or both mammæ may be involved. The swelling often extends forward on the abdomen and backward between the thighs. The teat of the affected side is also engorged, stiffened, and enlarged, and yields, when drawn, some thin reddish serum having a fœtid odour. The character of the secretion changes during the progress of the disease, as in acute bovine mastitis.

Though occasionally mares make a good recovery in from two to three weeks, the majority of cases end in abscess formation or induration, and occasionally gangrene follows.

Abscess Formation.—One or more abscesses may form, and their evacuation gives the patient considerable relief. Sometimes after the bursting of a well-formed abscess no others are formed, and the gland becomes changed into a hard, unyielding mass, which gradually becomes less sensitive to manipulation.

Often, however, the formation of abscesses continues for some time, and we have known a neglected case in which eighteen months after the attack there were several discharging in the region of the gland and surrounding tissues, but this we diagnosed as a botriomycotic condition.

Gangrene supervenes in some cases, causing death, but occasionally the dead part is sloughed off and the animal recovers.

Prognosis is unfavourable. The mare is unfit to nurse her foal, which must have a foster-mother or be hand-reared, and the mammæ are usually so utterly destroyed that she is useless for the future rearing of offspring.

Treatment—*Systemic.*—The patient must be comfortably housed and clothed, and put on simple laxative food. A dose of physic should be administered at once, with the object of lessening the activity of the organ. Febrifuges, opiates, and internal antiseptics, should be given when indicated.

Local.—Prolonged fomentations with warm water containing an antiseptic and repeated stripping of the teats should be followed by thorough drying of the parts with a soft clean towel; then the whole of the organ should be heavily smeared with a cataplasm composed of kaolin and glycerine, and covered with cotton-wool, which can be kept in position by a suspensory bandage. Repeat in twelve hours, and as often as necessary.

Linseed or hop poultices containing tincture of opium or belladonna may be similarly applied, or the parts may be smeared with anodyne ointments, or well rubbed with a camphor and belladonna liniment.

Abscesses should be opened as soon as possible, and when the condition of the mammæ and the patient warrant it, the whole organ may be removed surgically. This operation has been performed with success by Scott of Bridgwater; by Monconet, Violet, and Cadiot, in France; and by several German practitioners.

MAMMITIS IN BITCHES.

While acute mammitis cannot be considered a common disease of the bitch, a chronic type of the disease is frequently met with.

Acute cases usually occur soon after parturition, and are the result of the entrance by the teat of virulent pathogenic bacteria.

The disease is usually seen in the anterior glands, and one or more of these may be affected. Such cases usually end in abscess formation and destruction of the glands affected, but occasionally the disease becomes chronic.

The affection is not uncommon in instances where milk is retained in the udder as the result of the pups having died, or being removed, and the animal neglected.

Pyæmia with arthritic or other complications may result. Lucet and Leblanc have isolated a staphylococcus from the diseased secretions.

Symptoms.—The animal is seldom on her feet, refuses food, and is feverish. The glands affected are swollen, œdematous, hot and painful, and the secretion is altered. Abscesses form, burst, and discharge, and the ulcers and fistulæ that result heal very slowly.

Chronic mammitis, when met with in the anterior glands, is usually the relict of acute mammitis, but when seen in the posterior glands may be the result of injuries, which, being unobserved and neglected, provide a venue for the invasion of bacteria. Gray has found that this condition is often due to the recurrent lactation so frequently observed in the non-pregnant bitch, and arising about nine weeks after her "heat." The type of inflammation set up is the chronic interstitial, and the irritation of the organisms slowly leads to a sclerosis of the mammary tissue, which is often spoken of as a fibroma.

Prophylaxis.—Cleanliness in the kennel, removal of long hairs in the region of the mammæ, disinfection of the parts before and after parturition, daily examination of the mammæ, and antiseptic treatment of all sores and scratches on the organ.

Treatment.—Give a purgative, such as grey powder; remove the offspring to a foster-mother; foment the affected parts with warm water (to which an antiseptic has been added) while the patient stands in the tub containing the solution; unload the mammæ by hand, dry, and apply a cataplasm of kaolin and glycerine, kept in position by means of cotton-wool and bandages. Later the parts should be rubbed with an anodyne ointment or liniment, and the abscesses which form should be opened as early as possible. Unsightly and pendulous sclerosed glands may be extirpated without fear of recurrence.

CONTAGIOUS AGALAXIA.

By HENRY GRAY, M.R.C.V.S.

Synonyms.—*French :* Agalactie infecteuse ; Agalaxie contagieuse.
German : Infektiöse Agalactie.
Italian : Agalassia contagiosa ; Mal del sito ; Mal del asciutto.
Spanish : Stornarella.

Definition.—It is a general contagious disease affecting sheep and goats, and characterised by inflammatory lesions in the mammary gland, eye, and joints. There is generally an absolute loss of milk secretion.

History.—This disease was first described in 1816 by Metaxa as occurring in sheep and goats. It was afterwards found in goats in Switzerland by Zangger. In 1862 Dinella and Provinzano studied it in Italy. In 1871 Brusasco described it under the name of "contagious agalaxia," which Oreste showed was the same disease as the "stornarella" of Metaxa. Rocco Marra published an account of its occurrence in Italy in 1891. Barthélemy described it in France under the name of "Rhumatisme arthritique" in 1894. Schossleitner in the Tyrol gave short descriptions of it in 1895. Hess and Guillebeau gave a good account of the eye lesions and the disturbance of the milk secretion in 1893. Oreste, who had studied the disease in Italy in 1882 and again in 1886, undertook, in collaboration with Marcone, a series of experimental researches, of which the results were published in 1896. During the same year Bournay studied the disease in the goat in the Eastern Pyrenees. Nocard and Leclainche give a good summary of it.

Etiology.—Although the disease is mostly encountered in the goat, it is also observed in the ewe. Age plays no predisposing part in its causation. It commonly makes its appearance in spring, and lasts during the summer, to disappear when the cold weather sets in. According to Brusasco, Marra, Labat, Bournay, and others, a diseased animal introduced into a flock or herd contaminates the healthy. But, on the contrary, Oreste, Hess and Guillebeau, and others, assert that close and prolonged cohabitation does not transmit the malady. The affection seems confined to certain localities, where it permanently remains, or appears after more or less long intervals. In some flocks or herds one-half, a fourth, or a tenth of the animals are affected, whilst the others escape. As a rule, the lesions start at the mammary gland, which is thought to be contaminated by the soiled ground or by the hands of the milker. Infection sometimes passes from dam to fœtus, which, according to Labat, shows at birth characteristic lesions.

Bacteriology.—Although various micro-organisms have been found from time to time in the lesions, the disease has been proved to be due to an invisible virus, and thus the causal organism belongs to the class of ultravisible microbes. Probably transmission of the disease takes place by the agency of flies or other insects.

Symptoms.—The manifestations of the disease may be divided, for the sake of description, into *acute* and *chronic* forms.

Acute Form.—This commences with high fever, great dulness, and loss of appetite. The animal seems tired, cannot keep up with the rest of the flock or herd, and remains in the recumbent position for a long time. In some cases the disease is so intense that the animal succumbs in a few days before any lesions have had time to appear.

The mammary gland is nearly always affected; it becomes swollen, hot, and painful; the small quantity of milk drawn off has a serous, clotted, dirty white or yellowish appearance; sometimes abscesses form in the gland; and in the majority of cases the inflammation gradually declines, leaving the organ supple and soft, or hard and atrophied, and containing here and there hard knots.

When the joints become involved, there is marked lameness. The knee and hock are especially liable; then the stifle, shoulder, hip, and fetlock. The symptoms are those of arthritis or periarthritis; progression is difficult or impossible; the animal remains lying; the affected region is swollen and painful; abscesses form and burst, and are followed by persistent suppuration and ankylosis.

When the eyes become involved, there is an intense external ophthalmia; the cornea becomes turbid and milky, and vessels radiate from the sclero-corneal region; the conjunctiva is swollen. Later on, the circum-corneal injection takes on a dark red tint, whilst the central part remains greyish or white. Suppuration invades the anterior chamber; ulcerations appear on the cornea; the eyeball becomes enlarged, tense, and painful; sometimes the cornea ruptures; the iris is pushed into the breach and projects, forming a staphyloma; the lens becomes dislocated, and sight is lost. In other cases the lesions remain circumscribed; the corneal ulcerations fill up, and the exudate in the anterior chamber is reabsorbed. It runs its course in three to four weeks. The mortality is about 15 per 100. The lesions become attenuated, and pass into a chronic state in the majority of instances.

Chronic Form.—The secretion of milk diminishes; the liquid becomes serous and yellowish-white; it contains small coagulated masses; collected in a test-tube, it shows on standing a greyish, viscid deposit, forming about a fourth of the total; immediately above it there is a thin, reddish layer formed of red blood-corpuscles ; the upper part is occupied by a serous fluid. In some animals the milk coagulates into a mass in the mammary gland. The taste of the milk is salty, and sometimes bitter. Cheese made from such milk is of bad quality, dry, sparse, and lacks cohesion ; on section it shows innumerable small grooves on the cut part, whereas normal cheese is hollowed out with a few large vacuoles. The mammary gland is partially or totally involved ; the affected region undergoes a gradual atrophy, and shows indurated knots.

The affected *joints* show signs of subacute or chronic arthritis, with swelling of the epiphyses and dropsy of the bursæ. Movement causes severe pain ; the animals move about on their knees or trail the hinder parts on the ground ; others remain constantly lying on the ground.

The *eyes* show simple corneal opacities, or a parenchymatous keratitis with consecutive ulcerations. There is, also, often an internal ophthalmia, with its possible complications of suppuration of the anterior chamber, synechiæ, staphyloma, and dislocation of the lens.

The various localisations coexist in the majority of the affected animals ; in others they are differently associated ; the alterations of the mammary gland may be absent in the milking females ; in a few cases the joints or the eyes remain free. Abscesses have been found in the parotid gland, mediastinum, muscles, and other parts. Rams and he-goats sometimes show an orchitis. The animals, in consequence of their emaciation, are valueless. The majority of them have persistent lesions which render them useless (Oreste and Marcone, Hess and Guillebeau, Nocard and Leclainche, etc.).

Lesions.—The internal organs are healthy, and the lesions remain localised. The wasted mammary gland contains here and there fibrous knots, badly defined or absolutely sclerosed. The joint lesions do not show any special characters ; they vary according to the rapidity of their course ; they often are manifested by the wear of the cartilage of incrustation and in true or false ankylosis. The eye shows various lesions of the cornea, signs of iritis or irido-choroiditis.

Diagnosis.—The coexistence of two or more of the usual localisations will enable one to differentiate it from the ordinary gangrenous mammitis. Again, the filtered virus which is sterile on ordinary culture media transmits the disease. It is not likely to be mistaken for Mediterranean or Malta fever.

Treatment.—The disease should be treated on the same lines as other contagious diseases. The local treatment should be carried out according to the ordinary rules adopted for the eye, joint, and mammary localisations.

THE HÆMORRHAGIC SEPTICÆMIAS

BY G. L. INGRAM, M.R.C.V.S. (THE BROWN INSTITUTION, LONDON),
AND E. WALLIS HOARE, F.R.C.V.S.

THE term "hæmorrhagic septicæmia" should include any condition in which the causal organism multiplies in the blood of the affected animal, and gives rise to symptoms of general poisoning, with the formation of submucous, subserous, or subcutaneous hæmorrhages, but without causing abscess formation—*e.g.*, anthrax in cattle. But it has come to be used to denote certain diseases occurring in almost all the domesticated animals, in small rodents, in fowls, and in many wild animals, caused by organisms which are spoken of as being "of the fowl cholera type," the disease of poultry known as "fowl cholera" being the first in which this type of organism was recognised.

Various names have been proposed for these organisms. Continental authors, following Lignières and Trevisan, call them "pasteurella," and though this is not inappropriate (fowl cholera being the first disease against which Pasteur used his method of protective inoculation), it is unscientific and likely to lead to confusion. Hutyra and Marek use the term *Bacillus bipolaris septicus;* Cadéac terms it the "ovoid" bacterium. The names *Bacillus bovisepticus, B. avisepticus,* etc. (Hertzog, Kinsley), according to the animal affected, seem to be preferable until the question of separate identity has been proved.

Horses, cattle, sheep, swine, goats, dogs, cats, rabbits, mice, rats (*B. pestis*), buffaloes, deer, and other wild animals, may become affected with diseases due to these organisms. It is possible that the outbreak of cattle disease described in the *Veterinarian* for 1854, and unsuccessfully investigated by Spooner, was of the nature of the German "Rinderseuche."

Canine distemper is among the diseases which have been ascribed to this class of organism, but wrongly so, as this affection is now generally believed to be due to an ultravisible virus. Lignières states that the disease of dogs known to English veterinary surgeons as "Stüttgart disease," and in Germany as "Hundetyphus" or "gastro-enteritis hæmorrhagica," is due to a bacillus of this type.

Fowl cholera has been known as an infectious disease of poultry since the end of the eighteenth century, and has been described in Russia and other parts of Europe, and also in the East Indies, whilst bubonic plague, due to the *B. pestis*, and still very prevalent in China, was the Black Death of the Middle Ages.

The *B. avisepticus* was first described by Rivolta and Semmer in 1878, and later by Pasteur in 1880. Bollinger, in 1878, described a disease of cattle and deer ("Wild und Rinderseuche"), and Kitt, in 1883, discovered an organism of this type as the cause.

Smith and De Schweinitz, in 1886, attributed swine plague ("Schweineseuche") to one of these cocco-bacilli. With regard to this disease, however, although Hertzog* confidently describes it as due to an organism of the fowl cholera group (*B. suisepticus*), there seems to be some doubt, especially as experiments by Ostertag and Wassermann, working independently, have shown that a vaccine prepared from one strain of this organism is of no use against another strain. Polyvalent sera from different strains also seem to have yielded inconstant results.

No certain results have been arrived at or published showing whether these so-called "organisms of the fowl cholera type" are separate entities or identical, and their clinical variations only due to passage through different animals. "The organisms of septicæmia hæmorrhagica in wild game and cattle, swine plague, rabbit septicæmia, and fowl cholera, are characterised by an almost complete agreement in morphological, biological, and experimental pathological properties" (Baumgarten). These properties, as well as the general type of symptoms which they produce, would suggest that they have a much nearer relationship than the bacilli which are collectively spoken of as "acid-fast."

The bipolar staining bacilli of the hæmorrhagic septicæmias are variously described as "cocco-bacilli," "diplococci," and "bacilli." The small forms under the highest magnification of the microscope are only just visible, but the larger usually show some distinctly bacillary forms. The organisms are as a rule from 0·3 μ to 0·6 μ in length, and about 0·1 μ to 0·5 μ in breadth. They are round or more commonly oval in form, are non-motile, and possess no flagella. They are present in the blood of affected animals in enormous numbers, and may even outnumber the erythrocytes. They stain well with the ordinary aniline dyes, and when lightly stained, or after decolorisation with weak acetic acid, the staining is seen to be more intense at the poles, leaving an unstained or only faintly stained central portion. This has given rise to their being mistaken for diplococci. The bipolar method of staining is the most characteristic staining feature, as they are not coloured by the method of Gram or its modifications.

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* "Disease-producing Micro-organisms," 1911.

These organisms show some very slight differences in their cultural characters, but as a rule grow well on nutrient gelatine or in broth, though not well on ordinary or glycerine agar, and in most cases there is no growth on potato. The media should be faintly alkaline. Gelatine, on which they form small white colonies, is not liquefied. After a short period of growth in broth the medium is rendered slightly turbid, the small organisms being held in suspension for some time, but the growth is afterwards deposited at the bottom of the tube. The cultural characters are not really distinctive on any known culture medium.

To laboratory animals these organisms are very fatal. Mice and rabbits inoculated intravenously or intraperitoneally with the smallest possible quantities of infective material die in twelve to thirty-six hours. They show marked tracheitis, with a brilliant scarlet-red coloration of the laryngeal mucous membrane (Kitt). The guinea-pig is more resistant to some strains.

The organisms are not very resistant to heat. The *B. pestis* is said to be killed by exposure to a temperature of 70° C. for an hour; the bacilli isolated from fowl cholera and " deer and cattle disease " by 55° C. for fifteen minutes, or 80° C. for ten minutes (Ostertag). Differences of opinion exist as to the length of time the organisms live outside the body. The fowl cholera organism can exist some time in soil; in manure for over a month (Gärtner); in putrefying cadavers for three months, according to Kitt. Desiccation soon destroys them, and they are easily acted on by disinfectants, such as 1 : 500 hydrochloric acid or 1 per cent. carbolic acid.

It seems quite possible that some forms of the organism are saprophytic and live in soil, becoming heightened in virulence by accidental introduction into a wound, and thus originating an outbreak.

The usual method of natural infection seems to be through the intestinal tract. Soiling of grass, fodder, etc., with infective material takes place in all these hæmorrhagic septicæmias, on account of the diarrhœa which is an almost constant symptom.

But natural infection may also occur through abrasions of the skin (exanthemic form of deer and cattle disease) or by inhalation (pectoral form). In the case of fowl cholera it is quite possible that lice or other parasites may convey the disease from one bird to another, especially in view of the fact that the rat flea (*Pulex cheops*, Rothschild, or *P. Philippinensis*, Hertzog) is known to convey the *B. pestis* from rat to rat and from rat to man. Natural infection in bubonic plague is said to take place through the skin, but probably inhalation is responsible for the more virulent pneumonic type.

The diseases caused by these organisms are all characterised by rapid onset of high fever, depression, and diarrhœa, and usually end fatally in a very short time. The post-mortem changes observed are intense inflammation of mucous and serous membranes and small hæmorrhages.

German veterinary police regulations for hæmorrhagic septicæmia are the same as for anthrax, so that in any case in which an animal dies showing symptoms which suggest the cause of death being anything of the nature of a hæmorrhagic septicæmia, strict disinfection of stables, cowsheds, fowl-houses, etc., should be carried out, and the bodies of dead animals either buried in lime or, in the case of small animals, burnt, together with all bedding, fodder, or other possibly infected material.

Treatment of outbreaks of these diseases is practically useless. Friedberger tried phenol and salicylic acid with no success, and, indeed, it is doubtful whether treatment is advisable. Probably the slaughter of all affected animals and the isolation of all in-contacts is the best method of dealing with an outbreak. It is stated of the *B. pestis* that curative sera which have been prepared have given no good results in cases in which the disease has been sufficiently far advanced to make diagnosis certain. A successful vaccine against fowl cholera has been prepared by Pasteur, but has not been widely used. Smith and De Schweinitz, in 1892, claimed success in inoculating against the *B. suisepticus*, but Ostertag and Wassermann have thrown doubt on their assertions.

In a disease of buffaloes occurring in Italy, known as "barbone," and similar in almost every respect to hæmorrhagic septicæmia in cattle, Oreste and Armanni state that by using an attenuated culture obtained by passage through pigeons they were able to reduce the mortality (in about 2,000 animals treated) from 45 per cent. to about 6 per cent.

No case of disease in man caused by the consumption of the flesh of animals dying of, or killed whilst suffering from, any of these diseases has been reported. However, the carcasses of such animals should be totally condemned, as the flesh is fevered and unfit for the food of man.

SEPTICÆMIA HÆMORRHAGICA IN THE OX.

Synonyms.—Infectious broncho-pneumonia.

French : Pneumo-entérite (Galtier); Broncho-pneumonie infectieuse (Nocard); Pasteurellose du bœuf.
German : Rinderseuche (Hueppe).
American : Cornstalk disease (Billings).

General Remarks.—The affection was observed by Bollinger in 1878. In Germany it was studied by Kitt, Frank, Reuter, Jöhne, Buch; in Switzerland, by Guillebeau and Hess; in Denmark, by Jensen; and in France, by Galtier.

Septicæmia hæmorrhagica of the buffalo is known as "barbone." It was described by Condamine in 1867 as occurring in Cochin China, and in 1869 by Germain and recently by Yersin in the same country. Pease studied it in India in 1898. It occurs frequently in Italy, Hungary, and Egypt, and can be transmitted from the buffalo to the ox.

Outbreaks have been observed in stags, wild boars, roebucks, and deer in Germany, and to the disease in these animals the term "Wildseuche" was applied by Bollinger.

The disease in cattle ("Rinderseuche"), in wild animals ("Wildseuche"), and in buffaloes ("barbone") depends on a similar micro-organism, and presents similar symptoms and lesions.

In America the disease has been met with by various observers. In 1900 several enzoötic outbreaks occurred in Minnesota, and were investigated by Brimhall and Wilson; also by Nörgaard in Tennessee. Law has observed the disease in cattle in New York on several occasions, and also states that it occurs in the Mississippi Valley.

So far as is known, the affection does not occur in the British Isles at present. We have, however, frequently met with cases in calves and cows which closely resembled the œdematous form of septicæmia hæmorrhagica (see p. 415).

Bowhill investigated the disease in South Africa in 1906.

The affection known as "cornstalk disease" in America was attributed to cattle being turned into stalk-fields in the later fall and early winter months, so as to glean up the remnants.* According to Billings, who investigated the affection in Nebraska, it is not the cornstalks, but the leaves and tender top shoots, that are eaten by the cattle, and were believed to indirectly cause the disease in question. He found that the real cause of the disease was a bacillus of the fowl cholera type. Beyond the fact that the affection generally appeared at the time when cattle were allowed access to the stalk-fields, and that the corn-leaves from corn fodder (brought from a district where the disease was rife), on being given to a rabbit, caused death, and the specific micro-organisms were isolated from its body, the rôle of the corn fodder as an etiological factor does not seem to be established. We shall refer to this affection later on (see p. 416).

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* Bowhill, "Cornstalk Disease in Cattle," *Veterinary Journal*, 1892.

Etiology.—The disease is caused by the "ovoid bacterium," also known as the "bipolar bacterium," a micro-organism of the fowl cholera type. The characters of this organism have already been described (see p. 408).

The disease occurs most frequently during the winter months, and may appear in a sporadic, enzoötic, or epizoötic form. Young animals are most susceptible. All parts of the infected body are virulent, including the blood, saliva, urine, milk, and dejecta.

The specific bacillus may be present in soil, especially in damp soils, marshy districts, etc. According to Kitt, it can flourish and increase in water contaminated by organic material.

The commonest mode of infection is by means of contaminated food, which infects the pharyngeal mucous membrane, either through erosions existing in this structure or through wounds of the mucosa caused by prickly particles of forage. Thus are explained the early symptoms so often met with of œdematous lesions of the posterior aspect of the mouth and of the pharynx.

According to von Ratz, infection by way of the intestine is not common except in the case of animals endowed with little powers of resistance. Infection by inhalation has not been established.

Symptoms.—Although various types of the disease are recognised, there are certain general symptoms common to all. These appear suddenly, and include marked constitutional disturbance, rigors, accelerated pulse and respirations, a temperature of 105° to 106° F., injection of the visible mucous membranes, cessation of rumination, painful mastication, a flow of viscid saliva from the mouth, and tenderness of the pharyngeal region to pressure.

These symptoms are succeeded by others, the nature of which depends on the form that the disease assumes.

Bollinger recognised the following forms: (1) *The Œdematous*; (2) *the Pectoral*; (3) *the Intestinal.*

1. Œdematous Form.—In this form considerable œdema of the head, intermaxillary space, neck, tongue, dewlap, and in some instances of a fore-limb, is observed. The skin of the affected regions is swollen, and

the subcutaneous connective tissue may be infiltrated with a sero-sanguineous or a sero-gelatinous material. The submaxillary and superior cervical lymphatic glands show enlargement and sero-sanguineous infiltration. The oral mucosa and submucosa are swollen and infiltrated, and stomatitis with salivation may be observed. The swellings on the face, intermaxillary space, neck, and dewlap, are very extensive, and are hot, tense, and painful to pressure. The tongue is greatly swollen, and of a purple or brown-red colour. It may protrude from the mouth, and the mucous membrane at the sides of the organ may form loose projections of a gelatinous nature. The visible mucous membranes are cyanotic, and often covered by ecchymoses. The fever runs very high, and in some cases is said to reach 110° F.

The course of the disease may be very rapid in some instances, the animal dying from asphyxia in twelve to forty-eight hours after the appearance of the first symptoms. Some authors state that death may occur within six hours. The asphyxia is due to the marked swelling of the tongue and pharyngeal region. In some instances evidences of intestinal lesions may be observed, such as colic and severe diarrhœa.

The œdematous form is most commonly met with in cattle (Friedberger and Fröhner).

McFadyean* drew attention to an outbreak which occurred in calves from two to four months old. The symptoms resembled in many respects those occurring in the œdematous form of septicæmia hæmorrhagica. Extensive swelling of the neck, head, and pharyngeal region was present. The swelling was neither painful nor emphysematous, and it rapidly increased till death ensued. In some cases it was so extensive that the eyes were invisible ; in others the swelling was confined to the pharyngeal region, and caused marked dyspnœa. The swelling was due to a watery exudate, and some blood extravasation was also present. The course of the disease was from four to eight hours after the first appearance of symptoms. The blood contained immense numbers of small bacteria morphologically similar to the organism of fowl cholera.

2. Pectoral Form.—This is characterised by marked dyspnœa and evidences of acute pneumonia or of pulmonary œdema. According to most authors, it is not of common occurrence in cattle, but is met with chiefly in wild deer. Kruger, however, has observed a large number of cases in cattle. The course is longer than that of the œdematous form. It averages from four to six days, and may extend to eight days. Young animals may succumb in a few hours.

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* *Journal of Comparative Pathology and Therapeutics*, vol. ix., p. 151.

3. INTESTINAL FORM.—The symptoms are those of enteritis. Colicky pains, diarrhœa, tenesmus, dysentery, and tympanites, are observed. This form is often associated with the œdematous type.

SUBACUTE AND CHRONIC FORM.—In some instances the disease develops in this form, with symptoms of pneumonia, broncho-pneumonia, or chronic bronchitis.

Morbid Anatomy.—The lesions vary according to the form of the disease that is present. In the œdematous form sero-sanguineous infiltrations of the subcutaneous connective tissue and marked thickening of the skin in the regions already mentioned are observed. The tongue is enormously swollen and infiltrated with a dark red effusion, and on section of the organ a large amount of yellow fluid exudes. Von Ratz has observed, in addition to the above lesions, inflammation and the presence of hæmorrhages in the mucous membrane of the abomasum.

The respiratory mucous membrane may be swollen and infiltrated. Ecchymoses may be present on the serous membranes, the endocardium, and the intestinal mucosa. Some authors state that the spleen is normal, others have found this organ enlarged. According to Friedberger and Fröhner, hæmorrhagic enteritis is generally present.

In the pectoral form a hæmorrhagic lobular pneumonia may be observed, or a gelatinous infiltration of the interstitial pulmonary tissue and well-marked pulmonary œdema. Or pleuro-pneumonia may be present, with extensive pleural effusion. Pericarditis and hæmorrhagic enteritis, are frequent complications, with ecchymoses in various region of the body.

In the intestinal form the chief lesions are hæmorrhagic enteritis, the gastric mucosa is sometimes involved, and ecchymoses are observed in the peritoneum.

In some acute cases the lesions found are not well marked (Friedberger and Fröhner).

According to Cadéac, grave alterations occur in the circulatory system; the heart is normal, but its cavities are filled with dark-coloured, tarry blood, and endocarditis with pericarditis are present.

In the chronic form the lesions occur in the lymphatic glands and in the lungs. The lymphatic glands in connection with the digestive and respiratory systems are enlarged, but those of every region may be involved, the lesions resembling those of lymphadenitis.

The pulmonary lesions, according to Cadéac, comprise centres of infiltration, congestion, induration, and caseation. In some lobules sclerosis is observed, in others the pulmonary tissue is replaced by a caseous substance encysted in a thickened and dense perilobular tissue.

According to Galtier, the caseated centres are of various sizes, and either circumscribed or communicating with a bronchus. In the bronchial tubes a large amount of a yellowish-white, viscid, muco-pus is found, containing the ovoid bacteria in abundance. The bronchial mucosa is swollen and thickened, and interlobular emphysema is frequently observed.*

Prognosis.—As from 50 to 90 per cent. of the affected animals succumb to the disease, the prognosis is always unfavourable.

On the other hand, Kruger observed that recovery took place in a large number of cases in which the course was prolonged, 61 cases recovering out of a total of 118 (Friedberger and Fröhner).

Differential Diagnosis.—Septicæmia hæmorrhagica requires to be differentiated from the following affections:

Anthrax.—From this disease it is distinguished by a bacteriological examination; also the blood does not show the alterations occurring in anthrax, nor is there marked enlargement of the spleen.

Here we may draw attention to an affection of cattle met with in the British Isles which simulates closely the œdematous form of septicæmia hæmorrhagica. It has been termed "gloss-anthrax," although the presence of anthrax bacilli has not been demonstrated in the lesions. No doubt the term "gloss-anthrax" is often loosely applied to cases characterised by lesions of the tongue and pharynx. The term "blain" is also applied to similar conditions.

Further investigation is desirable in order to ascertain the pathology and bacteriology of those obscure affections of the tongue and pharyngeal region associated with œdema of the head, face, and neck (see p. 413, and also Vol. II., Diseases of the Tongue and Pharynx).

Black-Quarter.—The differential diagnosis is based on the character of the swellings, on a bacteriological examination, and on the results of inoculation experiments on various animals.

In septicæmia hæmorrhagica the swellings are hot and have a doughy feel, and are not crepitating and cold, as in black-quarter.

Pleuro-Pneumonia Contagiosa.—The pectoral form of septicæmia hæmorrhagica may be confounded with contagious bovine pleuro-pneumonia. In the former the disease is more quickly developed, the course is far quicker, and the fatal termination more rapid; also in the latter affection cutaneous phenomena are absent. A bacteriological examination must also be carried out.

According to Friedberger and Fröhner, the anatomical conditions of the lung serve to distinguish the two affections. In septicæmia hæmorrhagica the pulmonary lesions are of the same age and of a uniform character, while in pleuro-pneumonia the lung changes are irregular.

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* Cadéac, "Pathologie Interne."

Rinderpest.—The intestinal form of septicæmia hæmorrhagica may require to be differentiated from rinderpest. In the former the intestinal phenomena are less marked, while in the latter there is an absence of the œdematous swellings, and the bipolar bacillus is not demonstrated by bacteriological examination.

Malignant Œdema.—The œdematous form of septicæmia hæmorrhagica might be confounded with malignant œdema. In the latter affection the swelling generally crepitates on palpation, and can only be transmitted by subcutaneous inoculation; in the former crepitation is absent, and the disease can be conveyed by cutaneous inoculation and by infected fodder.

Treatment.—All attempts at medicinal treatment up to the present time have failed. Prophylactic measures are of importance. Isolation of affected animals should be carried out. Some authorities advise that destruction should be carried out, so as to do away with centres of infection. Animals in contact with those affected should be isolated and kept under supervision.

Thorough disinfection of premises is necessary, and cattle should be prohibited from grazing on infected pastures. Litter and fodder contaminated by infected animals should be properly disposed of. The virus being very resistant to atmospheric influences, thorough disinfection of contaminated objects is necessary. The blood, when exposed to the air, is said to preserve its virulence for three months; hence precautions should be taken to prevent infection from this source.

As already remarked, a vaccine has been prepared by Oreste and Armannii by attenuating the virus of " barbone " (septicæmia hæmorrhagica of the buffalo) by culture in the presence of air at 30° to 32° C. This confers immunity on the buffalo and sheep. The passage of the virus through the pigeon also produces attenuation, and vaccination of buffaloes has been carried out by inoculating them three times at intervals of a few days with the blood of infected pigeons. It is suggested that similar treatment might prove of value in the case of other animals, but its application could not be carried out with facility (Cadéac).

CORNSTALK DISEASE.

As already remarked, the affection known by this term was proved by Billings to be due to a micro-organism of the fowl cholera type, and the corn-leaves were believed to carry the infection to healthy cattle.

The disease is said to have first received attention from Gamgee, who erroneously attributed it to ill-effects arising from ingestion of smut in corn, and later on to "frost-bitten grass" and to "impaction of the abomasum."

The outbreaks of the disease investigated by Billings occurred in Nebraska, U.S.A., and he concluded "that it was not a contagious affection, but its primary cause was to be sought for in the 'corn fodder' and perhaps the grasses which certain susceptible animals eat."

The lesions found were chiefly in the lungs, pleuræ, liver, kidneys, and small intestine. The lungs showed lobular pneumonia, and on section marked œdema was present, with swelling and engorgement of the interlobular tissue. The lobules were solidified and were not uniform in colour, and the bronchial tubes were filled with a straw-coloured, coagulated material.

The bronchi showed a swollen condition of the mucosa and infiltration of the submucous connective tissue. On microscopical examination of a section of lung numerous centres of consolidation were found, of irregular shape and extent, with more or less sharply defined outlines; the alveoli were infiltrated with cellular products, and those most intensely infiltrated were generally most distantly situated from the affected bronchioles. In some alveoli a yellowish-grey material was found, in which numerous nuclei were seen, and in many cases it was of an almost homogeneous character, and showed a few delicate thread-like lines running through it here and there. In other cases the material was composed of small round cells (red blood-corpuscles), and the interstitial spaces showed round-celled infiltration.

The pleuræ were inflamed, and an extensive pleural effusion was present. The liver showed lesions similar to those of acute parenchymatous hepatitis. The kidneys were affected by acute parenchymatous nephritis. The small intestine showed considerable inflammation.

The micro-organisms—*i.e.*, the bipolar bacteria—were found scattered profusely through sections of the liver, kidneys, lymph glands, and to a less degree in the lungs.

The symptoms observed were fever, the temperature varying from 102·2° to 107·6° F., a weak pulse, accelerated respirations, and evidences of pneumonia. Cerebral symptoms were often present; the animals showed evidences of excitement, by bellowing, and chasing other animals. These phenomena were ascribed by Billings to engorgement of the cerebral vessels and pressure on the brain. In other instances depression and disinclination for movement were observed. Constipation was present in some cases, and diarrhœa in others.

In July, 1891, Nocard brought before the meeting of the Central Veterinary Society of Paris the subject of "An Infectious Broncho-Pneumonia of American Cattle." The disease occurred on different occasions amongst American cattle sent for sale to La Villette, the Parisian abattoir. Only a small number were affected, and the presence of contagious bovine pleuro-pneumonia was suspected. The cases were investigated by Nocard, who arrived at the conclusion that the lesions were not those of contagious bovine pleuro-pneumonia, but of a malady special to American cattle, probably the disease known in the Western States as "cornstalk disease."

In the muco-pus from the bronchioles in the hepatised tissue and in the limpid serosity of the perilobular lymphatic sacs micro-organisms of the ovoid bacteria type were found in abundance. These were cultivated, and inoculation experiments were carried out, the result being that lesions similar to those observed in the affected cattle were produced in the experimental animals.

Nocard arrived at the conclusion that the disease had only very feeble contagious properties.

The chief interest that attaches to "cornstalk disease" in this country is with reference to the vexed question of the existence of contagious bovine pleuro-pneumonia in American cattle imported into Great Britain from 1879 to 1891.

Much difference of opinion amongst scientific authorities occurred during this period, more especially with reference to an imported ox at Deptford in April, 1891.

It is not our intention to discuss the evidence adduced by the authorities on each side; suffice it to say that some claimed that the case in dispute was one of contagious bovine pleuro-pneumonia, while others as confidently declared that the lesions were those of cornstalk disease, and similar to those occurring in the cases observed by Nocard at La Villette.

For full information on the subject the reader is referred to the *Veterinary Journal*, 1891 and 1892; to Williams' "Veterinary Medicine;" also to Nocard et Leclainche, "Maladies Microbiennes," tome i.

SEPTICÆMIA HÆMORRHAGICA IN SHEEP.

Several varieties of this affection are described as occurring in sheep. and some authors include braxy under this heading. The latter disease is considered under a special section. We describe here the affection known as "pneumo-enteritis of sheep."

Some authors recognise two forms of pneumo-enteritis—viz., one termed "lombriz," first investigated by Lignières in the Argentine

Republic, and afterwards in various parts of France, the micro-organism being a minute ovoid bacillus belonging to the pasteurella group; the other, first investigated by Galtier in 1889 in Algiers and various districts in France, and afterwards by Liénaux, Conte, Besnoit, and Cuillé. It was believed to depend on an ovoid bacterium, slightly larger than that of fowl cholera, and possessing certain cultural differences to the micro-organism described by Lignières as occurring in "lombriz," a further distinction being that the disease was closely allied to swine plague, and that transmission of pneumo-enteritis could occur from the pig to the sheep.

The duality of these forms has not been definitely determined.

In the epizoötics of the disease investigated by Galtier and others, the principal vehicles of contagion were found to be fodder and litter contaminated by the dejecta of infected animals, and the virulence of the micro-organisms could remain unaltered for a certain period in manure, water, and damp soil. The acute form of the disease is more readily transmitted than the chronic type.

The lesions in the acute type resemble those met with in cattle. The gastro-intestinal mucosa shows numerous petechiæ, the peritoneum shows a sanguineous effusion and fibrinous exudates. Congestion of the spleen, liver, kidneys, and lymphatic glands is also observed, with petechiæ in these organs. In the lungs lesions of broncho-pneumonia occur, and the pleural cavity contains a more or less abundant reddish or clear effusion, with a yellowish fibrinous exudate on the pleuræ. The myocardium may show fatty degeneration and petechiæ.

In the chronic form evidences of chronic broncho-pneumonia are observed, and the pleural exudate may become purulent.

The symptoms in the acute form are high fever and marked constitutional disturbance, followed by diarrhœa and evidences of enteritis. Symptoms of broncho-pneumonia are also present, and a sanguineous nasal discharge is observed.

The course of the disease may be rapid, death occurring in from six to twelve hours, or the animals may not succumb for two or three days.

In the chronic form, pulmonary symptoms are chiefly in evidence, a hoarse infrequent cough being the primary manifestation, followed by accelerated respirations, with a double expiratory movement and a slight muco-purulent nasal discharge. The disease may continue for six to twelve months.

Prophylactic measures are to be conducted on similar lines to those advised for pneumo-enteritis in cattle.

Curative treatment of any kind has not been found successful.

SOME DISEASES OF BIRDS, ETC.

By HENRY GRAY, M.R.C.V.S.

I. AVIAN CHOLERA.

Synonyms.—Fowl or bird cholera; Avian septicæmia; Avian pasteurellosis; Hæmorrhagic septicæmia of the fowl.

French: Choléra des poules; Pasteurellose aviare; Choléra des oiseaux de basse-cour; Choléra des volailles; Maladie épizootique des animaux de basse-cour; Maladie épizootique des poules; Typhoïde des volailles.

German: Hühnercholera; Geflügelcholera; Cholera gallinarum.

Italian: Colera dei polli.

Definition.—This is a malignant contagious disease of all species of birds, more notably the fowl, guinea-fowl, pea-fowl, turkey, pigeon, pheasant, partridge, grouse, goose, duck, ostrich, cage and aviary birds. It is conveyed by natural contagion and by inoculation. It is generally characterised by high fever, great thirst, and profuse diarrhœa, and by its great mortality, which is often rapid.

History and Geographical Distribution.—In 1600 Aldrovandus, in his "Ornithologia," speaks of a deadly epizoötic in fowls occurring in Italy. Moscati observed a similar disease in Brescia in 1770. This disease was, however, first studied by Chabert in France in 1780 to 1782. It was very prevalent in Lombardy and Piedmont from 1789 to 1798, where it was investigated by Brugnone and Toggia. It was also encountered by Baronio in 1789 in Lombardy; he considered it was of a verminous nature, as he found a quantity of worms in the cadavers. During this period it was widespread in France. In 1817 it raged in the East Indies. It was again very prevalent in France in 1825, and continued so up to 1830, when it prevailed in the neighbourhood of Paris. Huzard and Breschet gave a good description of it in 1832. From 1835 to 1836 it again occurred in France, where it was studied by Maillet, who gave the disease the name of "fowl cholera," by which it has since been known. In 1849 it reappeared in the suburbs of Paris, and was at its height in 1851. About this period it was studied by Renault, Reynal, and Delafond, of the Alfort Veterinary School, who concluded that contagion played the preponderating rôle in its causation, and that it could be transmitted to various species by inoculation. Reynal could not convey it by feeding fowls, geese, and sparrows on the muscles, heart, spleen, liver, intestine, and blood of diseased birds. A man in the Alfort School took home all the carcasses of the birds dead of the natural and experimental disease, and he and his five children consumed them without any injurious effects. Delafond demonstrated its transmission from the fowl to the rabbit, and *vice versâ*. During the same year (1851) Benjamin gave a good description of it, demonstrated experimentally contagion by cohabitation, and formulated rules for its prevention; he also observed that man and dogs could consume the flesh with impunity. In 1850 it occurred in Alsace-Lorraine. From 1860 to the present time it has caused great losses in France. About 1830-1832 the disease spread to Bohemia, Austria, and Russia. In 1864 it was very prevalent in Italy; in 1865 to 1866 in Southern Hungary; in North Italy in 1871 and 1872; and in Haute-Alsace in 1875. In 1869 Moritz investigated the disease, and pointed out the presence of a microphyte in the form of special granulations in the blood of the diseased birds. From 1877 to 1880 it caused a great mortality in the whole of Europe. In 1877 and 1878 both Perroncito in Italy and Semmer in Russia studied this affection, and pointed out about the same time the presence of a microbe having a rounded form, and appearing singly, or two combined. Shortly after (1879) Toussaint, of the Toulouse Veterinary School, confirmed these observations, and demonstrated that this infinitely small micro-organism was the sole cause of this malady, and obtained cultures of it in neutralised urine. In 1880 Pasteur studied the disease, and isolated, by pure cultures in chicken broth, the same micro-organism, which he inoculated into the cellular tissues of the pectoral muscle of the fowl, and produced a tumour, and by this method he was able to induce immunity in the fowl against virulent cultures, and thus laid the foundation of the principle of obtaining by artificial cultures of pathogenic organisms, vaccines against virulent diseases of man and animals. In the case of fowl cholera, Pasteur showed that "the oxygen of the air weakened and attenuated the virulence of the microbe," and that it was possible to obtain, in grading its action, a vaccinal virus, which did not kill, but gave a mild disease, and preserved the animal against a fatal disease. The disease appeared in the United States of America during the period of 1880 to 1882. It was introduced into Germany from 1897 to 1899 by geese and fowls imported from Russia, Poland, Silesia, and Italy. It was observed about this time in Austria and in Hungary. In 1899 it caused considerable loss in Italy. It prevailed in Chili in 1897, in the United States of America in 1898, in Canada and in France in 1899. It was also very prevalent in Great Britain in 1900, when McFadyean investigated several genuine outbreaks in various districts wide apart from one another. Prior to McFadyean's researches, Klein had made the assertion that the disease did not occur in these isles, such statement creating surprise in the minds of some Continental bacteriological experts. This is erroneous, as shown by McFadyean. The writer has observed the disease for nearly forty years in country districts, where formerly it did not receive any attention from veterinary surgeons, or create in the mind of poultry owners much surprise, unless they happened to be possessed of very valuable stock birds from a commercial and exhibition point of view, when they usually consulted the post-mortem man or adviser in poultry papers, or an ignorant quack-medicine vendor. Nearly all, if not all, poultry journals employ men who have never received any instruction in pathology to give advice to querists on the ailments of birds. Hence the ignorance generally displayed as to the existence of this disease in the United Kingdom. The disease has obtained a foothold in South Africa, and probably also in Australia and New Zealand.

General Remarks.—A great deal of attention was given to this disease on the Continent during the course of the nineteenth century, and in consequence important progress was made in the knowledge of the affection, which has had a very beneficial effect on human and veterinary medicine by placing protective inoculation (hitherto carried out in an empirical manner), and the nature of hereditary and acquired immunity on a scientific basis. In reality, when Pasteur demonstrated by the study of fowl cholera that virus could be so attenuated by artificial culture as to act as a vaccine against a virulent or fatal form of the natural disease, it was the crowning point of his work on the nature of contagium or the virus of microbial diseases, all further developments since that epoch being merely an extension of details of the great principles established by this immortal genius.

The organism of fowl cholera was included by Hüppe in 1886 in his class of bipolar or ovoid organisms, causing a group of diseases termed by him *hæmorrhagic septicæmias*. In 1900 Lignières adopted it as the standard of his class of organisms included in the genus of pasteurella created by Trevisan in honour of Pasteur, who was the first to thoroughly study the organism now taken as the standard of a great class of diseases. The diseases caused by the fowl-cholera type of organism are termed by

Lignières and his followers "pasteurelloses," which originally included all the following:

1. Avian pasteurellosis (fowl cholera, rabbit septicæmia, and, according to Nocard, Guèrin's fowl diphtheria, etc.).
2. Porcine pasteurellosis (swine plague or swine pneumonia).
3. Ovine pasteurellosis (pneumo-enteritis, hæmorrhagic septicæmia of sheep, lombriz, etc.).
4. Bovine pasteurellosis (wild animal and cattle disease, barbone of buffaloes, hæmorrhagic septicæmia of cattle, the broncho-pneumonia of American cattle, or corn-stalk disease, entéqué, white scour, etc.).
5. Equine pasteurellosis (influenzal or catarrhal affections, with all their forms and complications).
6. Canine and feline pasteurellosis (distemper and typhus).
7. Guinea-pig pasteurellosis (Phisalix's guinea-pig septicæmia).

According to Lignières, the specific characters of the pasteurella are invariably the following:

Cocco-bacilli, without movements of translation, not taking Gram, very polymorphic, manifesting involution forms, not liquefying gelatine, not coagulating milk having a normal reaction, not giving any visible culture on natural acid potato, nor indol in pancreatic bouillon, not reddening the agar of Würtz, chiefly aërobic, but also anaërobic, presenting an odour *sui generis* in the cultures. Form no spores, have no cilia. Virulence very variable; in general, great. By intravenous injection have a special affinity for the tendinous and articular synoviæ. These characters are fixed, absolute; the absence of one or other excludes the microbe from the group of the pasteurella. These microbes are easily coloured at both extremities, leaving a clear central space; they are not so readily stained as some other microbes—for example, the coli. They sometimes grow discretely on the usual culture media, and they are very difficult to detect in the centre of the system. According to Phisalix, there is another character common to all the pasteurella, and that is they render turbid the alkaline peptonised bouillon culture. Nocard and Leclainche consider the majority of pasteurellic septicæmias are caused by varieties of the same microbial type—namely, the ovoid or belted organism of Hüppe. Phisalix was also of this opinion. Nocard, and Chamberlain and Jouan adopt the view of Hüppe, that there is only one ovoid bacterium, which may readily pass from one species to the other, and which by its adaptation on a given species creates a pasteurellosis special to that species. Chamberlain and Jouan, like others, have shown that spontaneous transmission of fowl pasteurellosis to the pig is realisable. Stang and Pfersdorff encountered a simultaneous outbreak of what they considered to be fowl cholera in the poultry and swine in the same establishment. The isolated bacillus killed the pigeon in twelve hours, and only caused local lesions in the guinea-pig. The infected swine showed hæmorrhagic gastritis and swollen spleen, but no lung lesions. Lignières has demonstrated, by passing a pasteurella through the animal body several times, that it may become virulent for all susceptible species, and that immunity conferred by one variety of pasteurella may give immunity to any other variety. Chamberlain and Jouan and others confirm this.

Lignières' conclusions on the general characters of the pasteurella have been contested by many authorities; for instance, it has been shown that old cultures of the bacillus of fowl cholera contain indol, but only towards the end of a fortnight of their development. Again, the view that they play a prime part in the causation of many clinical affections of the domestic animals has not been accepted, but has been discarded by the majority of bacteriologists as untenable. The majority of the diseases (equine influenza, swine plague, swine fever, canine and feline distemper, etc.) attributed to the pasteurella and the salmonella by Lignières and his followers have now been proved to be due to ultra-microscopic organisms, the pasteurella and the paratyphoid microbes only playing a secondary rôle in their etiology. The pasteurella are often found in normal animals and in their surroundings as saprophytes.

Bacteriology.—The bacillus of fowl cholera, also called the *Bacillus avisepticus*, *Bacterium avicidum*, *B. choleræ gallinaceæ*, ovoid or bipolar organism, or figure-of-eight bacillus, when isolated from the blood in the vessels, has the aspect of a small ovular bacterium, which, when stained with methylene blue or gentian violet, after fixation with alcohol-ether, is coloured at both extremities. When derived from a culture in the guinea-pig's peritoneum, it appears as a cocco-bacillus; and in the case of a very great virulence it resembles a diplococcus. If obtained from the rabbit's intestine after infection by ingestion, it frequently has the form of a small bacillus. In none of the last three forms does it stain so well as in the case of the first. The method of staining may completely change its aspect. After fixation with alcohol-ether, by the use of the carbolic solution of Ziehl, it readily shows the clear central space. If it is previously stained with fuchsin, and warmed for half a minute before adding the Ziehl solution, it is shown in the form of small bacilli or cocco-bacilli. In none of its forms is it stained by Gram's method. It is, however, readily stained by the majority of the ordinary staining solutions. This very small cocco-bacillus does not exceed 0·5 to 1·25 μ in length by 0·25 to 0·40 μ in breadth.

It may be easily grown on the majority of culture media, and manifests most of the characters given under the general characters of the other pasteurella. According to Porcher and Panisset, cultures in peptonised bouillon contain *indol*, but only towards the end of a fortnight's growth. The organism acts on the system by the secreted toxins. Inoculation of the filtered cultures produces symptoms of intoxication similar to the natural infection. The toxin is more likely to be found in cultures of twenty hours' duration, after sterilising by chloroform or tricresol.

Inoculation of virulent cultures destroys every kind of bird. The rabbit seems more receptive to experimental infection by ingestion or by inoculation than the bird. Death rapidly takes place without any diarrhœa, although on post-mortem examination the intestine is found to contain liquid material. (In this animal this disease is known by rabbit-owners as "gotch.") The mouse and rat are susceptible. Field-mice may be exterminated by the use of cultures of this bacillus, as was demonstrated by Metchnikoff in Southern Russia. Adult guinea-pigs are resistant, as are the cat and dog. With virus exalted by a number of passages a degree of virulence may be obtained which will kill the guinea-pig, white rat, dog, cat, pig, sheep, ox, ass, horse, etc.

Virulent Materials.—The blood, viscera, mucous and serous exudates, fæces, contents of the crop, nasal and buccal mucous discharges, are virulent in acute cases. The eggs sometimes contain the specific bacilli, as has been demonstrated by Reynal, Barthélemy, and Kitt.

Period of Incubation.—Experimental infection by inoculation with a few drops of a virulent culture will kill a fowl within twelve to thirty hours. Wertheim inoculating pigeons with old cultures provoked a chronic form with symptoms of pyæmia, death only resulting ten or more days later. In forty fowls inoculated by Salmon, the period of incubation varied from four to twenty days, or on an average eight days. In Semmer's feeding experiments death was delayed for three weeks. Some Continental authorities have placed it as low as eight to sixteen hours. In the case of natural infection, however, it usually ranges from four to twenty-one days. It may yet be indefinite.

Method of Infection.—The natural method of infection is by ingestion of the virus contaminating the food and water. It may also be contracted by eating the blood, discharges, flesh, and eggs of diseased birds. The usual mode of the food and water being soiled is by the fæces of diseased birds; it may also occur from the bird pecking its own feet, soiled with fæces containing the virulent material. The disease is not always contracted by the healthy bird eating the remains of dead birds, as Reynal, Lignières, and others, have frequently seen fowls resisting the infection by this mode. Sticker considers maggots developing in dead birds are quite capable of containing the virulent bacilli for several days, and that birds ingesting them may contract the disease.

Sparrows, starlings, crows, hawks, pigeons, dogs, cats, rabbits, and mice, as well as insects, may spread the disease from an infected place to a distant locality hitherto free from the infection. The usual source of the contagion is by the purchase of birds at an infected place, or sending poultry to markets, bird-shops, and shows, where they get the infection, and then, when they are not sold, they are brought home to spread the disease to the regular inmates of the poultry-yard or aviary. Contaminated hampers, crates, corn from poultry-dealers, and the poultry-dealer's cart, are liable to spread the infection. Bosso relates the history of an enzoötic of the disease introduced by infected instruments used by a woman in caponizing a series of fowls. Man may carry the infected fæcal matter on his boots.

McFadyean investigated two outbreaks during 1900; they occurred almost simultaneously on two farms several miles apart. As to the origin of the outbreaks, the only discoverable circumstance was that at both farms the poultry were being fed with corn-sweepings from foreign ships that discharged at a neighbouring port.* According to Law, the sale and transportation of the guano from the infected farm is a direct cause of new outbreaks. On the other hand, as the virus may live in the soil of the farmyard or poultry-house for some time, the disease may reappear, even after clearing out the old contaminated stock, when fresh birds are introduced or chickens are hatched. The purchase of old poultry houses, coops, rearers, etc., may bring the disease into clean premises. It is, however, uncertain what part the non-pathogenic saprophytes, having all the characters of the avian cholera bacillus, and existing in healthy pigeons, poultry, and other birds, play in certain outbreaks.

It is possible that internal parasites, so common in domestic poultry, may play a considerable rôle in the genesis of the disease, and it is also possible that the epizoa may often convey it by inoculation.

Susceptibility.—In-bred and highly-developed breeds of poultry often kept in confinement in runs are more susceptible than the common barnyard fowl; pigeons are more susceptible than geese and ducks. Cage-birds and rabbits are attacked as severely as the highly-bred fowl.

Symptoms.—As the disease varies in the length of its course and in degree of virulence, it is usually described under three forms—a *peracute* or *sudden* form, an *acute* form, and a *chronic* form. The writer, however, will include another form—the subacute form.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xiv., 1901, p. 99.

Peracute, Sudden, Fulminant, or Apoplectic Form.—In this form the fowl is suddenly seized with a somewhat apoplectiform seizure during progression, or even when sitting on the perch or on the nest, and succumbs in a few hours, after having shown all the signs of a rapid intoxication. In many cases it is found dead, although a short time before it was observed to be apparently well. In those cases seen from the onset one finds the bird sleepy, indifferent to its surroundings; it does not move, but remains by itself in a cold, shady corner or under some obstacle. The wings droop and the head is brought under the shoulder, giving the bird the appearance of being in a ball; the feathers are opened or ruffled; the eyes are half or quite closed; the comb, wattles, and mucous membranes are purple; the temperature* may rise to 110° to 112° F.; and the bird falls and succumbs during a convulsive attack within two to five hours after showing the first signs of illness. In some cases the disease runs a still more rapid course; the bird suddenly ceases to feed, appears anxious for a few minutes, and then falls dead.

Acute Form.—In this form the appearance of grave symptoms is not so sudden as that of the peracute form. The birds lose their activity and brightness, and separate from their fellows; the appetite fails, although drink may be sought for; the wings and tail droop, the feathers are open, the gait is tottering or staggering; there is great weakness and drowsiness, and at intervals the bird attempts to rouse and shake itself, and to walk, when it falls down on its breast and lapses into a comatose condition. The comb and wattles become flaccid, and if the comb be an erect one it falls on to one side, and takes on a brownish-red, purplish, or black colour. The abdominal muscles and skin quiver or twitch. A profuse diarrhœa sets in; it is at first greyish and fæcal, then afterwards becomes bloody, frothy, and mixed with whitish masses resembling the boiled white of egg. There is a thick stringy or frothy mucus issuing from the nostrils and mouth, sometimes vomiting; the temperature attains 108° to 112° F.; the comb and the mucous membranes have a purplish colour; the skin is streaked with bluish patches; the breathing is difficult and noisy, and when the bird is roused it totters and falls. Sometimes it hiccoughs. The feathers round the anus become soaked and matted with discharge. In the last stage the extremities are cold, the temperature is subnormal, the comb black, the beak opens and shuts spasmodically, there are convulsive movements of the body, and the bird gives a cry, and dies after the disease has lasted for a period varying from ten to sixty hours.

Subacute Form.—The affected birds do not feed well, or have no appetite at all, but sometimes they feed shortly before death. Thirst is very great. Diarrhœa appears. At first the fæces are passed in large quantities, and consist principally of white urates, mixed with colourless mucus. Afterwards the urates are suspended in a thin, transparent, sometimes frothy mucus; the urates have a deep yellow colour, but in the later phases of the malady this may change to a greenish or even a deep green. This changing of the urates from normal whiteness in health to a yellowish colour is very noticeable in the earlier stage of the disease, and is important, as it enables one to make an effort to isolate the birds as soon as possible after the disease appears. It may, however, be observed in other diseases than cholera.

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* Average normal temperature of fowl, 106·5° F., and of smaller birds a few degrees higher.

The sick birds separate from their fellows, and can no longer maintain the alert, upright, and bright appearance; the feathers stand open, the wings and tail droop, the head is drawn towards the shoulder, and the birds have a ball-like appearance. There is great weakness, the birds become drowsy, and seem indifferent to their surroundings. This drowsiness becomes so profound as time goes on that it is difficult to rouse the birds from their stupor. If they are made to move, the fæcal material is profusely discharged, and after a time this is passed in smaller but repeated quantities.

The comb loses its bright red appearance, and becomes pale and even white. The birds rapidly lose weight, and are so light and weak that the slightest touch causes them to stagger or fall. This form may run through a flock in a week or two, destroying the greater part of the birds. It usually runs its course in from three to ten days.

Chronic Form.—This form is generally encountered in birds that have resisted the acuter forms, and usually appears towards the end of an outbreak, or when the infective material is not of such a virulent character. The birds show dulness, do not appear strong, seem tired or mopy; their appetite is capricious; diarrhœa persists or is intermittent; they are inclined to place their head under their wings in the form of a ball, and have the feathers open. Emaciation becomes very great; the mucous membranes appear pallid.

In some cases one or more joints, especially the femoro-tibial or tarsal in one or both limbs become swollen, hot, and painful, and ultimately fixed, and even ankylosed. Such cases may last for weeks and then die from exhaustion, and the lesions are often confounded with those described as occurring in tuberculosis, gout, and rheumatism. According to Lignières, the pasteurella is not recoverable from these lesions, which he has reproduced experimentally by inoculating, especially in the veins, more or less attenuated pasteurella. Birds may become absolute skeletons without having manifested any diarrhœa.

This form extends slowly, and continues in certain places for several weeks or months.

Prognosis.—The prognosis of avian cholera is always very serious, as quite 90 to 95 per cent. of the very acute cases succumb. Even in the milder form the mortality is quite half the number attacked, and of those that do not die, many are left sickly, are bad doers, and do not pay for their keep. The danger of this disease on a farm is that it is likely to be carried by birds, rabbits, and other animals to neighbouring places, or perhaps the recovered animals are sent by the farmer to the market, and thus the disease is spread all over a district.

Post-mortem Appearances.—The skin is spotted with black patches, due to hæmorrhages in the deeper layers of the skin and in the subcutaneous connective tissue. The mouth and nostrils contain a quantity of ropy or frothy mucus. The crop is filled with food, which has a very sour or nauseous odour, in consequence of the arrest of digestion.

The intestinal walls, especially the mucous membrane, more noticeably in the duodenum, are covered with an inflammatory exudate, which is often bloody; the intestinal contents are watery, frothy, brownish or purplish, and swarming with the specific bacilli. The mucous membrane is brownish or blackish, and is dotted with ecchymotic patches.

The mesenteric vessels are filled with black blood, and in consequence they stand prominently out on the greyish membrane. The liver is swollen, extremely friable, and congested; in some birds, even when the disease has run a rapid course, it contains *numerous yellowish caseous points of degeneration, which are either disseminated or run into one another.*[*] This may occasionally be observed in the intestine and lungs, in which the ovoid bacillus may be found. (Compare this with Bird Fever, p. 439.)

The spleen is swollen and soft in some cases, and normal in others. The kidneys are dark and friable, and often contain accumulation of yellow urates. The ureters are distended with yellow urates. The peritoneum sometimes contains a few fibrinous clots. The lungs may be slightly congested, or sometimes contain patches of catarrhal or hæmorrhagic pneumonia, associated or not with a sero-fibrinous exudation into the air-sacs. The pneumonic changes are more often encountered in land birds than in aquatic birds, which are, on the other hand, more subject to intestinal and cardiac lesions.

The pericardium may contain a small amount of a thin, clear, pale yellowish fluid, which forms on exposure to the air a diffluent gelatinous mass. Numerous ecchymotic spots are found in the auricles. Sub-epicardial hæmorrhages are very characteristic. The heart muscle has a washed appearance; it is soft, friable, and ecchymosed. The blood in the cavity of the heart is black, and does not clot well; it is teeming with the specific bacilli. According to Reynal, who gave this disease a great deal of attention, the blood may have a normal appearance, and clot well. The vessels of the pia mater may be injected, and the subarachnoid may show small ecchymotic spots. The muscles do not show any alteration in the acute forms, but may have an attenuated, greyish, and washed appearance in the chronic form.

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[*] Sticker, "Käsige Processe dei der Geflügelcholera," *Archiv für Thierheilkunde*, Band xiv., 1888, p. 332.

Diagnosis.—Avian cholera can only be distinguished definitely from avian plague by bacteriological examination. In *avian cholera* the specific bacillus is encountered in all the tissues. It appears in the blood after staining, free between the blood-corpuscles, in the form of micrococci, diplococci, or of an ovoid or belted organism. Inoculation of the blood into a rabbit or pigeon gives the disease. Sub-epicardial hæmorrhages, pericardial effusion, severe enteritis, and very frequently pneumonia, are often very pronounced in avian cholera. In *avian plague* no organism is usually found in the blood; inoculation of the blood into pigeon or rabbit does not reproduce the disease. Filtered blood reproduces the disease in the fowl, and when inoculated on ordinary culture media is sterile.

Avian cholera may resemble *bird fever*. The nodular lesions in spleen and liver of the latter disease contain a very small bacillus of the fowl-cholera type, but when inoculated into the pigeon it does not reproduce the disease.

It is impossible to distinguish by bacteriological examination avian cholera from *catarrhal roup, goose septicæmia, duck cholera, epizoötic pneumo-pericarditis of the turkey, epizoötic pneumo-enteritis of the turkey*, and some other infectious processes, especially some diphtheric manifestations in birds.

Avian cholera may resemble *tuberculosis* by the caseous lesions in the liver and other organs. Bacteriological examination will discriminate between the two.

It may also resemble *acute avian diphtheria*, but is distinguished from this by the absence of false membranes in the mouth. From *infectious leucocythæmia* it is distinguished by the absence of the specific bacillus in the blood and viscera, and by inoculation experiments, which require several weeks to reproduce the latter disease.

According to Kitt, who has made lengthy experimental studies of fowl cholera, inoculation of some of the fresh infective blood under the skin of the pigeon produces at the site of injection a yellowish nodular prominence, from the size of a sixpence to a shilling, and death results in from twelve to forty-eight hours. This reaction is said to be very characteristic of fowl cholera.

Treatment.—Not much can be done in the way of medicinal treatment, as the disease usually runs such a rapid course that the majority of animals are dead before the owner has had time to realise the seriousness of the malady. If its nature should be recognised in time, the apparently healthy birds should be isolated at a distance from the homestead, and the sickly should be destroyed, or, if valuable birds, treated by antiseptics, or even by the fowl-cholera serum. All dead birds should be burnt. Rats and mice should be destroyed, for if they should not suffer from the disease themselves, they are liable to carry it from place to place, or perhaps to contaminate the birds' food. It may be advisable to vaccinate, by the Pasteurian method, the apparently healthy birds, and also to inoculate them with the specific serum at the same time, so as to be sure no latent infection is present. The eggs laid by diseased birds should not be utilised for the feeding of young pheasants, partridges, and canaries.

The birds should have in their drinking-water 2 per cent. of sulphuric acid and some sulphate of iron. Where the serum treatment cannot be carried out on valuable birds, a subcutaneous injection of carbolic acid might be tried, or even a solution of permanganate of potassium given by the mouth.

The fowl-houses should be scraped and washed with limewash prepared from freshly-slaked lime, or the green creosote oil of commerce as used for pickling railway-sleepers and other timber. This latter is recommended, as its price is only a few pence per gallon, and it destroys, not only pathogenic organisms, but also ectozoa, which often live in the crevices in the building. The floor-earth should be removed, and fresh earth, or equal parts of cinder-ashes and freshly-slaked lime, put in its place. Fowls on a farm should be housed in movable houses in the centre of a field, which should be moved from one spot to another from time to time, so as to avoid unduly contaminating one part. In pheasant and partridge rearing on some estates in Great Britain the same ground for the purpose is not used two consecutive years, as experience has shown game-preservers that disease is almost certain to be rampant if the same ground is used year after year.

Freshly-purchased birds should be isolated for two weeks, so as to ascertain if they have any latent infectious disease in their system.

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II. EPIZOÖTIC PNEUMO-PERICARDITIS OF TURKEYS.

Definition.—An enzoötic contagious disease of the turkey, and occasionally of the fowl, due to an organism of the pasteurella or fowl-cholera type, and characterised by inflammatory lesions in the lung and pericardium.

History.—This disease was first observed in September and October, 1892, by Mr. G. E. King, of Abingdon, and studied in 1892 and 1893 by Sir John McFadyean. It occurred in a small flock of turkeys, of which more than twenty died before it appeared in the fowls. It was considered to have been introduced by a freshly purchased cockerel, which seemed unthrifty on arrival. A similar outbreak in turkeys was observed by Jowett in the vicinity of Cape Town in 1908. The mortality was great. Probably the disease was imported from the United States of America.

Susceptible Species.—In King's outbreak the disease appeared in turkeys long before the fowls were attacked. In McFadyean's experiments the disease was readily conveyed to the turkey by ingestion and inoculation. Rabbits were very susceptible by inoculation, but not by ingestion; the guinea-pig was easily overcome; the pigeon was more resistant; the fowl, sheep, calf, pony, and pig, were almost refractory.

Jowett found turkeys were very susceptible by ingestion and by inoculation of pure cultures; fowls were very resistant; pigeons were more susceptible; guinea-pigs readily yielded; while rats were not refractory. Geese and ducks seem immune.

Etiology.—Introduction of a sick or recently recovered bird seems to have been the mode of the spread of this disease. King considers the nasal discharge, which is, in his experience, always present, is the principal source of infection, by dropping on the ground and being picked up in the food and ingested by the birds.

Bacteriology.—The causal organism has all the morphological staining and biological characters of the fowl-cholera organism. It only differs from this latter by its physiological properties—viz., that the fowl, which is highly susceptible to fowl cholera, is relatively resistant to the ovoid organism of the turkey disease; that the pigeon is more resistant to the turkey disease than to fowl cholera; and that the turkey germ has no effect on the rabbit when taken into the alimentary canal, the opposite being the case with the fowl-cholera organism. McFadyean considers that, "judged by their effects on different species, the two organisms are closely differentiated, and deserve to rank as distinct species." But even two strains of the same species of organism vary in virulence, and therefore in effect on the same species, but perhaps different strains of animals. Reynal, in 1857, observed that in the great outbreak of fowl cholera in 1851 in France, the disease varied in its preference for certain classes of birds. On one farm it attacked the fowls, on another the geese and ducks, and afterwards it spread to every other species of birds. In some districts it raged with violence on the fowls, and respected the geese, ducks, and rabbits; and at other times, but more rarely, these last were alone affected, and the fowls remained free from its attacks.

Period of Incubation.—This is similar to that observed in fowl cholera, attacking all species of birds, simultaneously or successively, but principally fowls, ducks, turkeys, and geese, and to a lesser degree guinea-fowls, pheasants, pea-fowls, and pigeons, and sometimes rabbits.

Course.—The disease may run its course and end in death within one to four days from the time of infection.

Symptoms.—According to King, the first birds to be attacked are the best in condition. The first noticeable symptoms are drops of mucus clinging to the end of the beak, a peculiar twitching of the eyelids, stiffness of the neck and legs, drooping of the wings and tail, and ruffling of the feathers. Soon after these there is a characteristic discharge from the nostrils, a peculiar rattling or gurgling sound in the throat, while the mouth becomes filled with frothy mucus. The fæces are milky-white or yellowish, and very thin in consistence, and are frequently voided. There may also be loss of appetite, great weakness, oral breathing, sneezing, or snuffling, and frequent opening and closing of the beak. The wattles may be of a purplish colour.

Lesions.—The characteristic lesions in this disease are chiefly confined to the lungs and pericardium.

One or both lungs are more or less extensively consolidated, swollen, dark in colour, and firm on section, often showing a slightly mottled cut surface. The bronchi may contain yellow fibrinous casts.

The pericardial sac may contain a quantity of effusion with or without fibrinous lymph. At times the pericardium is inflamed, with numerous flakes of yellow lymph adherent to the surface of the heart; in other instances, in addition to the pericardium being inflamed, it is adherent to the heart wall through the medium of a layer of cream-coloured fibrinous lymph. In some instances the heart muscle may be of a palish colour without any pericarditis being present. The blood in the heart cavity may clot firmly. The blood in the hepatised lung, in the heart cavity, and other tissues, as well as the pericardial exudate, contains numerous ovoid bacilli.

The mucous membrane of the small intestine and cæca is often hyperæmic, or shows intense inflammatory congestion.

Diagnosis.—It is not likely to be mistaken for the infectious entero-hepatitis, or "blackhead," occurring in this species, and due to a protozoon, the *Amœba meleagridis* (which is probably the schizont stage of a coccidium), setting up inflammatory thickening of the walls of the cæca, and brownish, yellowish, or greenish areas of degeneration of the liver, and often manifesting during life a blackish congestive discoloration of the featherless parts of the head. These two conditions, however, may coexist in the same bird.

It is differentiated from *avian plague* by the absence in the latter disease of the ovoid or bipolar microbes in the blood, and by the resistance of the rabbit to inoculation of the virulent and sterile blood of avian plague.

From the *septicœmia of Lignières* and of Martel occurring in the fowl and turkey, in which the lesions consisted in an inflammation of the intestinal mucous membrane, tumefaction of the spleen, and sometimes pericarditis, with abundant false membranes, it is distinguished by the above disease containing in the blood, spleen, liver, and false membranes, a bacillus of the colon group, and by the absence of pneumonic lesions.

From the *epizoötic dysentery* of Lucet occurring in fowls and turkeys by the absence of pneumonia in this latter disease, which often shows pericardial and myocardial lesions, and is due to a microbe that grows on potato ; it usually takes one to two, and even three, weeks to run its course before death or recovery sets in.

Treatment.—Attempt should be made to isolate the birds away from the premises as soon as possible, and to thoroughly cleanse and disinfect the sheds, etc., occupied by the diseased birds. If possible, isolation should take place on stubble or ground likely to be soon ploughed up. Vaccination, sero-vaccination, or serotherapy, might be attempted.

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Lignières : Septicémie à Coli-bacille chez la Poule. Comp. Rend. de la Soc. de Biologie, 1894, p. 135.

Martel : Maladie à Coli-bacille de la Poule et de la Dinde. *Ibid.*, 1897, p. 500.

Jarosch : A Septicæmia in Turkeys due to a Microbe identical to that of Swine Erysipelas and of Koch's Mouse Septicæmia. Œsterreichische Monatsschrift für Tierheilkunde, May, 1905.

III. EPIZOÖTIC PNEUMO-ENTERITIS OF TURKEYS.

Synonym.—Big-head.

This disease was studied by Dodd, and occurred in various parts of Norfolk during July, August, and September, 1904. The mortality was very great, and occurred in young turkeys that were allowed their full liberty. No fresh birds had been introduced into the locality for a considerable time. The disease did not spread to the other species of farmyard poultry. It ran a course much longer than that observed in avian cholera or avian plague.

Bacteriology.—The incriminated organism was indistinguishable from that of avian cholera, either by its morphology, coloration, or artificial culture. It was found in the swellings on the head, the lungs, fibrinous lymph, and heart blood.

The cultures intramuscularly injected into the fowl did not kill it, although they made it ill for a few days. When subcutaneously introduced into the rabbit, they destroyed it in a few hours. No disease was produced in the adult rabbit by ingestion of the culture. Subcutaneous inoculation did not kill the guinea-pig. Intramuscular injection into the pigeon killed two out of the three birds experimented on. No experiments were made on turkeys.

Symptoms.—The first noticeable symptom was a swollen head, and about a fortnight later acute symptoms set in, and deaths then occurred daily. The turkeys lost their appetite, and evinced a desire to keep away from the other birds. The feathers became ruffled. Usually a swelling, which varied in size in different birds, developed under one or both eyes. The tongue was furred. Sometimes saliva issued from the mouth. There was a discharge from the nostrils and mouth. The fæces were very loose, and sometimes white in colour. Emaciation was progressive. There was " gaping " in some birds, which showed on post-mortem examination a few worms (*Syngamus trachealis*) in the trachea.

Lesions.—The body is usually emaciated. There is a fluctuating swelling in the oculo-maxillary sinus or sac under one or both eyes. This swelling, when cut into, allows a clear viscid fluid to escape from it. There is some exudate clinging to the nostrils and mouth, and occasionally a creamy diphtheritic deposit inside the mouth. One or both lungs are generally consolidated, and contain numerous light yellow foci. The large bronchi may be plugged with yellowish fibrinous exudate. The pleural air-sacs may contain a similar exudate. *There is* *no evidence of cardiac or pericardial changes.* The intestines may be markedly inflamed, and contain a colourless jelly-like material. In some instances these are only slightly congested, partially or totally; or sometimes ecchymosed. *Spleen and liver are normal.* The lesions generally contain the ovoid bacillus in abundance. In the inoculated pigeon, diphtheritic deposits in the mouth are manifested.

Diagnosis.—The "swelled head" was the usual feature in this particular outbreak. Pericarditis was absent. Enteritis was constant. Dodd's organism was non-motile. In McFadyean and King's epizoötic pneumo-pericarditis, "swelled head" was absent—at least, not mentioned—and pericarditis was generally present, while enteritis was not a marked feature. McFadyean's organism was motile.

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IV. GOOSE SEPTICÆMIA.

Synonyms.—Hæmorrhagic septicæmia of the goose; Pasteurellosis anserina.

This disease, which is only a type of avian cholera affecting the goose, occasionally causes a terrible mortality. The blood and tissues swarm with a bacillus having all the staining, morphological, and biological characters of that of fowl cholera. It only differs from the ordinary fowl cholera type of organism by its physiological effect on the fowl, which is neither infected by inoculation nor by feeding. Pigeons, rabbits, mice, and the guinea-pig, may be experimentally infected. Ducks do not take the disease by ingestion, but they may be infected by inoculation.

Symptoms.—As in fowl cholera, the geese are found dead, dying, or rapidly becoming ill, and in this last case rarely surviving thirty-six hours. The affected animals move in a slow or indifferent manner; they lag behind the rest of the flock, stagger and fall, hiding their heads in the earth or twisting them round. Death usually takes place during convulsive movements. In the acute cases no diarrhœa is observed. The disease may merge into the chronic form.

Lesions.—The skull and brain are markedly congested. The mouth and nostrils contain a thick, tenacious mucus. The mucous membrane of the alimentary canal contains much extravasated blood, which may be found in the lumen commingling with the ingesta; there may also be punctiform petechiæ or ecchymosed patches on the mucous folds. The liver may contain numerous minute hæmorrhagic and yellowish patches of coagulation necrosis, more or less in its deeply situated substance. The pericardium frequently shows petechial spotting. The other mucous surfaces are also spotted with minute hæmorrhages. The blood is usually black, tarry, and disinclined to become lighter in colour on exposure.

Diagnosis.—It is liable to be mistaken for goose plague or a form of avian plague affecting the goose (*vide* Avian Plague, also Goose Spirillosis.

Treatment and Prevention is carried out on similar lines to those mentioned under Avian Cholera. Cyllin might be given internally in this disease, as well as in all the septicæmic diseases in the avian species. It has been used with success in avian diphtheria.

V. DUCK CHOLERA.

(*a*) Cornil and Toupet investigated an enzoötic confined to the various varieties of the domestic duck, which succumbed, whereas wild and exotic species were immune. The microbe closely resembled that of fowl cholera, but its appearance in cultures on gelatine and agar, and its abundant growth on potato, exclude it from the genus Pasteurella. Experimental ingestion kills ducks within three days, with all the lesions of the natural disease. Subcutaneous inoculation kills ducks of every species, but whereas the domestic varieties are destroyed in one or two days, the others survive for a longer period, even from eleven to fifteen days. The fowl, pigeon, and guinea-pig, are refractory to subcutaneous inoculation. The rabbit is occasionally killed by massive doses.

Symptoms.—The most noticeable symptoms are diarrhœa, gradually increasing weakness, and muscular tremors. Death results in two or three days.

Lesions.—The visceral pericardium shows ecchymoses, and sometimes inflammatory lesions. The liver is congested and degenerated. The peritoneal surface of the intestine is very much congested, and often shows small ecchymotic spots. The mucous membrane of the intestine, especially the large portion, is distended by a muco-hæmorrhagic secretion.

(*b*) Willach studied a disease in waterfowl, pheasants, and geese. The blood contained an ovoid bacterium resembling that of fowl cholera. It gave a pure culture in agar. Contagion was readily transmitted to ducks, especially pure-bred ones. Fowls did not contract the disease by cohabitation; they were also refractory to the ingestion of cultures. Inoculation killed the duck, pigeon, fowl, and mouse; the guinea-pig was refractory.

Symptoms.—These were stupor, loss of appetite, intense thirst, difficulty in swallowing, and a hoarse noise.

Lesions.—Hæmorrhagic catarrh of the large intestine and rectum, parenchymatous degeneration of the liver, peritonitis, a fibrinous exudation into the pericardial sac, ecchymoses on the epicardium, and conjunctival catarrh, were observed.

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Willach: Eine Cholera unter dem Wasserflügel in Schwetzingen. Centralblatt für Bakteriologie, Band xx., 1896, p. 187.

VI. BIRD FEVER.

Synonyms.—Bird septicæmia; Canary fever; Septic fever; Bird plague.

French: Maladie des canaries.
German: Kanarienseuche; Kanarienfiebre.

Definition.—An infectious disease of every class of bird, including the seminivorous, insectivorous, frugivorous, confined to cages and aviaries, running a rapid course and causing a great mortality. The most characteristic lesions are confined to the spleen, which is generally enormously enlarged, and the liver, and are manifested in the form of small whitish or yellowish nodules, varying in size from the minutest point up to a hemp-seed or larger.

Geographical Distribution.—This disease has been observed chiefly in canaries in various parts of Germany, Austro-Hungary, Russia, Italy, France, and America. It is very prevalent from time to time in all classes of birds confined in bird-rooms or outdoor aviaries in various parts of Great Britain.

Susceptible Species.—It is more commonly seen in canaries, but no species of the seminivorous, insectivorous, or frugivorous birds are immune It attacks the starling, blackbird, mynah, troupial, tanager, cardinal, siskin, toucan, sugar-bird, finch, mannikin, etc.—in fact, birds indigenous to the tropical, subtropical, temperate, or arctic zones, and equally those brought from North and South America, India, Australia, South Africa, Asia, and Europe.

Season.—It occurs any time of the year, but is more commonly observed during the early spring months, and in the months of July, August, and September. Birds are more prone to it during the moulting process. Age and sex do not show any difference in susceptibility.

Etiology.—It seems to be a contagious disease spread by cohabitation or by contaminated cages, even by those that have long been untenanted. It is usually introduced by a bird suffering from the latent infection purchased at a dealer's shop or coming from an infected bird-room or aviary. Seed and other bird-foods coming from a contaminated bird-shop may introduce it to a bird-room or aviary hitherto free from it. Bird-shows may propagate it. Clarke and his admiring follower, Creswell, maintain it is produced by feeding canaries during the breeding season on non-sterilised hard-boiled egg. Their theory is that the saprophytic organisms of putrefaction contained in the egg are so increased in virulence bypassage through the bird's intestine that it produces a disease that may afterwards be maintained by contagion. Clarke is unaware that the malady can arise in birds fed solely on seed. If this theory be correct, then the malady should be more frequent than it is already, for nearly every breeder of canaries rears his young birds on hard-boiled egg or the yolk remaining as a by-product after the albuminous part has been separated for use in calico-printing. This yolk is dried into flakes, and it therefore to a certain extent becomes sterilised, and is put on the market for the feeding of birds and for culinary and other purposes. The disease is often introduced from the continents of Europe, Africa, and South America, by the importation of birds from these countries, and in particular from Germany, which sends us thousands of German song or Roller canaries every year. Putrid water and insanitary cages and surroundings have been incriminated, also bacterial mouse virus. But experience shows these are only surmises to explain what is not generally understandable—the apparently healthy yet infected animal being the carrier. It occurs in birds that are only living on seed, as well as those living on fruit, insects, artificially prepared food mixtures, and hard-boiled egg. The reason it is seen more in the canary than in any other bird is because the former is very much more numerous. It spreads with a greater rapidity and with more deadly certainty in a bird-room than in an open outdoor aviary.

Bacteriology.—As in the case of many other infective diseases of animals, the bacteriology of this malady has not been thoroughly elucidated, although several earnest investigators have each incriminated a special organism, which differ from one another. According to R. H. Clarke,* who has given a great deal of attention to the disease in this country, the causal organism is that of Koch's, and probably also of

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* R. H. Clarke, "The Bird-Plague, or Septic Fever in Cage Birds," being a lecture delivered before the London and Provincial Ornithological' Society on October 12, 1897. Reprinted and enlarged in brochure form, 1898. Idle, Yorks: *Fur and Feather* Printing Works.

Davaine's, septicæmia induced in rabbits and birds by the inoculation of putrescent fluids, impure water, putrid ox-blood, infusion of meat, etc. Koch's bacterium belongs to the ovoid or bipolar series, staining at both poles and leaving a clear central space. Clarke has found that in some outbreaks Davaine's bacterium, which has usually no central clear space, and stains all over, is the prevailing form, and in others both forms coexist. He also asserts that both are generated in hard-boiled egg exposed to the air.

Clarke and his faithful follower, George Creswell,* maintain that this putrefactive, and therefore saprophytic, organism, developing in hard-boiled egg, when given to canaries during the breeding season, becomes very virulent in a series of passages through their intestines, so that it becomes pathogenic, and is then maintained by contagion. Clarke gives no description of the biological characters of the incriminated microbe beyond what is mentioned above. He alleges, however, that it is found in the spleen, liver, heart, blood, and other tissues, and reproduces the characteristic nodular lesions in the spleen and liver.

Kinyoun† observed a great mortality in a bird-dealer's establishment during the year 1905 in Washington, U.S.A. He found on section of the parenchymatous organs various-sized small yellowish nodules, surrounded by a zone of inflammatory tissue. Between the nodules were small patches which had undergone coagulation necrosis. The air-sacs were the seat of a catarrhal exudate, and a few birds showed lesions of enteritis.

Microscopical examination readily showed the presence of a small bacillus having rounded ends, and analogous to the bacillus of bubonic plague.‡ It slowly developed on agar-bouillon-peptone, and did not liquefy gelatine. On Haffkine's saline agar it became pleiomorphic, in an exactly similar manner to the plague bacillus. It grew on agar or bouillon-peptone if a small quantity of horse or calf serum were added to the medium.

This bacterium was especially abundant in the spleen, but it could also be isolated from the heart, blood, and other organs.

It was pathogenic for the rabbit, guinea-pig, white mouse, pigeon, sparrow, canary, blackbird, and song-thrush.

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* "Bird Notes," *Journal of the Foreign Bird Club.*

† *Centralblatt für Bakteriologie*, 1906, Band xxxviii., p. 329, and *Revue Générale de Méd. Vét.*, 1906, tome viii., p. 676.

‡ It should not be forgotten that a great number of mammals, as well as birds, including geese, ducks, pigeons, and fowls, are, according to Cantlie and Hunter, attacked with bubonic plague in great epidemics of this malady. Some authorities assert that if the plague bacillus does not belong to the group pasteurella, it is very closely related to those forming this group.

Rieck* studied, in May, 1888, an outbreak in canaries in Dresden. He found a mobile aërobic and anaërobic organism in the blood, which was capable of growing on the usual culture media, and which formed on potato a greyish-yellow layer. The cultures on inoculation killed the canary, sparrow, pigeon, and white mouse. The microbes were abundant in the lesions in the liver and intestines, as well as in the blood and all the other tissues. Mice died eleven to twelve days after ingestion of the cultures. Nodular lesions in the liver were found in the birds, and simulated those observed in fowl cholera and the bird fever so frequent in Great Britain. McFadyean† found a very small bacillus in pure cultures in the nodular lesions of the spleen and liver. The bacillus did not on inoculation produce any disease in the pigeon. Probably the causal organism is only a variety of the fowl-cholera organism.

Symptoms.—Usually the bird seems to be breathing rather quicker than usual, does not eat so vigorously, has a duller appearance, sits lumpy on his perch, and has an increased thirst; the fæces are soft and greenish, often liquid, very fœtid, and passed frequently. The dulness increases, the appetite is diminished or absolutely in abeyance, the bird is in a state of stupor, hides his head under the feathers of his neck, and forms himself into a ball, with feathers standing out in an open fluffy manner. The breathing is rapid and panting, the bird grows weaker, is difficult to rouse, sways on his perch, from which he may fall and then die in convulsions. Many birds stay at the bottom of the cage long before they die. Extreme emaciation, a prominent breast-bone, and lightness in weight, is a prominent symptom in many. There are, however, numerous instances in which birds never waste away, and only appear a little dull and slightly off their appetites just before they die in a somewhat sudden manner. In many the floor of the abdomen seems to be enlarged and bulging, due to the enlarged liver. The skin of this part may be of a purplish colour.

Death usually terminates the malady within one to two weeks. Some birds recover; to be a menace to others in the future. Recoveries, however, are quite exceptional.

Lesions.—The lesions are chiefly confined to the liver and spleen, which are nearly always very much enlarged, friable, and have a blackish or purplish appearance, dotted all over with circumscribed yellowish nodular points, varying in size from the minutest dot up to a maw, fig, rape, millet, or hemp seed, and which may coalesce and form large conglomerate nodules or masses. In many advanced cases the spleen, which is always of a tremendous size in this disease, is almost replaced by the nodules, which are only here and there intersected by small bands of the proper tissue of the organ; it has then more the appearance of brawn. In a few cases the only observable lesion in the body is an enlargement of the spleen, without any nodules.

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* Rieck, "Eine infektiöse Erkrankung der Kanarienvögel," *Deutsche Zeitschrift für Thiermedicin,* 1889, Band xv., p. 69.

† Unpublished observation.

The lungs are very rarely affected with the nodules. The writer has observed a few such instances, but always when the disease has also attacked the spleen and liver. The liver in some instances may be of a lighter or brownish colour, as if it had been washed. Cheesy nodules have been found in the sublingual glands under the tongue, most frequently on the left side. The lesions in the liver and spleen are due to coagulation necrosis forming caseous centres, this lesion, according to Clarke, being sufficient to distinguish it from tuberculosis. (This view is, however, erroneous, as the histological appearances are somewhat similar in both avian tuberculosis and in bird fever, and, on the contrary, the avian tubercular lesions differ essentially from the human and bovine tubercle. Even in different species of birds tubercles differ somewhat in structure.)

In the acute form of the disease extravasations may be found in the tissues, especially in the cranial bones, spleen, intestine, and peritoneum. These are, however, not very characteristic, as they may appear in other infections.

The blood, lesions, and other tissues, contain numerous micro-organisms.

The writer has observed old lesions in birds that have lived a year or more after coming from notoriously infected aviaries or bird-rooms.

Diagnosis.—This disease is very often mistaken for *tuberculosis*, which is very rare, if not unknown, in the smaller cage-birds. The lesions in bird fever are nearly always confined to the liver and spleen, whereas in tuberculosis they may be situated on any tissue or in any organ. In the case of doubt recourse should be had to bacteriological examination. In *septic fever*, although the spleen is very much swollen, there are no other lesions, and the liver appears normal. However, in some of the acute cases of bird fever the lesions resemble those found in septic fever.

Treatment.—As this disease is nearly always fatal, it should be prevented by strict isolation of every single bird until all apparent danger of its further spread is passed. Newly-purchased birds should be isolated for at least three weeks. Old or used bird-cages should never be purchased, as they are often the means of introducing the disease.

The contaminated bird-cages and feeding utensils should be thoroughly washed and scrubbed, and then immersed in a 10 per cent. solution of lysol, or in the cresol preparation the formula of which is given in the article on Canine Distemper. After this immersion they should be exposed to the sun or daylight until dry. In lieu of this immersion, they may be soaked with the crude oily cresylic acid, and then allowed to dry. The bird-room should be thoroughly scrubbed, and washed over with a 10 per cent. solution of the cresol compound mentioned.

Outdoor aviaries should be washed, scraped, limewhited with freshly slaked lime, or painted, and the woodwork brushed over with the green creosote oil of commerce, care being taken that nothing contaminated or likely to be contaminated is overlooked in the cleansing and disinfecting process. The earth in the outdoor runs should be dug over, the sand or excreta in the inner houses removed, and the floor scrubbed and disinfected. Mice and rats should be exterminated in the vicinity of aviaries, which should always be enclosed with a small-mesh wirework, so that sparrows and other small birds cannot get into the aviaries and carry disease from one to the other.

Unscrupulous persons, as soon as they discover the disease on their premises, often dispose of the whole of their birds and cages to dealers and other fanciers, and thus disseminate the contagion. Bird-shows may also spread the malady, which now exists in various centres in Great Britain.

VII. CATARRHAL ROUP.

Synonyms.—Roup ; Wet roup ; Avian contagious catarrh ; Fowl distemper.

French : Catarrhe oculo-nasal contagieux aviare ; Maladie des yeux des oiseaux.

German : Roupkrankheit.

Definition. — A contagious disease of birds, especially the fowl, pheasant, partridge, and other gallinaceæ, characterised by an elevation of temperature and a catarrhal condition of the ocular, nasal, pharyngeal, and laryngeal membranes, without any diphtheritic membrane, deposit, or ulceration of the mucosa.

Remarks.—The term "roup" or "roop" is of Scandinavian origin, denoting hoarseness. It is generally used by poultry-breeders and pheasant and partridge rearers to designate any catarrhal affection of the head. When a catarrhal fever exists without any false membrane, it is distinguished as "wet roup"; when accompanied by a diphtheritic exudate or ulceration, it takes the name of "diphtheritic roup," "diphtheritis," or "diphtheria."

According to many authorities this "wet roup" is only a milder or uncomplicated form of the diphtheritic roup. They base this assumption on the fact that an ovoid bacillus of the fowl-cholera or pasteurella type is found in both forms. But since there is so much difference of opinion respecting the actual causal organism of avian diphtheria, and as a catarrhal condition of the bird may exist without any diphtheritic lesion, the question of the unity or the duality of origin of these two maladies must for the present remain in abeyance. It is not even settled if there are not several bacteria capable of inducing a diphtheria in the bird. Bordet's researches in 1907 on the etiology of avian diphtheria have given a fresh impetus to further inquiry, which it is hoped will lead to some useful purpose in establishing the bacteriology of that malady on a surer foundation than is the case at present. Until then one had better take into consideration that an enzoötic catarrh without diphtheritic lesions often appears in birds, and it is only reasonable to suppose that it is a different disease—at least, clinically—to diphtheria. Therefore the writer will continue the usual custom, and describe it as a special morbid entity.

Symptoms.—These appear, according to severity, in three forms—namely, the *mild*, the *severe*, and the *severest*.

The Mild Form.—In this the affected birds are more or less dull, rather disinclined to move, do not seem eager for food, sneeze, and give an occasional hoarse cough. After a short time the mucous membranes of the eyes and nostrils become reddened and swollen, and discharge a thin fluid, which is soon replaced by a thick, viscid, or muco-purulent material, which often plugs up the nostrils or glues the eyelids together. In this form the disease runs its course in three to seven days, when recovery is usually complete.

Severe Form.—Beyond the above symptoms, the birds appear sleepy, the feathers are erect and open, the nostrils are completely plugged by a very thick discharge, so that the breathing becomes purely oral and accompanied by a wheezing or hoarse croaky noise, and often by an escape of watery liquid from the mouth. The appetite is completely in abeyance, and emaciation rapidly supervenes.

Severest Form.—In this the prostration and dulness are generally greater, and the discharges give off an offensive odour. The conjunctival membrane is so swollen that the eyelids bulge out and the membrane completely hides the eyeball. Very often the mucous membrane of the oculo-maxillary sinus or sac under the lower eyelid is involved in the catarrhal condition, the secretion of which accumulates in the lumen of the sac, and after a time undergoes a yellowish purulent or cheesy change, and forms a large tumour-looking body, which may eventually press the eyeball out of its cavity. A similar material may plug the nostrils, and in accumulating in these channels presses down the palate to such a degree that the breathing is obstructed and swallowing is often prevented. When both eyelids are intensely involved the bird cannot see its food, so that unless carefully attended to it may die of starvation. In this form, when the bird is suffering from somnolence, great prostration, and rapid emaciation, death very frequently results.

Treatment.—This disease spreads rapidly, and is very difficult to prevent, and still more difficult to eradicate once it has got a start in a large collection of birds. It often appears as if of spontaneous origin. Newly-purchased birds should be quarantined for a week at least. The usual media of contagion should be burnt, disinfected, or limewashed.

The affected birds should be kept in a warm, dry place, free from direct draughts, and fed on hot barley-meal, skim-milk, boiled grain or potato, with which some sodium salicylate or aspirin should be incorporated. The eyes and nostrils should be freed from the thick exudates, and sponged two or three times a day with a 1 per cent. solution of creolin, permanganate of potassium, or 1 in 1,000 perchloride of mercury or chinosol. The oculo-maxillary or suborbital sinus should be evacuated of its contents. In very valuable birds a pill, composed of strychnine, gr. $\frac{1}{32}$; quinine sulphate, gr. $\frac{1}{2}$; pulv. capsici, gr. i.; and ext. gentian, *q.s.*, should be given twice a day. The dose for pigeons or birds of that size is one-quarter to one-sixth of that required for a fowl. It might here be remarked that the common fowl will bear a much larger dose of strychnine than a man.

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VIII. RABBIT SEPTICÆMIA.

Synonyms.—Gotch.

French : Pasteurellose du lapin ; Septicémie du lapin ; Septicémie spontanée des lapins.

German : Kaninchenseptikämie.

History.—This disease was first studied by Gaffky, who in 1881, on inoculating some rabbits with the water of the Panke, induced a septicæmia which he found was due to an ovoid bacterium. In 1887 Theobald Smith, of the Bureau of Animal Industry, U.S.A., also investigated it, and a further study was made in 1888 by Thoinot and Masselin. It is not the same septicæmia as that of Davaine or of Koch, who induced an experimental septicæmia by injecting putrid blood into the rabbit.

Etiology.—This disease often appears to arise spontaneously. It may, however, be readily transmitted from rabbit to rabbit by the ingestion of food contaminated by the virulent fæcal material, and by inoculation of the virulent blood and cultures. In its acute form death occurs from septicæmia, but if it takes a slower course, the animal dies with all the signs of an intoxication.

Bacteriology.—The bacterium of this disease has all the forms and the staining properties and similar cultural characters to that of the avian-cholera organism. It kills the rabbit, guinea-pig, mouse, fowl, pigeon, and, in fact, most birds. Its virulence disappears in cultures in contact with the air after two or three weeks, and is killed in fifteen minutes by exposure to a temperature of 58° C.

Symptoms.—The rabbit appears dull, separates from his companions, eats very little, does not care to move, but forms himself into a heap or ball, with a dull, open coat. The eyes are fixed, and the ears droop. He gradually goes into a comatose condition, the appetite is completely lost, the breathing becomes quickened, and the animal falls on one side and dies without any convulsive movements. Diarrhœa is usually present at the last. As a rule the disease runs a complete course within twenty-four hours, rarely two to three days, and always ends in death.

Lesions.—The blood is black ; the muscles of the chest and abdomen are infiltrated ; the intestinal mucous membrane is congested and thickened, and covered with a brownish-red material ; the lungs are congested and ecchymosed ; the pleural cavities contain a reddish albuminous fluid. The pericardial sac is filled with a sero-fibrinous exudate ; the urine is albuminous ; the liver and spleen are slightly altered.

Haaland has encountered an epizoötic in *mice* caused by a pasteurella.

IX. RABBIT INFLUENZA OR CATARRH.

Synonyms.—Contagious snuffles.

French : Septicémie de Beck ; Pasteurellose du lapin ; Rhinite purulente épizootique du lapin.

German : Brustseuche beim Kaninchen ; Influenzaartigen Kaninchen.

History. — This disease was studied by Beck of Berlin during the winter of 1891. Ten years later (1901) Roger and Weil described it. Krauss studied a similar, probably the same, disease, but isolated a bacterium not having the characters of that found by the previous investigators.

Etiology.—The nasal discharge seems to be the principal medium of the virus, which evidently gains the mucous membrane of the nostrils and sets up infection. The hutches are likely to be contaminated. The disease chiefly occurs as an enzoötic in the rabbit. Sometimes the guinea-pig becomes infected, and Beck has seen a monkey affected by it.

Bacteriology.—The bacterium has all the morphological and staining characters of the microbe of rabbit septicæmia. It is also cultivable on all the usual media. By inoculation it kills the rabbit, guinea-pig, and mouse. The rat, fowl, and pigeon, are refractory. The bacterium is killed by desiccation in eight days at 37° C., and in seventeen days at room temperature.

Symptoms.—The disease appears in the form of a profuse nasal discharge, which soils the nostrils and sticks together the hairs of the upper lip. There is frequent sneezing, the eyes sometimes discharge a mucopurulent material, the appetite is lost, and the animal wastes away rapidly. The disease runs its course within a week to ten days, and nearly always ends in death.

Lesions. — The mucous membrane of the nostrils and bronchi is slightly congested. The pleural membrane is in some instances coated over with a fine greyish-white network of fibrinous exudate ; in others with an abundant deposit, which compresses the lungs and heart. The inferior part of the lungs is congested, infiltrated, and shows atelectasis. The liver is of a dark brownish colour, and engorged with blood. The spleen is not swollen.

The *hare* suffers from a similar disease due to an ovoid bacterium. The lesions are chiefly confined to the lungs in the form of a fibrinous pneumonia.

X. OTHER FORMS OF RABBIT SEPTICÆMIA.

(*a*) Eberth and Mandry studied a *spontaneous septicæmia* in the rabbit (Spontane Kaninchenseptikämie) in 1890. It was first observed by Eberth in 1882, and frequently since. They have incriminated a mobile Gram-proof bacillus with rounded ends, isolated or associated end to end, in the form of a diplo- or strepto- bacillus, or in the ovoid form. It does not liquefy gelatine, but grows on potato. It is pathogenic for the rabbit, guinea-pig, mouse, pigeon, and sparrow. The clinical characters and post-mortem lesions are similar to those mentioned under Rabbit Influenza. Sometimes, however, there are patches of broncho-pneumonia or peritonitis, the fibrinous exudate gluing the abdominal viscera together.

(*b*) In two outbreaks introduced by freshly-purchased rabbits, Koppanyi isolated an organism resembling that of Eberth and Mandry, Lefébure and Gautier, by its shortness and thickness, but differing by having a capsule. The constant lesions found were a fibrinous inflammation of the pleural and pericardial membranes, consecutive to an infectious catarrhal bronchitis with patches of broncho-pneumonia.

(*c*) *Davaine's Septicæmia* also attacks rabbits as well as mice, fowls, pigeons, and sparrows. Guinea-pigs and rats are not susceptible.

(*d*) Lucet has studied and described an epizoötic of a septic disease of the rabbit in the Loiret. He isolated a mobile bacillus measuring about 1·3 μ. It did not liquefy gelatine nor develop on potato. It was pathogenic for the rabbit and guinea-pig by inoculation.

Etiology.—The disease was not transmitted by cohabitation or by ingestion of the soiled food. Probably the infection gains an entrance to the system by wounds on the skin or upper part of the alimentary tract, caused by prickly herbaceous plants in the litter or in the food. Intestinal parasites, especially the *Strongylus strigosus*, may act in a similar manner. Ingestion of the virulent organic liquids or of cultures remains without effect.

Symptoms.—The intermaxillary or laryngeal region was swollen, painful, red, and firm; the under part of the neck and head was œdematous. Soon after a cough appeared, the respirations became difficult, and the nostrils discharged a greyish material. The animal was dull, mopy, hunched up in a ball, the hair was dull and open, the ears drooped, and the eyes were half closed. Later on the animal fell on its side, and died during convulsive movements and piercing screams. The disease ran its course in a few days.

Lesions.—In the centre of the primary swelling there was an infiltration of greyish sero-purulent material; a pinkish gelatinous œdema occupied the neighbouring parts. The muscles were soft and discoloured; the peritoneal cavity contained a reddish fluid, and sometimes adherent false membranes; the intestines were congested; the liver was enlarged; the spleen was enormous, black, and diffluent; the glands were infiltrated and hæmorrhagic; the pleural and pericardial cavities contained an amber-coloured transudation, or an inflammatory exudate with false membranes; the pus, exudates, and parenchymæ contained the bacteria in abundance.

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XI. DUCK SEPTICÆMIA.

Lisa in Italy investigated an outbreak in which all the affected ducks succumbed. The isolated bacterium was slightly larger than that of fowl cholera. It grew on gelatine, which it liquefied slowly, and gave on potato a hazel-coloured culture. It was encountered in abundance in the various media of the eye, also in the serous cavities and parenchymatous organs, and in small quantity in the blood.

Symptoms.—The disease commenced suddenly. The eyes were closed, and discharged tears. The birds were dull and very disinclined to move. Death set in within four days.

Lesions.—The eyelids were glued together by purulent secretion, the cornea was dotted with whitish spots, the lens was sometimes opaque. The pericardium contained a gelatinous exudate, and the myocardium was pale. The liver was soft, the intestine and mesentery congested, and in some birds the peritoneum was coated with a fibrinous exudate.

XII. SWAN SEPTICÆMIA.

(*a*) Fiorentini, in the summer of 1895, studied in Milan a disease affecting swans and certain species of geese, especially the Egyptian goose. He designated it the "hæmorrhagic septicæmia of the swan," and considered it was due to a slightly motile ovoid bacillus measuring $\frac{1}{2}$ to 2 μ in length by $\frac{1}{2}$ μ in breadth, and resembling in many respects a bacillus of the colon group. It was pathogenic for the guinea-pig, rabbit, fowl, pigeon, duck, goose, etc. The swan and Egyptian goose contracted the disease by cohabitation, but other species of birds remained free by this mode of contamination.

Symptoms.—These were not described. Young birds died in a few hours, and the adults after a few days' illness.

Lesions.—In the peracute form ecchymoses were found on the serous membranes, especially at the base of the heart, and a slight congestion of the intestine. In the acute cases there were patches of grey hepatisation of the lung, hæmorrhages, parenchymatous degeneration of the liver, and ecchymoses on the serous membranes.

(*b*) Trétrop, in January, 1900, investigated a disease occurring only in one species of swan—the *Coscoroba candida*—in the Antwerp Zoological Gardens. Other species of swans and geese, teals and ducks, were refractory. The organism incriminated was a cocco-bacillus, having a clear central space, and measuring 1·5 μ to 2·8 μ in length. It had oscillatory movements, and was aëro-anaërobic, but preferably aërobic. It grew equally well on bouillon, milk, agar, and gelatine, not so well on peptonised solution, and less on potato. It did not liquefy gelatine. It formed a very slight culture on potato. Milk was coagulated. Indol was formed in peptonised solution. The organism was pathogenic for the swan, various finches, mouse, and guinea-pig. It was well stained by carbol-thionine, but did not take Gram.

Symptoms.—The affected birds separated from the rest, and appeared stupefied. Movements were difficult and uncertain. Appetite was lost, but thirst continued. The diarrhœal evacuations were yellowish or greenish-yellow, with small whitish clots resembling the undigested discharges of infants. The acute form runs its course in four or five days, but it may last longer. Death takes place without convulsive movements.

Lesions.—The abdominal glands were enlarged, often reaching the size of a hazel-nut, and had a caseous appearance. The intestines contained a quantity of greenish-yellow fæcal material. The liver was very large, and dotted over with whitish spots and caseous nodules the size of a millet-seed. The lungs were congested, and occasionally contained one or two caseous nodules.

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XIII. INFECTIOUS ENTERITIS OF PHEASANTS.

Fiorentini studied an enzoötic in pheasants in the public gardens of Milan; a few months later the same disease, but in a septicæmic form, occurred in the swans. It was attributed to a bacterium similar to that of avian cholera, but was longer (1 to 2 μ) and thicker (0·7 μ), and very mobile. In culture media it formed streptococci, which sometimes were very long. It attacked by preference cock birds. It did not seem very contagious, and after a few months the majority of the birds became free from infection. Ingestion of the cultures produced in the pheasant a similar disease to that arising from accidental infection. Subcutaneous inoculation under the skin or into the veins of a few drops of a recent broth culture induced the disease. Fowls received large doses, but were refractory; it did not immunise them against avian cholera. Rabbits and guinea-pigs were also refractory.

Symptoms.—The birds were very much depressed, and staggered when they attempted to walk. The appetite was poor. Later on a profuse diarrhœa appeared; the dejecta were of a greenish or greyish-yellow tint. The disease ran its course in five to eight days. No coma or sleepiness was observed.

Lesions.—The mucous membrane of the intestine was congested, of a brownish-red appearance, and covered with a thick mucous or a croupous exudate. There were no ecchymoses on the serosa, nor exudates in the serous cavities. The liver was friable; the spleen was swollen, but without hæmorrhages.

XIV. OTHER SEPTICÆMIAS OF POULTRY.

The *septicæmia of fowls,* described by Lisi as occurring in 1892 and in the summer of 1893 in the provinces of Carrara and Massa, is, according to Lignières, Nocard and Leclainche, only avian cholera. Lignières has shown that the *hæmorrhagic septicæmia of the duck and fowl* studied by Rabieaux belongs to the same category. The *epizoötic disease of the fowl* described by Carlo Mazza as occurring in Upper Italy runs a similar course to that of fowl cholera. The micro-organism is motile, larger than that of fowl cholera; in cultures it takes on the form of short ovoid rods, isolated or in pairs, and nearly always having no clear central space. The *symptoms* and *lesions* are similar to those found in fowl cholera or fowl plague. There is, however, nearly always a serous infiltration of the lungs, extensive or localised, or an exudation into the pectoral cavity.

The bacteria are not found by direct examination of the blood, and only exceptionally in the exudates of the lung or intestine. Guinea-pigs are refractory to inoculation; the rabbit only slightly susceptible; the pigeon and fowl most surely succumb either by subcutaneous, peritoneal, or intrapectoral inoculation of cultures. Young fowls are infected by inhalation of spray cultures.

Severio Santoni describes a form of septicæmia which closely resembles fowl cholera. It is determined by a short immobile bacterium, which liquefies gelatine and gives in every culture medium maintained at ordinary temperature for twenty-four to forty-eight hours an intense red coloration. Sometimes it is difficult to distinguish the bacterium from a coccus. The virulence of this microbe is so great that water in which a blood-clot from a bird dead of the disease has been washed kills, when injected under the skin, fowls, rabbits, and guinea-pigs. On post-mortem examination, nothing to attract attention is discerned. But on microscopical examination the microbe is found swarming in every tissue (*Centralblatt für Bakteriologie*).

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XV. PARTRIDGE CHOLERA.

Karlinski has observed in Herzegovina, in the Balkans, a disease in the summer partridge (*Perdix saxatilis*) in which there is progressive emaciation and complete loss of feathers. It causes great mortality. On post-mortem examination the muscles of the breast and limbs contain numerous small abscesses, the size of a hemp-seed or pea. The visceral mucous membranes are congested and softened. The lungs are hyperæmic and consolidated; the liver and spleen are congested.

The blood contains numerous micrococci or diplococci; they are very abundant around the abscesses. Cultivated in the temperature of the room, the long rods transform themselves into entire colonies, taking on a round form, slightly granular on the surface. They do not liquefy gelatine. Passed through pigeons, they increase in virulence. An old culture accidentally thrown into the poultry-yard caused a great mortality among the fowls. Carnivorous birds, such as eagles, vultures, and hawks, consuming the dead birds, died shortly after.

XVI. SEPTICÆMIA OF YOUNG PHEASANTS.

Klein studied an enzoötic in young pheasants which destroyed about 700 out of a total of 1,800 during the months of June and July, 1893. He isolated from the heart blood a pure culture of a motile bacterium resembling the *Bacillus coli communis*, but not quite so mobile, nor did it coagulate milk at 37° C., as does the latter. Inoculation of a drop of culture under the skin of a young pheasant killed the bird in twenty-four hours. The fowl, pigeon, rabbit, and guinea-pig, were not killed by an inoculation of $\frac{1}{4}$ to $\frac{1}{2}$ c.c. of the culture.

Symptoms.—The disease ran its course in a few days, or this was prolonged for a week or more. The birds were depressed and sleepy; diarrhœa was present in some, but absent in others.

Besides avian cholera, avian plague, avian diphtheria, and catarrhal roup, pheasants are often affected by gape-worm, aspergillosis, coccidial enteritis, etc., which see under their respective headings.

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XVII. INFECTIOUS FOWL ENTERITIS.

Definition.—This is an acute fatal infectious disease affecting fowls, but not pigeons and rabbits.

History.—Klein, from 1888 to 1895, studied five enzoötics of this malady, three of which occurred in Ireland and two in England. Since that period it has been very prevalent, and causes great mortality.

Etiology and Bacteriology.—The bacterium (*Bacillus gallinarum*) incriminated by Klein is immobile, aërobic and anaërobic, and grows well in broth, on gelatine, but not on potato. It is found in small numbers in the blood, but in a greater quantity in the spleen, cæca, and rectum. Natural infection takes place through the alimentary canal, as ingestion of food contaminated by virulent fæcal material insures the development of the disease. Subcutaneous inoculation with the blood and splenic pulp induces the natural disease in the fowl. Guinea-pigs and mice, when inoculated, died within five to six days. Rabbits were only overcome by a very large dose. Pigeons were refractory. Animals recovered from the disease cannot be reinfected. Cultures in alkaline veal-broth become attenuated under the influence of a temperature of 55° C. for twenty minutes, and of this virus $\frac{1}{8}$ c.c. no longer kills fowls, but instead gives them immunity.

Period of Incubation.—The period of incubation after inoculation lasts as long as five days, at the end of which diarrhœa appears, to be followed twenty-four to thirty-six hours later by death.

Symptoms.—The disease commences with diarrhœa, the dejecta being fluid and of a yellowish-green or greenish colour. There is great depression or dulness, but never sleepiness or drowsiness. In a day or two after the diarrhœa has set in the fowls are found dead.

Lesions.—Examination of the birds half an hour to an hour after death shows that the blood in the heart is not coagulated. The liver is enlarged, soft, and friable. The spleen is two or three times its normal dimensions, soft, but never hæmorrhagic. The serosa of the intestine is injected; the mucosa congested, but without hæmorrhage, yet coated with grey or yellowish mucus.

Diagnosis.—Pigeons are insusceptible, rabbits only very slightly susceptible. The heart blood contains relatively few bacilli; the latter are a little longer and thicker than in fowl cholera. The spleen contains the bacilli numerously, and also the vessels of the liver; the mucus of the intestine contains the bacilli in almost pure culture. Although resembling in many respects in cultural characters those of fowl cholera, the bacillus of fowl enteritis grows in disc-like colonies on the surface of the culture medium, and is more cylindrical; in gelatine streak culture it grows much faster, and forms a broader, less translucent band than that of fowl cholera. Feeding fowls with the intestinal contents, subcutaneous inoculation with blood or splenic tissue, or with artificial cultures, induce the disease, but not until the third, fourth, or fifth day. By the sixth or seventh day most of them are dead. They rarely survive till the eighth day, nor do they die before the fifth day. Thus are the differential characters of this disease placed opposite those of fowl cholera.

Prevention.—Clearance of the fowls from the contaminated premises, isolation of the diseased, and disinfection of the houses or sheds in order to prevent further contamination, are advised by Klein.

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XVIII. GROUSE DISEASE.

Whether the so-called "grouse disease" includes more diseases due to various bacterial or parasitic organisms than one specific morbid entity is not known. According to Cobbold, who investigated the grouse epizoötic of 1872, this particular outbreak was due to the injuries produced by a nematode worm (*Strongylus pergracilis* of Cobbold or the *Trichostrongylus pergracilis* of later authorities). He asserted, "As regards the so-called 'grouse disease,' during one season it may be due to tape-worms, during another to strongyles, during a third to excessive abundance of both these parasites. Unfortunately, other avian epizoötics not necessarily due to parasites of any kind may be mistaken for helminthic epizoöty."

During 1889 and 1890 Klein investigated a grouse disease occurring in the North of England and in Scotland. It attacked chiefly the red grouse (*Lagopus scoticus*), and although only one county was infected in 1887, it had by 1889 invaded nearly every part of Scotland. In the summer months thousands of the birds succumbed.

Bacteriology.—Klein incriminated a very motile bacillus, oval, coccus-like or rod-like in outline. It forms gas bubbles in shake culture, and rapidly curdles milk. It does not form spores. Bouillon-peptone is rendered intensely turbid. On agar it forms a thick white opaque layer.

It does not liquefy gelatine, but gives an abundant culture in it. It easily and profusely grows on potato. It alters the agar of Würtz in twenty-four hours, and forms a quantity of indol. It is decolorised by Gram, but can be stained with the ordinary aniline dyes. There is a great similarity between it and the *Bacillus coli*. By inoculation into the guinea-pig and mouse general infection and rapid death are produced, mice being more susceptible than guinea-pigs. In both animals it produces bilateral pneumonia. Sparrows are also susceptible, but less so than the common bunting and yellow-hammer, which are highly susceptible, and in which it also produces bilateral pneumonia. Fowls, pigeons, and rabbits, are insusceptible.

Etiology.—Grouse alone appear only susceptible to natural contagion, but the mode of infection has still to be elucidated.

Symptoms.—The diseased birds are easily and early recognised by the keepers by the manner in which they fly, as they do so in a zigzag manner, quickly regaining the ground. During the breeding season the characteristic clear call of the healthy bird, especially the cock, becomes hoarse; the plumage is dull and open; the eyelids, which are normally bright red, become purple in tint. After a short period the prostration increases, the birds fly with difficulty, and, if followed, can easily be caught. Thirst seems to be extreme, and in consequence the birds make for streams or watercourses, and here a great number of carcasses are often found. The disease runs its course in a few days, and generally ends in death.

Lesions.—The carcass is extremely wasted; the intestines are ecchymosed, especially on the serous surface, and the mucous membrane has patches of congestion on it. The liver is congested, and has greyish-yellow points in its substance corresponding to patches of necrosis; the spleen is not enlarged; the lungs are hepatised; the heart contains coagulated blood.

In the diseased lung and in the liver there occur in the vessels and in the extravasated blood numerous bacilli, singly, or more commonly in larger or smaller groups, sometimes forming emboli in the capillary bloodvessels. Only a few bacilli are occasionally seen in the heart blood.

The Committee of Inquiry on the Grouse Disease presented a magnificent report in the form of two volumes, entitled "The Grouse in Health and Disease," 1911, which concluded that the grouse disease was due to the nematode parasitic worm *Trichostrongylus pergracilis*, and thus confirmed Cobbold's conclusions drawn in his work in 1872. The investigators also found many cases of disease due to the presence of a coccidium, the *Coccidium* (*Eimeria*) *avium*. They negative Klein's theory, which they think was based on the misconception of facts (*vide* Grouse Disease in Vol. II.).

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XIX. INFECTIOUS PARALYSIS OF THE OSTRICH.

Synonym.—Leg-weakness.

French : Maladie de l'autruche.
German : Infektiöse Krankheit der Strausse.

General Remarks.—Enzoötic paralysis is not a rare disease in the fowl, turkey, goose, and duck. It is also seen in all classes of indigenous and foreign birds confined in aviaries or cages. In the domestic poultry it is usually observed in well-bred, heavy, fast-growing birds, especially if they are confined to a too limited space or housed in damp sheds. Not much attention has been given to the affection, in consequence of the low price of the subjects attacked. In the case of the ostrich it is quite another matter, as such birds are generally of a high value, and a disease of this kind renders them quite useless. Therefore it behoves the veterinary surgeon to inquire into its nature, so as to be able to advise as to its prevention.

History.—Marx in 1900 observed this affection in forty ostriches in the Frankfurt Zoological Gardens. They had not long been imported from the Soudan. Robertson observed in 1909 a similar disease in the neighbourhood of Cape Town, where it had been noticed by ostrich-breeders to be on the increase during the previous six years, and seems to be yearly becoming more common. Several farmers have sustained severe losses, to the extent of many thousand pounds.

Etiology.—Nothing is known for certain how the birds become infected. Robertson is convinced that the common, if not the only, source of infection is contaminated food and water. He thinks certain grounds are contaminated in consequence of birds being continually bred and reared on the same spot.

Bacteriology.—In the Frankfurt outbreak, Marx isolated an immobile ovoid bacterium, which did not form spores, but was destroyed when kept at 60° C. for an hour. It was easily grown on the usual culture media. In bouillon it rendered the medium turbid in a few hours, and afterwards threw down a deposit. On gelatine its aspect is similar to the growth of the coli. On agar, after twenty-four hours, one observes greyish-white colonies, iridescent by transparence, and then a thick, moist, greenish-white layer. It grows on potato fairly easily, giving a brownish viscid layer. Milk is coagulated in three to twenty-four hours; glucose is fermented, but no indol is formed. Inoculation of $\frac{1}{100}$ c.c. killed the grey mouse within forty-eight hours. The pigeon is killed in two or three days by an intramuscular injection of ½ c.c. of culture, showing before death conjunctivitis and lachrymation. Small birds, such as the weaver and chaffinch, were killed with a trace of culture or blood within twenty-four to forty-eight hours. The rabbit and guinea-pig were refractory.

In Robertson's cases, smears from the tissues under the inflamed mucous membrane gave an almost (in many cases a quite) pure culture of a short bacillus, fairly regular in shape, which stained with the ordinary aniline dyes, but not by Gram. In ordinary culture media it was anaërobic and aërobic, and grew fairly well. On plain agar it gave a copious white growth along the needle track, the culture being slightly raised in the centre. On glycerine agar it gave a copious white growth, as on plain agar or on malachite green agar. The colour was discharged along the needle track, with a copious raised growth. On sugar agar stab, growth occurred along the stab and on the surface, and gas was evolved. In plain glycerine or sugar bouillon it gave a growth in all, rendering the media turbid and evolving a foul odour. On potato it gave a free growth, raised on the surface, and a brown colour, somewhat resembling an old glanders culture. Milk was not coagulated. Gelatine was not liquefied.

Inoculation subcutaneously of 25 c.c. of a twenty-four hours bouillon culture into a cock ostrich killed it in ten days; the bird had its head twisted round to its flank, and three days previously showed paralysis, and crawled about on the points of its hocks, just like a natural case of the disease. The duodenum was red, congested, and inflamed, and covered on its lining membrane with a thick croupous exudate, dirty grey in colour. Cultures were grown which corresponded with those obtained from a natural case of the disease. Robertson thinks that the bacteria do not invade the general tissues of the body, but remain located in certain parts, and form toxins and poisons which produce symptoms peculiar to this disease.

Symptoms.—In Marx's cases the ostrich was reluctant to come out of its place, its legs were doubled up under its body, and were paralysed and limp. The head and neck were drawn behind on its back, and seized with spasmodic movements. The appetite was preserved. Death supervened after three to four weeks. An abundant lachrymal discharge was noticed in every case. In Robertson's cases there was an inability to rise, the interference seeming to be in the points where the toes join the legs; the toes are bent on themselves at right angles, and the birds make futile (and, if scared, persistent) efforts to rise, sometimes staggering along for a considerable distance on hocks or heels. The disease is sudden in its onset, and occurs at irregular intervals in certain areas. Birds of all ages are affected, but chiefly the young ones. It may occur in the sitting hen. The appetite remains good. There does not seem to be any pain. They may live a long time in this paralysed condition, even several months, but during this long period they become emaciated, the thighs atrophy, and the feathers become matted with fæces. The temperature may be quite normal (98·5° to 100° F.). Similar symptoms to these are seen in other species of birds, including farmyard poultry, aviary and cage birds.

Lesions.—In Marx's cases the lesions were confined to the abdominal cavity. Peritonitis of a more or less intense degree was present. The spleen contained a few purulent foci in every instance. A few showed enteritis with necrotic patches confined to the mucous membrane. In Robertson's cases the duodenum was congested and thickened, the mucous membrane resembling velvet pile of a dark maroon colour. This inflammation was traced through the intestines for about a third of their length. The cæca were congested and stained with blood, and showed traces of inflammation. The submucous layer of the intestine was thickened and infiltrated with a clear, straw-coloured fluid.

Diagnosis.—This disease cannot well be confounded with avian cholera, which also affects the ostrich, emu, and cassowary, as well as other avian species. The same may be said of tuberculosis. It is quite different from rickets, rheumatism, and the chronic articular form of avian cholera. In the chronic form of spirillosis of poultry, paralysis is occasionally encountered.

Treatment.—Probably this disease is conveyed by some ectozoal, or even entozoal, parasite, as it does not seem to be transmitted by direct infection. Robertson advises removing the troop to fresh ground, as in his experience the disease was stayed entirely by taking the birds away to new pastures, and feeding them with freshly-cut lucerne and rape. Intestinal disinfection may be tried on the affected birds. For this purpose cyllin, β-naphthol, sodium salicylate, quinine, or bismuth, deserve a place.

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XX. PIGEON SEPTICÆMIAS.

(*a*) Veranus A. Moore studied an affection resembling in its clinical manifestation avian plague, in several outbreaks occurring from 1891 to 1895 in New Jersey, U.S.A., and causing a considerable loss of pigeons in that region, where it was known to pigeon fanciers under the name of "megrims."

Bacteriology.—He isolated a motile bacillus of 1 μ to 1·6 μ in length, and which was cultivable in bouillon, on gelatine, agar, and potato. It was not isolated from the blood, but the cerebral exudate was very virulent. The bacillus resembled the non-virulent type of the paratyphoid bacillus of swine fever, and which has been designated by Theobald Smith as the γ variety.

Etiology.—It attacks by preference young birds or squabs, and is not uncommon in Great Britain. The mode of infection has not been determined, but probably it is conveyed from bird to bird by the many epizoa infesting pigeon-lofts. Moore's bacillus kills the rabbit within thirty-six to forty-eight hours, and the guinea-pig in five to ten days. The pigeon is only destroyed by large doses (0·2 to 0·5 c.c.). The pig is refractory to subcutaneous or intravenous inoculations of 5 c.c. of the culture. The bacillus is very resistant to antiseptics ; lime-water is without effect on it, and a 2 per cent. solution of carbolic acid destroys it only after an hour's contact.

Symptoms.—The symptoms are chiefly confined to the nervous system. The bird is dull, twists or turns his head from one side to the other, or only to one side ; the eyes remain closed, there is no appetite, and sometimes fits of vertigo or convulsions are observed. It usually runs its course in from ten days to a fortnight. Nearly all the affected birds die.

Lesions.—The subarachnoid spaces in the vicinity of the cerebellum and posterior lobes of the cerebrum contain a yellowish-grey and friable exudate, which is about 1 millimetre in thickness, and formed of degenerated cells and micro-organisms. No other lesions are observed.

(*b*) Leclainche observed in 1893 a septicæmia in freshly-caught wood-pigeons confined in an aviary, in which they had been for a few days before the disease appeared. In about a week quite one-third of the birds had succumbed.

Bacteriology.—An aërobic and anaërobic bacillus was isolated. It grew on simple or peptonized bouillon, gelatine, agar, and potato. It killed the wood-pigeon, rabbit, guinea-pig, and pigeon. The fowl, cat, and dog were refractory. The organism was found in the blood, at least in the later stage of the disease. It was abundant in the spleen, kidney, and swarmed on the surface of the inflamed intestinal mucous membrane and in the dejecta.

Etiology.—The disease was readily transmitted from wood-pigeon to wood-pigeon by contact, less readily to the domestic pigeon. Food soiled with the virulent fæces conveyed the malady. The disease was also conveyed to the wood-pigeon by ingestion or by the various methods of inoculation of virulent cultures. The rabbit was killed in forty-eight hours by intravenous inoculation. A caseous degeneration was set up in the muscles in contact with the seat of subcutaneous inoculation. The pigeon was killed in four to six days by intravenous inoculation. The guinea-pig succumbed in about five days following an intraperitoneal inoculation. Subcutaneous inoculation set up local changes, which were sometimes followed by a fatal intoxication. The fowl resisted an intravenous inoculation of 1 to 3 c.c. of culture bouillon. The microbe in cultures remained virulent for several months. It was killed in a few minutes when submitted to a temperature of 60° C.

Symptoms.—As in many septicæmic processes in doves, pigeons, and other birds, the invasion is marked by stupor; the bird seems fixed to one spot, the eyes are closed, the feathers are open and dull, and the body is "all of a heap" or "lumpy." The symptoms increase in a few (three to twelve) hours, when the dove can no longer stand, but, being so weak, rests on its abdomen on the ground. There is diarrhœa, which is at first fæcal, then becomes of a greenish albuminous liquid material which contains gas bubbles. The course is rapid, the weakness increases, and death takes place in a complete state of sideration, on an average within twenty-four to forty-eight hours. Sometimes the disturbance takes on the convulsive form; the bird falls on its side, some contractions are observed, and the head is turned backwards, and the creature maintains this attitude until death takes place. (This latter form is, however, common to many affections in birds.)

Lesions.—The mucous membrane of the small intestine is congested, thickened, and contains here and there hæmorrhagic spots. The mesenteric vessels are distended with blood. The liver is enlarged and congested. The lungs are normal. The myocardium has a washed pale tint; the ventricles contain coagulated blood. There is no pericardial effusion. The muscles have an ochreous tint, and appear as if boiled.

(*c*) Sanfelice in 1894 studied a septicæmia of pigeons, but gave no clinical description of it. The lesions consisted of a pseudo-membranous exudate, covering the peritoneum, in the region of the intestine and liver, and which glued the organs to one another. The spleen was swollen, and sometimes there was intense inflammation of the mucous membrane of the oviduct and some purulent foci in the kidneys.

The exudates, and the blood of the heart, liver, and spleen, contained a bacillus having all the characters of the colon group. The microbes were especially abundant in the exudates; they were not so plentiful in the liver, and less so in the spleen, where they were united in a mass and included in the round cells. The bloodvessels only contained a small number.

Inoculation under the skin of a pigeon of 1 to 1·5 c.c. of the culture only caused an abscess. Peritoneal injection of a small dose killed in twenty-four hours. On post-mortem examination a peritoneal exudate with a fluid transudate, and tumefaction of the spleen were observed. The guinea-pig and rabbit reacted in a similar manner. Subcutaneous inoculation did not confer any immunity against a peritoneal injection. Healthy pigeons nearly always contain a bacillus identical to that isolated from the sick birds.

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XXI. EPIZOÖTIC DYSENTERY OF THE FOWL AND TURKEY.

Lucet described this as occurring during spring and autumn on a certain number of farms in the Loiret. It had little tendency to spread, but seemed confined to the place. It was frequent during summer, but always disappeared when the cold season set in.

Etiology.—The affection is not transmitted by cohabitation. Ingestion of the viscera is certain to transmit the disease. It attacks fowls and turkeys of any age or breed.

Bacteriology.—The causal agent is considered to be a short aërobic and anaërobic bacterium, cultivable in bouillons and on the different solid media, including potato. The cultures ingested do not give the disease. The pigeon and guinea-pig are refractory. The rabbit is only killed by intravenous injection. Subcutaneous inoculation of the organic juices or the cultures only kills about half of the cases.

Symptoms.—At the beginning of the disease the birds show a want of appetite, they isolate themselves from the rest, the comb or wattles become pallid, and the internal temperature is raised a few degrees beyond the normal (106·5° F.). Later on diarrhœa sets in; the fæcal material is at first bluish-green, and then becomes yellowish and contains strings of blood. The wings droop, and the feathers are dull and open. The birds appear sleepy, and when made to move stagger. Towards the end the weakness becomes profound, the temperature goes a few degrees below the normal, and death takes place after nine to thirteen days' suffering. Some birds linger for three weeks after showing changes of improvement and relapses, and others completely recover.

Lesions.—These vary according to the rapidity of the course of the malady. In the ordinary acute form the blood does not coagulate; the pericardial sac contains a greyish serous fluid; the myocardium is soft and ecchymosed; the liver is enlarged, friable, and engorged with blood; the spleen is enlarged; the intestine is congested and contains mucus and a greenish fluid. After the disease has been in existence for some time, there is parenchymatous myocarditis, pale blood, and a fatty atrophic liver. The blood, intestinal contents, and the parenchymæ contain the specific microbe.

Prophylaxis.—This should be carried out on the usual hygienic principles.

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XXII. SEPTICÆMIA OF THE FOWL AND TURKEY.

This disease has been studied in 1894 by Lignières in the fowl, and in 1896 and 1897 by Martel in the fowl and turkey. Like McFadyean's, Lucet's, and Dodd's diseases of the turkey, it has raged on as an enzoötic; probably they are only different clinical manifestations of the same etiological factor.

Bacteriology.—Lignières isolated a colon bacillus which did not kill the fowl by ingestion or by subcutaneous or intramuscular inoculation; even intravenous injection of 1 to 2 c.c. of the culture did not always prove fatal. The pigeon is killed in twenty-four hours by an intravenous injection of 1 to 2 c.c. of the culture. The rabbit and guinea-pig are killed in twenty-four to forty-eight hours by an intrapleural or an intraperitoneal injection, whereas a subcutaneous inoculation only produces an abscess.

The colon bacillus isolated by Martel kills the fowl by intramuscular inoculation; ingestion and intravenous inoculation remaining without effect. The rabbit is killed in nineteen days by subcutaneous inoculation, in seven days by intravenous injection, and in twenty-four hours by intraperitoneal injection. The guinea-pig succumbs in fifteen to twenty-eight hours by intramuscular or intraserous inoculation. The white rat and grey and white mice are destroyed in a few days by subcutaneous inoculation. The pigeon and dog are refractory.

The ingestion of cultures or virulent animal materials always give negative results in all species. The blood, spleen, liver, and pericardial false membranes contain the bacillus, which has all the reactions of the paratyphoid group.

Symptoms.—The symptoms are not well defined, but Martel has mentioned loss of appetite, a spumous diarrhœa, sleepiness, and conjunctivitis.

Lesions.—The lesions consist in an inflammation of the intestinal mucous membrane, accompanied by tumefaction of the spleen and sometimes pericarditis, with abundance of false membrane.

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XXIII. PARATYPHOID DISEASE OF THE FOWL.

Lignières and Zabala described in 1905 a new disease in the fowl, which they named *avian salmonellosis*, as the incriminated micro-organism closely resembled the bacillus of swine fever.

This disease, occurring as an epizoötic, was observed several times in the neighbourhood of Buenos Ayres, where it occasioned great losses. It does not affect the pigeon, guinea-fowl, duck, or goose.

The **Symptoms** and **lesions** closely resemble those of avian cholera and avian plague. The spleen is, however, very much enlarged. Inoculation of healthy fowls, pigeons, guinea-pigs, and rabbits with a few drops of blood from birds dead of the disease do not prove fatal. But intravenous injection of cultures kills the fowl within four or five days, with lesions of the natural disease, especially the enormous enlargement of the spleen. The pigeon is readily destroyed by intravenous inoculation, and sometimes by subcutaneous inoculation. Geese and ducks are refractory. The rabbit is killed in forty-eight hours with an intravenous injection of 1 c.c. of the bouillon-peptone culture, septicæmic lesions being produced. Subcutaneous inoculation, which does not often kill the fowl, causes in the rabbit œdema, which disappears and leaves the animal quite well. Intraperitoneal or subcutaneous inoculation does not kill the guinea-pig.

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XXIV. "GOING-LIGHT" IN BIRDS.

Synonyms.—Infectious asthenia; Asthenia in poultry; Bacterial infection of the duodenum.

"Going-light" is a common condition in every class of bird, whether at liberty in the farmyard, or confined in the aviary or in the cage. Its precise nature has not been determined, but many authorities assert it is a septicæmia.

Dawson found in the fowl suffering from a wasting disease, without any appreciable symptom beyond gradual and profound loss of weight and emaciation, a catarrhal duodenitis, which he considered was due to a pure culture of a micro-organism, closely resembling the *Bacillus coli communis*, occurring in the lumen of the intestine. This microbe was 1 to 1·3 μ in length, and 0·5 μ in width, with rounded ends, and often in pairs. It did not stain with acid or alkaline methylene blue, carbol fuchsin, or alcohol dyes, but was readily coloured with aqueous solutions of the same dyes and by Gram's method. It was aërobic, facultative anaërobic, cultivated at 50° to 120° F. in acid or alkaline beef bouillon, with fœtor; in glucose, saccharose, or lactose bouillon with the production of acid. It coagulated milk, and grew on gelatine, agar, and potato. Cultures injected subcutaneously killed the guinea-pig, but not the rabbit, the latter, however, succumbing to intraperitoneal injections. Pigeons, rats, mice, and poultry were refractory.

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XXV. SEPTIC FEVER IN CAGE-BIRDS.

Synonyms.—Bird typhoid ; Bird typhus ; Bird cholera.

French : Choléra des canaris.
German : Kanariencholera ; Kanarienseuche of Zürn.

Definition.—A contagious and inoculable disease in cage-birds, more especially in canaries and other finches, and producing symptoms of enteritis and diarrhœa.

History.—Kern studied this disease occurring as an enzoötic in canaries in October, 1895, in Budapest. He isolated a mobile bacterium from the blood, and cultivated it on bouillon, gelatine, agar, and potato. The cultures killed the canary, sparrow, goldfinch, and white and grey mice. The pigeon, duck, and fowl were refractory. By inoculation the cultures killed the sparrow within three days, and by ingestion the canary and sparrow within three to six days. It was extremely contagious.

Tartakowsky studied in 1898 a septic enteritis in various birds of the passerine order, including the crossbill, titmouse, goldfinch, siskin, and, to a lesser degree, the canary, in St. Petersburg, where it was repeatedly observed. He isolated a very mobile Gram-negative bacillus measuring 2 to 2·5 μ in length by 0·6 to 1 μ in thickness. In bouillon its growth was rapid, causing a uniform turbidity, and after three or four days a thin, somewhat solid, layer on the surface. It did not liquefy gelatine. Milk was not coagulated. On potato the culture was feeble ; when the medium became acid it was *nil.* No indol was produced. The bacilli were found in the blood, spleen, liver, free between the cells, singly or united ; in the intestines they were sometimes in a pure culture. Ingestion of an emulsion of liver or a pure culture by crossbills gave the disease ; subcutaneous or intramuscular inoculation of a pure culture on agar rapidly killed the crossbill and the bullfinch. The pigeon and fowl were killed by the injection of certain cultures only ; they resisted it by ingestion.

Gilruth observed a contagious disease in canaries during the summer of 1899 in Christchurch, New Zealand. He isolated a facultative anaërobic bacillus, which was very irregular in length, varying from almost a coccus to the length of the *Bacillus typhosus,* often in pairs, motile, staining bipolarly, and Gram-negative. It was cultivable on the various nutrient media. It did not liquefy gelatine. It rapidly grew on neutral broth, causing a uniform cloudiness. It did not coagulate milk. Neutral red was completely decolorised. On potato it formed an almost invisible growth. Indol was not formed. Pure cultures were pathogenic for the rabbit, guinea-pig, mouse, and pigeon, but not for the ordinary fowl. The canary, when inoculated with a drop of the broth culture, showed all the symptoms and lesions of the natural disease. The cage vacated by the dead bird was highly infectious to fresh birds placed in it.

Pfeiler encountered an enzoötic disease in canaries, of which over 100 succumbed. It was extremely infectious, and considered by him to be due to a bacterium of the paratyphoid group. This organism was found in the blood of the heart and other organs, and, when stained by the usual aniline stains, closely resembled the organism of avian cholera. It readily grew in profusion on the usual culture media. It was Gram-negative, and, when examined in hanging-drop preparations, very motile.

During life, the birds manifested the symptoms characteristic of most of the acute septicæmias of the bird, and in addition a well-marked profuse diarrhœa.

The post-mortem lesions were a catarrhal or hæmorrhagic enteritis. In addition, the majority of the dead birds showed a fibrinous or sero-fibrinous pleurisy, peritonitis, and subepicardial and subendocardial hæmorrhages. Nearly all had a swollen or enlarged spleen.

The writer has frequently observed this disease in birds sent to him from various parts of Great Britain during the last ten years.

Etiology.—Although this disease is generally introduced into a bird-room or aviary by sick or recovered birds coming from a dealer's shop or other infected place, the infection remains in a cage for years, killing off, one after another, every bird placed in it. This is very noticeable in the spring-time, when householders purchase canaries for their song. In some instances coming to the notice of the writer five or six birds have been purchased one after another, and all have died; in other instances the newly-purchased bird has killed off every one of the acclimatised birds in the household. It is not known if the causal organism is of saprophytic origin.

Period of Incubation.—This varies according to the virulence of the infection. In acute cases it may vary from a few hours to one to three days. In less virulent cases it may be as long as one to three weeks. Cold, wet weather, draughty rooms, and dirty surroundings, favour its propagation.

Mortality.—Although the mortality is great, it does not reach that of bird fever, in which nearly every bird attacked succumbs.

Symptoms.—The affected bird has a capricious appetite, often goes to the seed-pot and throws out the seed without eating any; at other times he feebly attempts to shell the seed without finishing the act. He seems dull and drowsy, pants or breathes quickly; the feathers have a dull, lustreless, and open appearance; the wings and tail droop. He seems weak, has a difficulty to get on to his perch, and prefers to rest on the bottom of the cage. Thirst is often very great, and diarrhœa sets in. In an advanced stage the bird places his head under his wing, and forms himself into a ball, breathing quickly, and in doing so moves his body at each inspiration and expiration. His eyes are closed, and he remains in a stupefied state. If roused, he attempts to move, and in doing so dies during a series of convulsive movements. If the bird lives two or three days, the body becomes very light in weight, the breast-bone is prominent, and emaciation becomes profound. Sometimes towards the end there is an opening and shutting of the beak, and the bird makes a crying or squeaking noise, as if complaining of his condition.

Very often the bird dies within two or three days after being in a state of semi-stupor. At times he seems to rally for a few days, and then weakness and somnolence returns, to end in death, after being ill from seven to fourteen days.

Sometimes the disease occurs by preference in the hens, some o which may be found dead on the nest.

Lesions.—The breast-bone is usually very prominent, showing that emaciation is profound; the muscles often have a boiled or dirty yellowish appearance; the liver and spleen are very much enlarged, but do not show any nodular lesions; the kidneys, heart, and, in fact, all the muscles of the body, often have a bile-stained appearance; the intestinal mucous membrane is occasionally congested.

Diagnosis.—This disease differs from bird fever by showing no nodular lesions in the spleen and liver. Sometimes it may be difficult to differentiate from it a given case of acute bird fever when the swollen spleen or the liver does not show any nodules. As a rule, however, there are several cases of bird fever at the same time to examine, and generally some of these at least show the characteristic nodular lesions. It also differs from pneumonia and other climatic diseases so commonly encountered in freshly caught or freshly imported birds by these latter complaints not being conveyable by cohabitation to acclimatised birds.

Treatment.—Scrupulous cleanliness and disinfection should be observed. The sick birds should be isolated. Some bird-fanciers give them tincture of aconite in the drinking-water. Hyposulphite of soda or aspirin may also be tried. Cages vacated by sick or dead birds should either be burnt or thoroughly cleansed and disinfected. Prevention should be carried out as in the case of bird fever.

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XXVI. PSITTACOSIS.

Synonyms.—Septic fever of the parrot and parrakeet; Parrot septicæmia; Sepsis; Typhus; Mycosis of parrots.

French: Septicémie des perruches; Mycose des perroquets; Typhus des perruches; Psittacose.

German: Psittacosis.

Definition.—A contagious septicæmia of parrots and parrakeets, characterised by fever, great weakness, stupor, and diarrhœa. It is considered to be caused by a bacillus of the paratyphoid, B. (human), or swine-fever type, and alleged to be transmissible from bird to man and from man to man.

History.—Although this disease has been for a very long time well known to bird-dealers and bird-fanciers, it did not attract much scientific attention until several outbreaks of an obscure pneumonic disease in man were attributed to infection derived from recently imported parrots and parrakeets which were sick or dying of some peculiar malady.

In 1879 Ritter observed in Switzerland, in the canton of Ulster, a series of cases of an atypical pneumonia in several families, following the introduction of parrots imported via Hamburg. It seemed as if the affected people had been infected by the transport cages rather than by the birds themselves. Three years later Ost of Berne, and then Wagner, in 1882, and again in 1886, reported similar cases. In 1880 Eberth studied a disease of the African grey parrot. In 1882 Wolff also investigated it, and considered it was possible for the disease to be transmitted to man. In 1892 an endemic of a contagious pneumonia having unusual characters occurred in Paris, which was ultimately attributed to diseased parrakeets imported from Buenos Ayres.

Nocard investigated this outbreak, and from the feathers of birds dead of this disease, and from portions of dried medulla obtained from the medullary canal of several intact human humeri, inseminated various culture media, and obtained an abundant culture of a particular microbe. In 1895 Palamidessa studied in Florence five cases of infectious pneumonia in the same family which appeared to have been derived from a parrakeet. He isolated from each patient a microbe having all the characters of the bacillus of Nocard. In 1896 Gilbert and Fournier isolated the same microbe from the heart blood of an infected woman. Leichtenstern considered at one time that he had observed this "parrot disease" in Cologne in 1898, and brought forward evidence to support its avian origin. But later on he went back on this view, because other centres of infection were in existence about the same period in Cologne, Crefeld, and Essen, which could not have had any relationship with parrots, and the bacillus of Nocard was not discovered in persons dead of the infectious pneumonia and in the suspected parrots.

In Rome several cases of psittacosis, terminating in death of the affected persons, were encountered in 1899, alleged to originate from diseased parrots imported from Paraguay.

It seems remarkable that, notwithstanding the enormous number of parrots and parrakeets coming every year into this country via Southampton, London, Liverpool, Hull, etc., from various parts of the globe, but few authentic cases have been observed by the medical profession in the human subject in Great Britain. The writer has for many years made numerous inquiries of those engaged in the foreign bird trade and of individual purchasers of parrots or parrakeets if they have ever come across any illness in people having birds suffering from this disease which they could attribute to the birds. In every case the answer has been in the negative.

The late Dr. Karl Russ, the greatest aviculturist the world has ever known, who had a very great opportunity of seeing and observing this as well as other avian maladies, is absolutely silent on the question of its transmission to man. The late Dr. W. T. Greene, who had kept the majority of known species of the *psittideæ*, and who wrote upon their habits and diseases, is also silent on this point. A great number of other medical men who go in for aviculture, and are therefore often brought in contact with newly-purchased parrots which have fallen ill and mostly soon die of this malady, have never observed a similar disease in mankind to which they could attribute transmission from the bird. Friedberger and Fröhner observe that a proof of the alleged association between this parrot disease and endemic pulmonary disease in man has no more been established than one of the earlier hypotheses of the origin of human diphtheria from avian diphtheria.

Etiology.—Although contagion, either direct or indirect, plays a part in the dissemination and maintenance of this disease, it is of lesser importance than the conditions under which the affection generated. It usually arises in birds during the period from the time they are first caged to the time they are landed in Europe. When one considers that freshly-caught birds are crowded so thickly together that they can scarcely move in boxes or bamboo cages, their food flung on to the floor of the cage, which is soon covered with fæces, accumulating and decomposing for days or weeks together, and from which the birds have to pick it; that they are often kept short of water, and maintained for a rather long period in a very high temperature, and then somewhat suddenly submitted to a low European temperature, especially in inclement weather, often after a boisterous passage on board ship, there is no wonder that the system becomes weakened, and the constant passage of saprophytic germs enables these to become virulent, and thus set up disease in creatures that have hitherto led most sanitary lives.

Every tissue and discharge of the bird is virulent. A healthy parrakeet placed in an infected cage, or a clean cage containing infected feathers, soon becomes ill, and dies within three weeks from the time it was first placed in the contaminated cage. Contaminated food and water, utensils, and hands, may also transmit the disease. Mice may contaminate the food by their feet being soiled during their wanderings in infected cages.

Bacteriology.—The incriminated organism is included in Lignières' group of Salmonelloses, of which the so-called bacillus of swine fever is taken as the type. It is a short and fairly thick bacillus, having rounded ends, aërobic and anaërobic, and extremely mobile. It does not take Gram. Cultures are obtained in all media having a neutral or slightly alkaline reaction. On peptonised broth at 37° C. the culture forms rapidly and abundantly. The medium is uniformly turbid, and gives a cloudiness which the least shake breaks up and causes to fall to the bottom of the tube. On gelatine inclined, it gives at first a shining, transparent, bluish, iridescent trail, which soon becomes thick, opaque, creamy, and porcellaneous white. The medium is not liquefied. On agar its growth is very rapid, and forms a thick, whitish, opaque layer, without special characters. On potato the culture is similar in appearance to that of the *Bacillus coli communis*. It does not ferment lactose nor coagulate milk.

By inoculation it is pathogenic for the parrot, parrakeet, pigeon, fowl, mouse, rabbit, guinea-pig, etc.

The bacillus is found in the blood, bone-marrow, feathers, and other parts of the sick bird. In man it has been found in a pure state in the heart-blood. It has never been encountered in the sputum, urine, blood, and serosity, in living subjects.

Achard and Bensaude have isolated Nocard's bacillus in a case of sterno-clavicular abscess, and in another of purulent cystitis. In neither instance was it connected with the parrot disease. Probably the isolated organism was only the *Bacillus paratyphosis*, B. (human) type.

The antityphoid serum only slightly agglutinates it; the minimum quantity of a serum capable of agglutinating the *Bacillus typhosus* is without any action on Nocard's bacillus. The serum of persons affected with alleged psittacosis agglutinates very irregularly Nocard's bacillus.

Gilbert and Fournier have isolated an analogous microbe from the intestinal contents of parrakeets having all the appearance of good health.

Mode of Contagion.—It may be transmitted by infected cages, contaminated food or water, feeding utensils, feathers, and sick birds. As the bacillus resists desiccation, it may also be conveyed by the atmosphere; but ingestion seems to be the usual way of its gaining an entrance to the system.

It is alleged that it is conveyed to man by feeding infected birds out of one's mouth, by contamination of the hands from handling sick birds, infected cages, or soiled feathers, and that it may be conveyed from man to man.

Inoculation of the cultures obtained on various media under the skin or into the muscle gives varying results. Sometimes death occurs in two or three days, but usually after a week or two, when the animal becomes very much emaciated, accompanied by a profuse diarrhœa. Recovery rarely takes place, and then only after a lengthy period of convalescence. Ingestion and inhalation produce similar but slower effects. Intratracheal, intraperitoneal, and intravenous injection kills the parrakeet, pigeon, fowl, mouse, rabbit, and guinea-pig, in less than forty-eight hours.

Period of Incubation.—By natural infection or contagion symptoms of the disease manifest themselves within three or four days to as many weeks. Occasionally they are delayed beyond this period. Very often the disease declares itself a few days after the arrival of the birds in this country.

Mortality.—This is rarely under 50 per cent. of the total. Frequently quite 90 to 95 per cent. succumb; some have computed it at 999 per 1,000. The majority of birds die within three to five days after the first signs of illness.

Symptoms.—The bird shivers, loses its appetite, seems dull, has a frothy, greenish diarrhœa, which sometimes contains blood, becomes drowsy, disinclined to move, and has great thirst. After a short period the wings droop, the feathers stand open and have lost their gloss, the eyes are closed, the bird hides his head under his wing or in the feathers on his shoulders, and when made to move shows signs of great weakness. Towards the end convulsions appear, and are soon followed by death. If the bird should happen to live long enough, there is a discharge of clear mucus or yellowish muco-pus from the nostrils, sneezing or snuffling, a watery or purulent discharge from the eyelids, cough, and difficult breathing, which is sometimes of a panting kind. Emaciation, lightness in weight, and a prominent breast-bone, are usually present. Occasionally vomiting may be observed. As a rule, there is a disinclination or inability to sit on the perch, the bird preferring the floor of the cage, and especially one corner of it.

Lesions.—The muscles are often striated, and show greyish spots. The medulla of the bone is congested. The lungs often present small atelectasic patches, or yellowish patches of pneumonia, or reddish areas of congestion, or even consolidation. The air-sacs, especially the thoracic pair, are sometimes lined on their interior with a yellowish or greenish-yellow layer of an adherent, more or less soft fibrinóus membrane, and occasionally contain a small quantity of a greenish seropurulent fluid. The myocardium is often stained a dark red colour; the endocardium and pericardium show some ecchymoses. The abdominal organs are usually intensely congested, and the peritoneum ecchymosed. The liver may contain small grey, greyish-white, or yellowish nodules, varying from the size of mere points up to a fig-seed, hemp-seed, or something larger. Similar lesions may be found in the spleen and kidneys, and rarely in the lung. As a rule, the spleen, like the adrenal glands, is enlarged, soft, and hæmorrhagic. The intestine shows some catarrh, often some superficial ulceration in the small intestine, is dotted with greyish, irregular spots, and mostly presents, in lieu of these lesions, general congestion.

Nocard's bacillus is found in abundance in the heart blood, splenic pulp, intestinal contents, and bone-marrow.

Diagnosis.—This disease is likely to be confounded with the parrot form of avian plague, which is due to an invisible organism capable of passing through the Berkefeld or Chamberland F and even B bougies or filters (*vide* Avian Plague, p. 484). Tuberculosis in parrots generally occurs in acclimatised or aged birds, and rarely runs an acute course, as does this disease. In man serum diagnosis should be adopted.

Prognosis.—Death is usually the result of this affection. If the bird survive the eighth or ninth day, some hope may be entertained, especially if there should be some appetite.

Treatment.—The usual means of disinfection and isolation should be strictly carried out. The bird should be kept in a room having the temperature of from 90° to 100° F. maintained day and night. Aspirin or cyllin should be given in the drinking-water. Bread-and-milk, honey, boiled maize, sponge-cake soaked in sherry, banana, etc., should be offered from time to time. If the bird should show improvement, the aspirin may be replaced by aromatic sulphuric acid and quinine sulphate.

Transmission to Man.—According to Osler, a disease in man characterised by an atypical pneumonia, great weakness, and depression, and signs of a profound infection, has been ascribed to contagion from birds, particularly parrots, in Germany, France, and Italy. These have usually been house epidemics, with a very high rate of mortality. A few cases have been reported in England, and Vickery of Boston has reported three probable cases. Dujardin-Beaumetz and Dubief consider that itinerant bird-dealers, who usually travel about to sell infected birds, and bird-shops should be under sanitary control, and the bird trade treated as an unhealthy occupation. Assuming that the disease is transmissible to man, bird fanciers should be warned of the danger they run by feeding diseased birds from the mouth, and of becoming contaminated by the discharges or by handling cages, etc. According to Dupuy, the mortality from this disease in Paris from 1892 to 1897 was twenty-four out of seventy cases, or on an average 34·28 per cent. He believes, however, that this total is not high enough, and that a number of cases returned as influenzal pneumonia really belong to psittacosis.

A proposition for the regulation of the transport of birds was discussed by the Veterinary Hygiene Section of the Madrid (1898) Congress of Hygiene and Demography.

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XXVII. KOCH'S MOUSE SEPTICÆMIA.

This disease is of some importance in consequence of its etiological relationship with swine erysipelas, and its presence in mice may be a possible explanation of the mysterious outbreaks of swine erysipelas that sometimes occur.

History.—In the course of his study on wound infection in 1878, Koch found that, when mice were inoculated on the skin with putrid fluids or the very impure water of the Panke, a fatal septicæmia was set up and caused by a special bacillus. The morphological, biological, and physiological characters of this bacillus were in 1880-81 investigated by Gaffky. It was afterwards studied by Preisz, Lorenz, Löffler, Prettner, Overbeck, and numerous other investigators.

Etiology.—The disease may arise spontaneously in house as well as field mice, especially when confined in badly-kept cages by their captors. The bacillus is found in the blood and putrid flesh of mice, in their fæces, and in the mud of certain rivers. The disease may also occur in hamsters. Löffler considers it is possible that mice may contain a virus inoffensive to themselves, and, dying from whatever cause, may be eaten by pigs, which afterwards become affected with swine erysipelas.

Bacteriology.—The morphological appearances and staining properties of the bacillus of mouse septicæmia (*Bacterium murisepticum*) are in common with those of the swine erysipelas bacillus. The cultures of both bear a strong resemblance to one another. Still, the cultures of the mouse septicæmia organism in gelatine give a more cloudy growth, and do not show so clearly the radiate prolongations. When inoculated into other mice, it is not so rapidly fatal as the swine erysipelas organism is to them. It is not pathogenic to the pigeon or rabbit; but when its virulence is reinforced by a series of passages through the mouse, a virus is obtained which, when intravenously inoculated, will give the pigeon a fatal disease. The serum of immunised animals against swine erysipelas agglutinates the bacillus of mouse septicæmia. This fact alone suffices to establish in a definite manner the identity of the two bacilli. Pigs which develop a local reaction to the virus of mouse septicæmia are rendered refractory to the bacillus of swine erysipelas.

Symptoms.—The affected mice have a heaped-up or lumpy appearance; the coat is dull and open; the eyelids, which are closed, discharge tears, and ultimately an agglutinating material which glues them together; the breathing is quickened or of the panting kind; and death takes place, after a more or less profound state of sleepiness or of coma, within two or three days.

Lesions.—The intestines are congested. The liver and kidneys are enlarged and congested. The spleen is swollen, soft, and brownish-red. The blood, especially that in the capillaries, contains a great number of bacilli, which are enclosed in a mass in the white corpuscles.

Diagnosis.—This disease resembles Löffler's mouse typhus by its course, symptoms, and lesions. This latter disease, however, is due to a bacillus (*Bacillus typhi murium*) which belongs to the paratyphoid B. type, and therefore resembles that of psittacosis and of swine fever, and the *B. enteritidis* (*vide* Löffler's Mouse Typhus).

Public Health.—It is not known if this mouse septicæmia is conveyable to man, as is the case with swine erysipelas.

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XXVIII. LÖFFLER'S MOUSE TYPHUS.

Although this disease is not likely to set up any similar disease in the domesticated animals, a knowledge of its pathology should be of interest to the veterinary surgeon, in consequence of the fact that it supplies the microbe for the cultivation of virus for the destruction of the common grey mouse (*Mus musculus*), the field-vole (*Mus arvicola*), the field-mouse (*Mus agrarius*), rats, etc.

History.—This disease was first described by Löffler, who encountered it as an enzoötic in his laboratory mice. It was afterwards investigated by H. Laser, who found it amongst field-mice kept in the laboratory. Merechowsky and Issatchenko described similar epizoötics, the first in spermophilæ, and the second in the white rat. Danysx also observed an epizoötic in the field-vole and wood-mouse.

Bacteriology.—Boutroff has shown that Löffler's mouse typhus bacillus (*B. typhi murium*) should be included in the paratyphoid human B. group—the Salmonella of Lignières), to which the *B. enteritidis* or *Gârtneri*, the bacillus of swine fever, and the bacillus of psittacosis, belong. The observations of Trommsdorf, confirmed by Mayer and Bonhoff, and Shibayama, show that Löffler's bacillus is capable of infecting man. Similar observations have been made in Great Britain.

The *B. typhi murium* manifests all the morphological characters of the paratyphoid B. type or of the group of Salmonella. Field and common mice contract the disease by inoculation or by ingestion. It is equally virulent for tame mice. The guinea-pig, rabbit, grey rat, and birds, are less sensible to inoculation, and cannot be infected by ingestion. The dog and cat are refractory.

Economical Utility.—The *B. typhi murium* enters into the domain of agricultural industry as well as of human and of veterinary hygiene, for it is used for the purpose of manufacturing virus for the destruction of common and field mice and rats, which have been shown to be not only the carriers, but also the sufferers, of bubonic plague, swine erysipelas, and other maladies, as well as doing a very great amount of damage to agricultural crops, corn-stacks, farm and other buildings, and to young poultry. *Danysx virus* is grown in tubes of gelatine. But as Danysx found that the grey rat was not very susceptible, and that the bacillus lost its virulence after a few passages through this creature, he overcame this immunity by cultivating the bacillus in hermetically sealed tubes, and afterwards by a series in passages in collodion sacs included in the peritoneum of the rat; then inoculating the mouse with this prepared bacillus, and terminating the process after passing it through rats of gradually increasing ages. By such means he was able to obtain a virulent bacillus for the grey rat (*Mus decumanus*), the black rat, and the white rat (*Mus ratus*). These cultures are deposited on cubes of very stale bread, which are afterwards placed in suitable places within the reach of these vermin, in which it produces an enzoötic, and thus ridding sewers, houses, and other buildings, of the majority of rats. As it is difficult to completely remove all the rats, because after three or four passages the virus loses its virulence, it is advisable to renew the distribution of the treated bread every ten days or fortnight, preferably when the rats are young and more easily influenced—namely, between April and June, and again from September to December.

G. Neumann of Aalborg uses a bacillus originally derived from the urine of a two-year-old child suffering from cystitis. With this Ratin bacillus several practical tests were carried out, notably in Halle, but from the official report it proved unreliable in certain sharply-defined areas. Dunbar used a bacterium obtained from certain rats that died in the laboratory, but, as the animals on which he tested it manifested similar symptoms to those of bubonic plague, he relinquished the use of his virus for the destruction of rats.

According to Trautmann and Xylander, the general appearances of Löffler's, Dunbar's, and Neumann's Ratin bacilli on the various culture media indicate they all belong to the large group of so-called "ptomaine producers," and that by their power of agglutination they seem to be identical with Gärtner's *B. enteritidis*.

Besides these mouse and rat viruses, there are the Liverpool rat and mouse virus, and another, but in powder form. This last has been several times tried by the writer for the eradication of mice, but in his hands it absolutely failed to destroy the vermin, which throve on it and got quite fat. The results were quite different with the Danysx mouse virus; in every instance in which the writer has used it, if it did not destroy all the mice, it diminished their numbers.

Rat and mouse viruses to be effectual should be used systematically, and after regular intervals covering the outline of the premises simultaneously.

So far as the writer's experience goes, the Danysx virus is quite harmless to birds and to the domestic animals. Mice dead of the disease given to various animals did not cause any disturbance in any one of them.

Palmirsky used cultures of the *Vibrio Metchnikovi* sprinkled on fodder to destroy field-voles in Southern Russia.

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XXIX. SLEEPING DISEASE OF FOWLS.

French : Maladie du Sommeil.
German : Schlafsucht der Hühnes.

History.—Nocard studied a disease of fowls which he, at the time, considered was due to a very small bacillus, especially abundant in the spleen, and gave it the name of "maladie du sommeil." It manifested itself as a chronic form of avian cholera, and was chiefly characterised by stupor and an almost uninterrupted sleepiness. The affected birds died at the end of eight to fifteen days. After further lengthy researches, he came to the conclusion it was a milder form of avian cholera, complicated by various secondary microbial infections.

In 1904 and 1905 Dammann and Manegold, of the Hanover Veterinary School, studied a disease in fowls having similar clinical appearances, and appearing in Westphalia. They arrived at the conclusion that it was due to a special streptococcus, which they named *Streptococcus capsulatus gallinarum.*

Bacteriology.—The micro-organism isolated by Dammann and Manegold from the heart blood appeared in the form of a long streptococcus. In liquid media it appeared as a streptococcus having a hundred or more articles, measuring in diameter about 0·5 μ. It took Gram, and showed, after coloration with Klett's modified method, a pale capsule. It is aërobic as well as anaërobic. It may be cultivated in all the usual media. By inoculation it is pathogenic for the fowl, which it kills about the seventh day, after producing symptoms of somnolence. It is also pathogenic for the pigeon, rabbit, white and grey mouse, and lamb.

Symptoms.—The fowls (about 100) died at the rate of about three a week. They appeared dull and stupid, their eyes were much swollen and closed, the comb was pale and drooped, lameness appeared in one leg, and the disease lasted about eight days. The mucous membranes of the head were affected with catarrh ; this was accompanied by a diarrhœa, which did not seem to affect the birds in some cases when the appetite was good. All traces of disturbance disappeared in a few days. Some died after being emaciated for a little time, and others after refusing all food and appearing somnolent.

Lesions.—The post-mortem examination may show adhesions between the pericardium and epicardium by means of fibrinous strings and a slight enlargement of the spleen. In other cases there may be pericardial and peritoneal exudations. Others show lesions common to the hæmorrhagic septicæmias.

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XXX. STREPTOCOCCIC SEPTICÆMIA IN POULTRY.

Synonym.—Apoplectiform septicæmia in the fowl.

Norgaärd studied this affection in 1901. It caused great mortality. The symptoms appeared to be identical with those of avian cholera or avian plague. The disease was some weeks clearing off nearly all the fowls, which would suddenly stop feeding, or would drop from their perches and die without a struggle.

Bacteriology.—The streptococcus was found in pure cultures in all the diseased centres except in the intestinal contents.

The organism was composed of short chains, having two to eight or more articles of 0·6 to 0·8 μ in diameter. Involution forms were common, and fission-forming tetrads were observed by Norgaärd. It stained with all the usual aniline dyes, and also by the Gram and Gram-Weigert method. It was non-motile, aërobic, and facultative anaërobic. It readily grew in neutral, slightly acid or alkaline, but not strongly acid, solid and liquid media at 98·6° F., and slower at the room temperature. In alkaline peptonized bouillon in twenty-four hours it formed threads and bulbs on the sides and bottom of the tube, leaving the liquid clear. On agar there were formed small shining, greyish colonies, 1·5 millimetres in diameter, with a brownish centre and bluish circumference. With all sugars it produced acid, but no gas. It did not coagulate milk. On gelatine colonies were formed in four days, but there was no liquefaction.

Young chickens, then pullets, and afterwards laying hens, were most susceptible to the ingestion of cultures as well as of the chopped-up viscera of rabbits dead of the disease. Such experiments proved that death occurred from three to thirteen days after ingestion of the virulent products. Intravenous inoculation killed the birds in two or three days. Intramuscular injection of large doses did not always produce the disease. Intravenous injection killed the pigeon, which was refractory to intramuscular inoculation and ingestion infection. Mice, rabbits, ducks, and dogs were susceptible to the experimental methods of infection. Guinea-pigs and sheep were refractory.

The lesions were similar to those encountered in avian cholera. The liver and spleen show centres of coagulation necrosis if death was delayed for twenty-four hours or more. All the lesions contained pure cultures of the streptococcus.

It was possible to immunize the fowl against this infection. An injection into the wing vein of 0·5 to 2·5 c.c. of bouillon culture, sterilised by heat, was used.

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XXXI. AVIAN PLAGUE.

Synonyms.—Fowl plague; Bird plague; Bird pest; Exudative typhus of birds; Infectious peritonitis of fowls; Brunswick bird plague; New bird plague.

French: Peste aviare; Peste des oiseaux.

German: Hühnerseuche; Putenseuche; Vogelpest; Geflügelseuche; Kyanolophiea gallinarum.

Italian: Peste aviaria; Tifo essudativo dei gallinacei; Epizoozia tifoide dell pollame.

Definition.—An acute infectious disease of birds, running a short course, generally ending in death, and due to an ultravisible virus, which is not arrested when passed through Berkefeld or Chamberland F and even B filters. It is similar in its manifestations, course, and pathological anatomy, to avian cholera.

Species Affected.—Fowls are chiefly attacked, but also turkeys, pheasants, geese, ducks, pea-fowls, guinea-fowls, pigeons, parrots, blackbirds, thrushes, nightingales, robins, starlings, sparrows, canaries, etc.—in fact, no species of bird seems immune to some one or other of its various forms or types of virulence.

History.—In 1880 Rivolta and Delprato, two Italian veterinary surgeons, distinguished avian cholera from exudative typhus purely on clinical grounds. In 1894 Perroncito drew attention to an epizoötic in fowls in Novara, which had not the usual characters of avian cholera. In 1899 Mazza observed in Piedmont an epizoötic characterised by a brownish discoloration of the comb and by exudation into the serous cavities, without the heart blood giving any culture; but from the visceral parenchyma this authority isolated a micro-organism which he thought might be the causal organism. Foá, Cesario Demel, and Abba, in Piedmont in 1899, Belfanti and Zenoni in Lombardy in 1899, Brusaferro in Parma, and Reggio in 1900, studied the disease, and severally believed they had isolated the causal organism, although some came to the conclusion that it did not resemble avian cholera, whilst others endeavoured to maintain the organisms they isolated were the cause of the malady. In 1900 Centanni and his pupil Savonuzzi investigated the disease occurring among fowls in Ferrara, and in 1901 declared it was not avian cholera, nor due to a virus detectable by the usual methods then in vogue. Shortly after their first communication they made known that the avian plague (*peste aviaria*) could be transmitted by the filtrate obtained from a mixture of blood and normal saline solution passed through a Berkefeld filter. Soon after this announcement fresh studies were commenced in Germany, where the avian plague declared itself in February, 1901. The result of Jess's researches on an epizoötic unlike ordinary fowl cholera led him to consider the disease he had studied was due to the bacterium of avian cholera acting in symbiosis with another agent. Scheurlen and Buhl studied the disease in Würtemberg under the name of "epizoötic peritonitis of fowls." They could not find any microbial agent in the blood, parenchymæ, or the peritoneal exudate. The exudate, however, when inoculated under the skin, killed the fowl, but not the rabbit and guinea-pig. Greve found the same disease in Oldenburg. He could not induce the disease in the pigeon by inoculating the blood under the skin. The heart blood gave a liquefying bacillus, which did not kill the fowl. He came to the same conclusion as Jess: the disease was a mixed infection. Lüpke the same year (1901) made known the result of his researches carried out at the Pathological Institute of Stuttgart, which included 130 post-mortem examinations. He concluded that the Brunswick disease differed from avian cholera by the lesions and by no specific organism being discovered, and whilst it was easy to pass the disease from fowl to fowl, the rabbit, mouse, and pigeon were refractory. These German authorities were evidently ignorant of the work of Centanni and Savonuzzi.

In 1901 Maggiora and Valenti made an exhaustive study of the disease in Italy, and whilst confirming the conclusions of their compatriots, Centanni and Savonuzzi, affirmed that they were unable to infect ducks or pigeons. They also studied an epizoötic occurring in blackbirds and other species of the family Turdidæ, starlings, and other birds at liberty in a wild state during September, 1901, in the province of Modena, almost coincidentally with an outbreak in poultry. Lode and Grüber in the Tyrol also confirmed Centanni and Savonuzzi's observations. Ostertag and Wolffhügel studied the disease in Germany in 1903, and came to the conclusion that the specific organism could not be detected by modern methods of investigation up to that time in general use, and that it was contained in the blood, fæces, and nasal mucus of diseased fowls, and is spread by these vehicles, and could be destroyed by a temperature of 70° C. Kleine and Möller have shown by their researches that the disease closely resembles rabies, by the nervous centres of infected geese being always infective; and Rosenthal (1905) has pointed out the presence of corpuscles resembling those of Negri, also observed by Högyes, Krauss and Clermont in the experimental rabies of birds. Marcone, in 1902, in Italy, studied the disease in the pheasant, and Stazzi, in 1906, in parrots.

Greve, Jöst, Depperich, Künnemann, Bugge, Krauss and Schiffmann, and many others, have made important studies of the nature of this malady. Freese, of the Hygienic Institute at the Veterinary College, Hanover, made, in 1908, a very important communication on the differential symptomatology and pathological anatomy of avian cholera and avian plague, and concluded that the symptoms and the post-mortem appearances alone are not sufficient to settle the diagnosis.

Marchoux, in 1908, obtained a culture *in vitro*. In 1908 McFadyean, in a concise and lucid paper on the Ultravisible Viruses, gave a good description of the nature of the virus of this malady.

Epizoötiology.—There is no evidence that this malady is an old one, but it is very probable it was confounded in former days with avian cholera and other septicæmic maladies of birds. It is evident that it has existed in the greater portion of Italy since 1880. It is known in Novara, Ferrara, Padua, Lombardy, Venice, Reggio, and Pisa. It has been encountered in the Tyrol. In Germany it was supposed to have been introduced by a poultry-show held in Brunswick in February, 1901. From this show it was scattered to Würtemberg, Oldenburg, Hesse, and extensively in Prussia. It was also partly imported direct from Italy. It has now been demonstrated to be present in Italy, Germany, Belgium, and France. It has been suggested that it was introduced from the Soudan and Egypt. Probably it occurs in other countries that traffic largely in foreign poultry and birds, or hold large international poultry-shows, but is not yet recognised. It is quite possible for it to be introduced into a country by birds on their migrations.

Period of Incubation.—The incubation period in natural infection or contagion varies from three to five days. It may, however, be as short as one day or as long as a week. In inoculation experiments it may only be a few hours.

Etiology and Pathogenesis.—This disease is due to an ultravisible virus, the active agent of which is, according to Schaudinn, probably a protozoön, as all the other filtrable viruses are. In favour of this view, advanced by Lode and Kleine, and held by many authorities, Landsteiner puts forward the action of saponine, which is harmless to bacteria, but destroys the virus of avian plague, like all the other filtrable viruses, after a few minutes' contact.

The virus is found in the blood, bile, intestinal contents, mucous secretions, central nervous system, and other parts of the body. The blood of birds dying early is very virulent, and, according to Landsteiner, the virus is contained in the red corpuscles, but very little in the serum. The pericardial and peritoneal effusions are also very virulent.

Blood mixed with from 40 to 60 parts of normal saline solution, and passed through the Berkefeld or Chamberland F and even B filters, gives a filtrate that is very virulent. Even blood diluted in the proportion of 1 to 125,000,000 gives a filtrate which, when 4 c.c. is injected into a fowl, kills it in forty hours. When the blood was passed through a close-grained Chamberland K filter, under a pressure of not more than 1½ atmospheres, it was, it is said, not infective. In the goose the virus confines itself chiefly to the central nervous system, where it may remain active after the virus has disappeared from the blood and the rest of the system.

Marchoux has been successful in cultivating the virus *in vitro.* He used a trace of virulent blood to inseminate a tube of peptonised agar (1 per cent.), sugar (2 per cent.), on which is poured 10 c.c. of defibrinated fowl's blood. This was maintained at a temperature of 37° C. The culture did not develop in the bulk of blood, but in a zone bordering on the agar. After ten successive passages, $\frac{1}{5}$ c.c. of a culture of three days at 37° C. killed a fowl in two days. This could not be attributed to the dilution of the virus. The least impurity stopped its development.

The virus is not readily destroyed by desiccation, and in this state it may remain virulent for at least three or four weeks or even months. It is destroyed by exposure for a few minutes to a temperature of 65° to 70° C. Blood kept in a hermetically sealed Pasteur pipette in a cold dark place is still virulent at the end of three months. When exposed to the air and diffuse sunlight, it was virulent at the end of a fortnight, but it was innocuous after exposure to the direct hot sun. Virus enclosed in a collodion sac and placed in the peritoneum of the rabbit loses its activity after four days. Filtered virus loses its virulence after the first week. By the addition of sugar and peptone to virulent blood the virulence is conserved at 37° C. and resisted at 51° C., whereas all the control tubes have absolutely lost their activity. Pieces of infective parenchymæ preserved in glycerine retain their activity for four or five months, but when the virus is mixed with glycerine it loses its virulence within about three weeks. According to Landsteiner, saponine in a 1 per cent. solution destroys its activity after a few minutes' contact. It is unknown if the bile has any bactericidal power.

Putrefaction, or the bacteria of putrefaction, and the coli and bacterium of avian cholera, destroy its virulence. A thick limewash freshly prepared from lime slaked at the time of using, perchloride of mercury (1 in 1,000) solution, 5 per cent. solutions of carbolic acid, creolin, lysol, or hydrochloric acid, and a saturated solution of salicylic acid, destroy its activity in a few minutes.

Mode of Contagion.—The disease is transmitted by the medium of the nasal, oral, and intestinal discharges. Blood and fæces of infected birds ingested by healthy birds give the disease and kill in three days to a week. Possibly the malady may be inoculated by means of some of the many species of epizoa usually living on birds. According to Maggiora and Valenti, it may be conveyed by coitus. Sparrows and other birds, especially the predatory species, which may contract the affection naturally, may be the means of carrying disease from one place to another. Rats and mice, although not susceptible, are possible carriers of the virus by means of their soiled feet contaminating the bird's food. Poultry shows and markets, bird-shops, contaminated cages, aviaries, fowl-houses, hampers, eggs, contaminated food, etc., may be the medium of infection. Maggiora and Valenti consider that, outside direct or indirect contagion, the smaller birds at liberty may contract the disease through insects and worms on which they feed. Marcone affirms that the disease is not transmitted through the air, but by the dejections and by contaminated water and food. In an outbreak occurring in pheasants in the Capodimonte in 1902, and studied by him, he considered it was introduced by small birds. Out of about 800 pheasants scarcely 50 survived.

Marchoux failed to infect fowls by feeding them on contaminated food or water, or on fæcal matter, or by cohabitation. He was more successful with the nasal discharges, which are, however, generally absent. He considers it is more probable that the disease is transmitted by an intermediary bearer, possibly a special acarus.

The disease generally declares itself towards the commencement of spring, gradually declines in the summer, and remains dormant during the winter. Why it should respect certain birds at one time even in the same premises, and attack the whole at another time is inexplicable. In certain places in an infected locality some poultry-yards are attacked, whilst others remain free.

Transmission from one Avian Species to Another.—A wide difference of opinion prevails between certain authorities as to whether the disease is conveyed by natural means from one species to another species of birds. Some have witnessed it in fowls, ducks, and geese, at the same time; others in fowls first, then geese; others in the fowl, sparrow, turkey, pheasant, guinea-fowl, etc. Maggiora and Valenti failed to experimentally transmit it from the Turdidæ and other species of small birds to the fowl, notwithstanding the disease having appeared in wild birds soon after an outbreak in poultry. In these respects avian plague, acting in this peculiar manner, by having at one time a preference for one species and then another, resembles avian cholera.

Parrots are more susceptible at times than other birds, and *vice versâ.* Young birds, especially goslings and squeakers, or squabs, are more susceptible than aged ones. Pigeons are not very susceptible to fowl plague, whereas the pea-fowl and pheasant are equally as susceptible as the fowl.

Symptoms.—As in fowl cholera, the affected birds may die quite suddenly, without having manifested any symptoms to attract attention. They may even die on the nest, or only after showing uneasiness, and then rapidly forming themselves into a ball.

In the majority of instances, however, the sick bird isolates itself from its companions, often hiding itself under some shady or dark obstacle or in a corner. It is dull, mopes, squats on its breast, does not care to move, places its head under its wing or in the shoulder-feathers, thus forming itself into a ball, with the feathers standing out and open. The wings and tail droop, the eyelids are closed, food is refused, and the bird staggers or falls if made to move on. The breathing is rapid or difficult and deep. The comb may be purple, mottled, or streaked with dark brownish spots ; later on it may become quite brown, and covered with bran-like scales. Although diarrhœa is not usually present, it may occasionally occur in the form of liquid fæces, which may vary in tint towards the end. The beak is often opened and shut convulsively. The temperature, which was at first very high (110° to 112° F.), gradually becomes lowered (103° to 100° F.) below normal (106·5° F.). Coma sets in, and death takes place within twenty-four hours from the commencement of the symptoms.

In many cases the course of the malady is not so rapid, yet the birds refuse food and do not drink. They seem mopy, and gradually become worse, and it is with difficulty that they can be made to move, when the body sways. The head is shaky, and the sight seems blunted or lost. The hen may sit on her nest, and yet appear not much amiss until handled. There may be a mucous discharge from the mouth or nostrils, difficult or noisy breathing, and sometimes a dirty, stringy exudate or mucus on the lining membrane of the nasal channels and pharynx. The fæces are either normal in colour and consistency, or pasty, of a yellowish-green tint, or thin, watery, and greenish. The comb may become hot, swollen, and at the base purplish, and eventually deeper in colour, until death supervenes during a deep comatose state after the temperature has become subnormal. The course of such cases may extend from three to five days, or even nine or ten days. In the smaller species of birds the period of illness lasts longer than it does in the larger species.

In some cases there may be swallowing movements, labyrinthine vertigo, hemiplegia, hiccough, or convulsions. Such phenomena are more commonly seen in geese, parrots, pigeons, and smaller birds, than in the fowl. (In small birds convulsions are seen in many of their infections, contagious as well as autogenous.)

In the goose there is often a stupid, semi-intoxicated immobility, with a great disinclination to move when an attempt is made to stimulate it. The conjunctival membrane may be deeply injected, and the fæces semi-fluid and whitish in colour. In the smaller flying birds there seems to be an inability to fly, and, when confined in a cage, to keep on the perch, preference being given to one corner of the floor of the cage, where the bird seems to remain immobile and indifferent to its fate.

Lesions.—In many cases there may be no visible changes observed on post-mortem examination. In the majority of instances some or other of the lesions resemble those seen in avian cholera. Depperich, after an extended study, gives the average of the principal lesions as follows:

1. Catarrhal changes in the upper air-passages and the fauces, 95 per cent.
2. (*a*) Acute gastro-enteritis, with or without hyperæmia, 85 per cent.; (*b*) acute gastro-enteritis, with hæmorrhages, 40 per cent.
3. Diseases of the liver, 84 per cent.
4. Diseases of the kidneys, 80 per cent.
5. Changes in the peritoneal cavity: (*a*) Exudative peritonitis, 33 per cent.; (*b*) subperitoneal hæmorrhages, 20 per cent.
6. Disease of the heart: (*a*) Cloudiness of the muscular fibres, 66 per cent.; (*b*) pericardial petechiæ, 22 per cent.; (*c*) subendocardial hæmorrhages, 1·4 per cent.; (*d*) pericarditis, 4 per cent.
7. Pneumonia, 3·6 per cent.

In a few cases the subcutaneous areolar tissue in the region of the breast, neck, and head, is infiltrated and œdematous. The muscles may be pallid, and contain ecchymotic spots. In a great number of instances there are minute or small superficial or deep hæmorrhages in the proventriculus, especially in the smooth mucous membrane just before it passes into the gizzard, and not uncommonly between the conical prominences, or rarely under these prominences on the mucous surface. Minute hæmorrhages may be found in the fatty tissue surrounding the gizzard and the intestine at its origin; larger ill-defined hæmorrhages are also found beneath the cuticle of the gizzard. The duodenal portion of the *small intestine* may be the seat of catarrh, intense inflammation, or hæmorrhages. More rarely the cæca and large intestine may be inflamed. In a great many instances the *peritoneal cavity* contains a serous or sero-fibrinous exudate. According to Künnemann, it is quite exceptional to find no exudate, but Freese only found it in about one-third of his observations. The *kidneys* are usually swollen, enlarged, and prominent, often of a greyish-brown colour, and turbid, or, especially in acute cases, dark brown and moist on the surface of a section. In the hen in which the *ovary* is developed there is a pronounced injection of the vessels in the serosa of the Graafian vesicles. Not rarely small hæmorrhages are seen immediately under the serosa covering the yolk vesicle. As in other diseases, the yolk may break down and escape into the peritoneal cavity, and form a yellowish or chylous-looking material, which should not be looked upon as an exudate or anything peculiar to avian plague. The *spleen* is sometimes slightly swollen in acute cases; Marcone, of Pisa, in the pheasant found this organ swollen, red and soft; also numerous hæmorrhagic points on the *pancreas*. The *liver* is occasionally swollen or congested when the disease is acute. The *heart blood* may be firmly clotted, and of a dark red colour, or it may not clot, or the tendency to clot is less than usual, or at times it may clot in masses. The *pericardial sac* may contain a serous or sero-fibrinous exudate, totally or partially clotting. Other lesions may resemble those observed in a great number of cases of avian cholera or of other septicæmias.

Differential Diagnosis.—As a great number of the septicæmic maladies of birds assume similar clinical manifestations, and often analogous pathological lesions, the diagnosis of avian plague without a microscopical examination of the blood and an experimental testing of the blood on various species offers many obstacles in the way of arriving at a correct solution.

As a rule, the absence of the ovoid bacillus in the blood and scrapings of the parenchymæ, in an epizoötic running a very rapid course, generally ending in death, of birds manifesting a "huddled-up" appearance, coma, and without diarrhœa in the majority attacked, should lead one to suspect avian plague. If a series of pigeons be inoculated with some of the heart blood, and the majority do not die, one should have further recourse to the rabbit, which is refractory to avian plague, but very susceptible to the avian cholera bacillus. More than one of the same species of the testing animal should be utilised, as sometimes a single animal may be immune, but the others very susceptible to the virus inoculated. In the goose the virus should be sought for in the central nervous system, as it is usually retained there long after it has disappeared from the blood and the other tissues of the body. Old geese and adult pigeons seem more refractory than young ones. Fasting renders them more susceptible to the disease by ingestion of the virus.

According to Freese, one is entitled to assume a bird has died of avian plague if one finds hæmorrhages in the glandular stomach (proventriculus), swelling of the kidneys, marked injection of the bloodvessels in the serosa of the Graafian vesicles, not infrequently accompanied by hæmorrhages, and providing the disease assumes a plague-like form. But the post-mortem appearances alone are not sufficient to settle the diagnosis.

In naturally contracted avian cholera, subpericardial hæmorrhages are generally well marked, and diarrhœa occurs more frequently than it does in avian plague.

It should not be confounded with phosphorus, salt, or other mineral poisoning.

Treatment.—The same means recommended for avian cholera should be adopted. In Germany the State intervenes in cases of avian plague as well as in avian cholera. The regulations against imported birds, especially from Russia, are stringent.

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XXXII. INFECTIOUS LEUCOCYTHÆMIA OF BIRDS.

Synonyms.—Infectious leucæmia; Pseudo-leucæmia.

Definition.—A chronic infectious disease of fowls characterised by symptoms of leukæmia or leucocythæmia, due to a filtrable virus, and probably transmitted from fowl to fowl by some epizoal parasite.

History.—This disease was first described by Veranus A. Moore, who investigated it in 1894 and 1895 in the United States of America. He ascribed it to a special organism named by him the *Bacterium sanguinarium*. In 1908 Ellermann and O. Bang further investigated it in Denmark, where it is not uncommon. These authorities found that when the liquid, obtained from a centrifuged emulsion of diseased organs, was passed through an infusorial earth filter, the clear filtrate inoculated into five fowls gave two of them typical leucocythæmia, thus indicating that the disease was due to an invisible virus.

Etiology and Pathogenesis.—This disease usually occurs in an epizoötic form, and, according to Moore, is quite common. It does not appear to be conveyed by direct contagion or cohabitation, as Moore found that, when healthy fowls were placed in cages with diseased ones, not one of the former contracted the disease. Fowls inoculated intravenously with emulsions of liver, spleen, or bone marrow, contracted the disease, either in a leukæmic or pseudo-leukæmic form. No bacteria or other parasites could be recognised by Ellermann and Bang with the microscope; and culture experiments, whether under aërobic or anaërobic conditions, were invariably negative. Moore, however, claimed to have discovered a non-motile, rod-shaped organism in the blood and tissues. It measured from 1·2 to 1·8 μ in length, and 1 to 1·3 μ in breadth. It frequently appeared in small clumps, but as a rule in pairs united end to end in dumb-bell form. Inoculation and feeding experiments induced disease and death in poultry, pigeons, rabbits, and guinea-pgis, within three days to a fortnight. The feeding experiments only destroyed about one half of the fowls.

Period of Incubation.—This appears to be from one to eight or more weeks. Old hens and chickens are as susceptible as young adults.

Symptoms.—The disease manifests itself with a very high temperature, reaching 109° to 112° F. The birds become drowsy, weak, and dull. The comb, wattles, skin, and mucous membranes, are pallid. The fever is usually of a continuous type, and in acute cases death ensues in four or five days after the first appearance of the disease. In many cases death is delayed for two or three weeks, or even months. Emaciation is gradually progressive. The appetite is capricious or lost. The plumage is ruffled, open, and dull, and the bird in advanced cases hides its head under its wing or in the feathers covering the neck and shoulder. Diarrhœa is generally absent.

Microscopical examination of the blood shows that the number of leucocytes may rise from the normal (about 30,000 per cubic millimetre) to 600,000 per cubic millimetre, the increase being accompanied by a diminution in the number of red cells. In one of Moore's cases the red corpuscles decreased from 3,744,000 to 2,133,000 per cubic millimetre, and the leucocytes increased from 21,000 to 140,000. In another case the red corpuscles were diminished from 3,534,000 to 1,745,000, while the leucocytes increased from 19,000 to 245,000. The increase is mainly due to the large mononuclears, which may form from 80 to 84 per cent. of all the leucocytes, as against the normal 23 per cent. The normal relationship of the leucocytes to the erythrocytes is equal to $\frac{1}{100}$, and out of 100 leucocytes 37 are polynuclear, 40 lymphocytes, and 23 large mononuclear. There is a diminution of the polynuclears, and also of the myelocytes. Accompanying these blood-changes there is an infiltration of the cellular tissue of the liver, bone-marrow, and spleen.

Course.—Although this disease may run an acute or subacute course, it usually takes one, two, three, or four months before death supervenes.

Lesions.—The liver is somewhat enlarged and dark-coloured, and covered with minute greyish areas. The spleen and also the bone-marrow show changes as in ordinary mammalian leucocythæmia.

Treatment.—Probably this disease is transmitted from fowl to fowl by the agency of one or other of the many epizoa infesting poultry or poultry-houses. Intestinal disinfection may be tried. For this purpose cyllin or β-naphthol is recommended. Citrate of iron and quinine should be given as a tonic. The fowl-houses and poultry-runs should be cleansed and thoroughly disinfected. To remove epizoa, the fowls should, if the weather permit it, be immersed in baths containing sulphurated lime or potassium.

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SEPTIC PNEUMONIA OF CALVES AND LAMBS

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Septic pleuro-pneumonia of calves; Contagious inflammation of the lungs and pleuræ of young cattle.

French: Broncho-pneumonie infectieuse des veaux.

This affection has been met with in various parts of the Continent, and investigated by several authorities. In some of the enzoötics described intestinal lesions were present, and the disease would appear to resemble in some particulars what is known as "pneumo-enteritis of bovines" (septicæmia hæmorrhagica).

A consideration of the literature of the subject leads one to infer that both on the Continent and in the British Isles a disease in calves exists characterised by the presence of pleuro-pneumonia of a septic type, and that in some of the enzoötics the affection is associated with intestinal lesions.

The disease known as "white scour" must be regarded as distinct from the above affections, as the symptoms, lesions, and bacteriology entitle it to be classed as a separate entity; hence we have devoted a special chapter to its consideration (see p. 501).

In Holland and Belgium, Poels investigated enzoötics of septic pleuro-pneumonia in calves, the leading features of which were a very acute course, high fever, and lesions resembling those of contagious bovine pleuro-pneumonia. The cause of the disease was said to be a micro-organism closely resembling that of septicæmia hæmorrhagica—*i.e.*, a bipolar bacterium.

According to Cadéac, various micro-organisms may be responsible for the occurrence of the disease, including the ovoid bacterium. One or more varieties are believed to gain entrance to the bronchial mucosa, and set up the infection. The younger the animal, the more readily does infection take place, and the first calf attacked transmits the disease to others, more especially when a large number are kept together in the same building. The infection spreads rapidly unless preventive measures are adopted. Although very young animals are most susceptible, later on, as the virulence of the micro-organisms becomes exalted, the disease attacks calves from four to six months old. Infection is believed to occur occasionally from ingestion and inhalation of material expectorated by infected animals.

In Great Britain the disease was described by Smith of Huntingdon in a paper read before the Lincolnshire Veterinary Association in 1892.* The outbreak occurred in a stock composed of forty calves, aged from three to six months, a few of which were bred on the farm; the others were purchased from outside. This was the third outbreak within three years. Six calves had died, and ten were found seriously ill. The symptoms observed were loss of appetite, emaciation, a short husky cough, followed by evidences of acute pneumonia. The course was rapid, and one animal was found dead in the morning, although no symptoms of illness were observed the previous evening. The lesions found on post-mortem examination were those of an acute pneumonia, with gangrenous patches of various sizes scattered over both lungs. In some cases the pulmonary tissue was transformed into a dirty grey or blackish mass; in others, when the gangrenous foci were close to the pleural surface, the latter was raised in the form of large blisters; and in some instances the softened mass had ruptured into the cavity of the chest. In the calf that died rapidly a cavity was found which had contained about a pint of pus, and had ruptured.

The treatment adopted consisted of inhalations of steam medicated with carbolic acid twice daily, the isolation of the infected animals from the healthy, and the cleansing and disinfecting of the eyes, nostrils, and feet, so as to prevent the spread of infection. Attention was also devoted to hygienic and dietetic measures, and the buildings were thoroughly disinfected. Under this treatment there was marked improvement, and only two more calves succumbed.

From the discussion on this paper that followed it was clear that the disease was known to several practitioners in the county of Lincolnshire, and the losses appeared to be very serious, as the outbreaks recurred annually.

Penberthy† gave a full description of the disease, and stated the results of his investigation of several outbreaks. In these, as well as in Smith's cases, the lesions were confined to the respiratory system, as in only two instances was diarrhœa observed, and in one only were gastro-intestinal lesions found on post-mortem examination. The diarrhœa was ascribed by Penberthy to the presence of parasites. The ages of the animals in one outbreak investigated ranged from six months to two years. The history given was as follows: Three calves newly purchased were introduced into a byre; they developed pulmonary symptoms and died. Two months afterwards the young stock reared on the farm, and aged from six to eighteen months, were put into this byre. On the seventh day four of these animals were observed to be ill, and one died during the night; the other three cases showed on examination evidences of pleurisy and pneumonia. Three fresh cases occurred, and were found to have a temperature of 104° to 105° F., and on the following day pulmonary symptoms were in evidence, and four of the cases died within four days. Two cases recovered, but throve very badly.

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* *Veterinary Journal*, 1892.

† *Journal of Comparative Pathology and Therapeutics*, vol. x., 1897.

In another outbreak investigated by Penberthy, nine cases were observed; the ages of the animals ranged from six to ten months. The symptoms presented were cough, a temperature of 104° to 106° F.; a discharge, more or less profuse, from the eyes and nostrils was observed in some cases. A physical examination of the chest revealed evidences of pleurisy and pneumonia. The animals remained in the standing posture during the course of the disease, and only lay down when a fatal termination was approaching. In two cases diarrhœa was observed. Death occurred on the third day after the first appearance of symptoms, and seven animals out of twelve succumbed. These animals had never been on pasture, but were housed and hand-fed. On the same farm several calves were attacked by a mild form of the disease, and all recovered, but they showed a persistent cough for several weeks.

In another outbreak twenty calves were attacked, and the majority died within ten days. The lesions observed in some of the cases simulated those of contagious bovine pleuro-pneumonia. The organs chiefly involved were the lungs and pleuræ, but pericardial lesions were not infrequently met with. In only one case was the entire structure of both lungs involved; the anterior lobes were chiefly affected. The pneumonia was of the "patchy" type; the affected lobules were dark in colour, and mapped out by the septa infiltrated with lymph, giving the lung a marbled appearance.

The lesions, however, were differentiated from those of contagious bovine pleuro-pneumonia by the following points: "The altered pulmonary tissue was less firm; the interlobular bands were narrower, contained less fluid, and were more easily broken down than in pleuro-pneumonia contagiosa; also, the pleura was less thickened, and was darker in colour; the diseased foci were more numerous, and showed more tendency to break down and become gangrenous."

Penberthy was of opinion that the affection was contagious, and that the virus remained virulent for a considerable time outside the animal body, and was capable of contaminating buildings, thus accounting for the recurrence of the disease as an enzoötic.

He also pointed out that the period of incubation was short, and the course in fatal cases was rapid, the latter being rarely of longer duration than three days. Calves under twelve months of age were more readily and seriously affected than older animals. No mention was made of the bacteriology of the affection.

The treatment adopted was similar to that found successful by Smith (see p. 497).

We have referred to the above outbreaks at length because the clinical history and lesions correspond to those occurring in enzoötics of the disease that we have observed ; also, it is apparent from a consideration of these cases that an infectious pneumonia of calves exists in the British Isles which may show no intestinal lesions.

The bacteriology of the disease has not been definitely ascertained. Sir John McFadyean informs us that the affection may depend on different micro-organisms, and that in some of the cases investigated in Great Britain, bipolar bacteria were found to be the cause of the disease.

Cadéac recognises an infectious broncho-pneumonia of calves as a disease *per se,* and also a secondary form of intestinal origin, and one arising from infection of the umbilicus. The intestinal infection lessens the resisting power of the animal, and the micro-organisms spread from the intestine to the pulmonary tissue, and induce lesions therein.

According to this authority, the disease is very fatal, and the animals that survive remain enfeebled and never thrive.

The symptoms described are similar to those already mentioned by British observers. In some cases the pulmonary lesions were very extensive, and the smaller bronchi were obstructed by the exudate, so that death occurred in less than a week. In others—and in France these were more frequently observed—the course of the disease lasted from one to two months ; the animals became enfeebled, and died from profound intoxication. Numerous foci of suppuration were found, especially in the anterior lobes, or each lobule was transformed into a small abscess. Pleurisy, pericarditis, and enteritis, were present as complications.

Some observers record outbreaks of septic pleuro-pneumonia in calves not more than a week old. Again, the disease has been met with as an acute septicæmia, with an absence of pulmonary lesions, and large numbers of bipolar bacteria were found in the blood.

Treatment of any kind was not found to be successful.

Prophylactic measures should be carried out, which include isolation of the affected animals; disinfection of the buildings, and leaving them unoccupied as long as possible.

SEPTIC PNEUMONIA OF LAMBS.

This affection is met with in the British Isles, and we have met with several outbreaks in which not only were lambs of all ages attacked, but also the disease extended to the full-grown sheep. The leading symptoms were high fever, accelerated respirations, nasal discharge, cough, loss of appetite, and the ordinary evidences of pneumonia. In many instances the course was rapid and the mortality high. No special cause could be discovered beyond the fact that in one instance the animals had been shorn during cold weather and grazed on exposed lands, and in another they had been dipped during cold weather.

The lesions found in every case were those of pleurisy and broncho-pneumonia. According to Cadéac,* the disease occurs in various parts of France, being more common in animals that are housed. Young lambs are those chiefly attacked, and the affection is not frequent in those that have passed the age of three mouths.

The bacteriology of the disease is not definitely determined; probably more than one micro-organism is responsible.

The mortality is very high. In some instances death may occur within twenty-four hours from profound intoxication, without appreciable pulmonary lesions. Generally, however, the cases last from ten to fifteen days, and show extreme feebleness and emaciation, with evidences of pneumonia.

Prophylactic measures are of importance, and consist in isolation of the affected animals and all suspicious cases, attention to hygienic and dietetic measures, and thorough disinfection of buildings.

As regards medicinal treatment, Cadéac advises that salicylate of soda be added to the drinking-water—about 1 gramme to each pint.

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* "Pathologie Interne."

WHITE SCOUR

THE term "white scour" is the title popularly applied to an extremely fatal disease of new-born calves, owing to its predominant symptom being a whitish diarrhœa. Other new-born animals are, however, occasionally attacked, especially pigs.

HISTORY.

The disease is prevalent in several Continental States and in Great Britain, while in Ireland towards the end of last century its ravages were severely felt in the dairying counties of Munster. It has been clinically recognised by veterinarians and breeders for several decades, but it is only within comparatively recent years that it has engaged the attention of scientific veterinary inquiry. In 1891 Jensen began the scientific investigation of the malady in Denmark, and since then it has been the subject of important study by Poels in Holland; Willerding, Schütz, Bouget, and Joest in Germany; Dele in Belgium; Pernoncito and Piana in Italy; Galtier, Lesage and Delmer, and Nocard in France. Nocard also investigated the disease in Ireland in 1901.

ANIMALS SUSCEPTIBLE.

Although the disease is practically confined to new-born calves, it is possible to have naturally occurring outbreaks occasionally in other new-born animals, especially in swine. It is stated that in certain breeding establishments in Normandy *all* the new-born animals succumb to its ravages, unless special precautions are adopted.

ETIOLOGY.

All investigators agree that the disease is of bacterial origin, but there is a sharp difference of opinion as to what is the actual infecting organism and its portal of invasion. Thus Jensen, Poels, and the German investigators, believe that the disease, in the majority of cases, is caused by one or more types of the colon bacillus, and that infection mainly takes place through the mouth. On the other hand, French investigators, such as Nocard, and Lesage and Delmer, deny the etiological importance of the colon bacillus, or that infection takes place through the mouth, and assert that the disease is caused by a pasteurella, and that infection takes place via the umbilical vessels. Weighty arguments in the shape of experimental evidence are adduced in favour of both these views of the etiology by their respective supporters, and it seems possible that organisms of both the coli and pasteurella types may be responsible for outbreaks. It should, however, be pointed out that colon bacilli of various types have been found in the blood and organs of affected calves, *and that cultures of these bacilli have reproduced the disease in susceptible animals by feeding with small doses. A small quantity of fæces from an infected calf will, when given* PER OS *to a healthy calf, also produce the disease.* In addition, it has been proved that a prophylactic coli serum can be obtained which will protect animals in a large percentage of outbreaks. On the other hand, it is *found impossible to set up the disease in susceptible animals by feeding with even very large doses of the pasteurella culture,* which requires to be injected intravenously in order to produce effect, and it is not possible to protect animals on infected farms with a serum prepared from it.

The French investigators, however, assert that the pasteurella is found in the blood from the moment that the first external symptoms appear until the time of death, and that it is only during the last hours of the victim's life that colon bacilli invade the circulation.

But McFadyean and others have pointed out that the French and Irish investigations are quite insufficient to oppose the facts put forward by the supporters of the colon bacilli theory, or to disprove the intestinal origin of white scour.

Jensen, who has made a very comprehensive study of the disease, has grouped or subdivided the various types of colon bacilli according to their actions on the various sugars (dextrose, maltose, lactose, and saccharose), and found them to be partly coli, partly paracoli, and partly, but only rarely, aërogenes forms. In rare cases the disease is apparently caused by the *Bacillus pyocyaneus* or *proteus* forms. He (Jensen) states that in general the alimentary canal of the new-born calf is only very slightly resistant with regard to the action of bacteria, and in this, one has to seek for the explanation of the fact that so many different bacteria are able to produce the affection which has been so long known under the term "white scour." It would, however, be erroneous to suppose that these different bacteria excite diseases which are absolutely identical from the clinical or pathological standpoint. Indeed, it seems that with experience one can without difficulty recognise differences which Jensen classifies as follows :

1. The disease caused by *coli bacilli* shows itself under *two* forms. In the *first* of these, which generally sets in soon after birth, and runs a rapid course, one finds prominent enteric lesions, swollen, red mesenteric glands, generally swelling of the spleen, hyperæmia of various organs, and, lastly, masses of bacteria in the blood and in these organs. The *other* form runs a less rapid course. In most cases it sets in three to five days after birth, and it determines death after two to four days. The intestine is distended with gas, and often pale, but more pronounced enteric lesions are absent. The mesenteric glands are swollen, but usually pale, and the spleen, as a rule, is not swollen. In the blood and organs one generally finds no bacteria, or, if present, they are only few in number, and the same applies to the mesenteric glands. Indeed, twenty-four hours after death the number of bacteria in the blood generally shows no important increase.

2. The disease caused by the *aërogenes bacilli* appears in the main to exhibit the same course and to have the same lesions as that caused by the coli bacilli.

3. The disease caused by the *paracoli* or *pseudocoli bacilli* may exhibit a variable picture. It may run the course of a hæmorrhagic enteritis; but the enteric lesions are frequently not very pronounced, whereas the mesenteric glands, the splenic enlargement, and the degenerative changes in the internal organs, are very prominent. In rare cases there is added to the before-mentioned condition fibrinous exudations into the serous cavities. One always finds numerous bacteria in the blood, and especially in the spleen and lymphatic glands.

4. The disease caused by the *B. pyocyaneus*, which has hitherto been observed only by Poels, has profuse diarrhœa and increasing weakness for its chief symptoms, and post-mortem it shows red specks on the intestinal mucous membrane and degeneration of the liver, but no swelling of the spleen. The *B. pyocyaneus* occurs in almost pure culture in the intestine, while the blood, on the contrary, is sterile.

5. The *proteus* infection sets in when the calf is a few days, or perhaps a week, old, and it runs a slow course. The fæces are very fœtid, but never mixed with blood. At the post-mortem one finds the intestines greatly distended with gas, the serous and mucous membranes pale, often to a pronounced degree, and the latter, after death, rapidly assume a macerated appearance. The spleen is not swollen, and the mesenteric glands are enlarged, but only slightly hyperæmic. Proteus bacteria are found in considerable numbers in the intestinal contents (which in normal calves contain no bacteria that liquefy gelatine). On the other hand, the spleen and the mesenteric glands are free from bacteria. Quite the same

clinical and pathological picture can be experimentally produced by infection with isolated proteus bacteria.

Nocard, in his investigation of the disease in Ireland, concluded that the causal organism was a typical pasteurella, which invaded the body via the umbilicus at the moment of birth. He recognised two forms or phases of the malady—viz., (1) *an acute septicæmic form,* which appears shortly after birth, accompanied by white diarrhœa, swollen umbilical vessels, and, if the animal survives for a time, acute joint complications often develop; (2) a form known as *lung disease,* which follows as a complication of " white scour " proper. It occurs in calves about six weeks old which have recovered or passed through a mild form of the disease. Nocard found the direct cause of the lung lesions to be an extremely polymorphous bacillus, identical with the bacillus of ulcerous lymphangitis in the horse and caseous lymphadenitis in the sheep (*i.e.,* the Preisz-Nocard bacillus). He found that this organism was not capable of invading the pulmonary tissue of healthy calves; but when the toxins of the pasteurella have paralysed the defensive action of the phagocytes, the bacillus penetrates the tissues and there multiplies, provoking the formation of encysted abscesses, the evolution of which, slow and progressive, continues even after the calf has recovered from the initial pasteurella infection.

LESIONS.

In acute cases in this country the lesions observed are those of septicæmia, but, of course, they vary somewhat according to the length of time the disease has been in existence. If the animal has been ill for some days, the carcass is found much wasted and soiled with diarrhœic discharge. The umbilicus, according to Nocard, is always swollen and hard, enclosing a clot easily broken down, and sometimes soft and purulent. The peritoneum shows a condition of diffuse congestion, and petechiæ and ecchymoses are often observed. In chronic cases a fibrinous peritonitis may be found. In the alimentary tract the first three divisions of the stomach may exhibit submucous hæmorrhages. In the abomasum and small and large intestines inflammatory changes of the mucous membrane, with petechiæ and ecchymoses, are common, especially in the colon, where hæmorrhages into the lumen are frequent. The solitary glands are found swollen, congested, and sometimes necrotic. The mesenteric glands, especially those of the colon, are swollen and engorged with blood. The spleen and pancreas are generally found normal. The liver is somewhat swollen and of a yellowish colour. The kidneys are congested, and the urine is highly albuminous, but never blood-stained. The bladder, especially towards the neighbourhood of the fundus, shows petechiæ and ecchymoses.

Lungs.—In the early stages no marked lesion may be noticed in the lungs. Livid, airless areas of atelectasis throughout the organs usually constitute the first noticeable alteration; later on, areas of broncho-pneumonia, accompanying which are sometimes seen hæmorrhagic centres resembling infarctions. In the areas of broncho-pneumonia small miliary abscesses develop, containing a thick material resembling pus, and rich in the polymorphous bacillus that is said to cause the lesion. Later the lesion develops into an interstitial pneumonia, and the small abscesses become confluent, the result being a consolidated lung riddled with abscess cavities. Pleuritis occurs over the affected portion of lung, and adhesions between the lung and chest wall are ultimately formed. The bronchial lymphatic glands are always found normal. The heart shows petechiæ on the epicardium, and a fibrinous pericarditis may occasionally be met with.

Arthritis is frequently present, especially in the hind-limbs. The hock and stifle joints are those most frequently affected. In the early stages the synovial membrane and articular surfaces are inflamed, and the tissues surrounding the joint are infiltrated with a yellowish gelatinous exudate. In the later stages the joint becomes distended with a viscid brownish synovia, which may ultimately become replaced by a thick fibrous exudate which fills up the joint. There is no tendency for the lesion to become pyæmic.

SYMPTOMS.

The disease is seen in new-born calves ranging in age from a few hours to ten days. Sometimes the calf dies the same day as born without showing the usual symptoms of the affection. As a rule, however, it lasts from three to six days. Occasionally the attack is slight, the animal apparently recovers, but is certain to afterwards become a victim to lung disease. The most characteristic symptom is a white frothy diarrhœa, which may become yellowish in colour, and is often slightly or deeply blood-stained. There is constant straining, which may lead to eversion of the anus. The victim quickly becomes weak, the hair stands on end, there is a thin clear discharge from the nostrils, and profuse lachrymation. The eyes are sunken, the belly is tucked up, and the back arched. The temperature is high in the early stage, and may reach 106° F., but drops to subnormal prior to death.

As the disease progresses the animal becomes unable to stand, and lies on its side groaning, with its head often turned round to its flank.

No colicky pains are present. Arthritis in one or more joints is a common complication. The joint is swollen, hot, and painful, and the animal refuses to put weight on the affected limb. When all four legs are affected, the calf appears as if paralysed. Brain symptoms may occasionally be noted. Necrosis of the mucous membrane of the mouth, especially of the gums and inside of the cheeks, is sometimes met with as a complication.

In cases suffering from the pulmonary form of the disease, the usual evidences of pneumonia are present.

DIAGNOSIS.

It is usually a fairly easy matter to clinically diagnose the disease, owing to its enzoötic or epizoötic character, its great fatality, and the presence of white frothy diarrhœa, with lung and joint complications.

In some instances there is no history of any primary intestinal affection, as, being of a mild character, it has been overlooked. In such cases the first symptoms presented may be those of pneumonia, and the real nature of the disease may not be suspected until a post-mortem is held, and the peculiar character of the lung lesions is discovered.

COURSE AND PROGNOSIS.

The disease runs a very rapid course, and in a very large percentage of cases terminates in death. The prognosis is extremely grave.

TREATMENT.

Medicinal treatment of affected calves is useless. Good nursing, hygienic surroundings, and attention to the patient's comfort, should greatly assist convalescence. In warm weather the animals do best in the open air.

PROPHYLAXIS.

Nocard, who, as the result of his investigation in Ireland, believed that white scour is generally the consequence of an umbilical infection by a specific pasteurella at the moment of birth or shortly afterwards (through the wound resulting from the rupture of the umbilical cord), stated that farmers might ward off the disease if they conformed exactly to the following rules:

"1. Provide the cows that are about to calve with dry and clean bedding, and keep it in good condition until after the calf is born.

"2. When the cow shows signs of being about to calve, the vulva, the anus, and the perineum, should be disinfected with a warm solution of lysol in water—20 grammes of lysol to 1 litre of water (2 per cent. solution). The vagina should also be washed by injecting (by means of a large syringe) a considerable quantity of the same warm solution of lysol.

"3. Whenever possible, the calf should be received upon a piece of clean cloth, or at least upon a thick bed of fresh clean straw, unsoiled by urine or excrement.

"4. The umbilical cord should be tied immediately after birth with a ligature of twine which has been kept in lysol solution. The umbilical cord should then be cut below the ligature.

"5. The portion of the umbilical cord still attached, as well as the umbilicus, should be dressed with the following solution, applied by means of a large brush :

Rain-water	1 litre (say 1¾ pints).
Iodine	2 grammes (say 31 grains).
Iodide of potassium	4 grammes (say 62 grains).

"6. The disinfecting of the umbilicus and the cord should be completed by dressing them with another brush dipped in the following solution :

Methylated spirit	1 litre (say 1¾ pints).
Iodine	2 grammes (say 31 grains).

"7. The operation is finished, after this spirit has evaporated, by painting upon the cord and umbilicus a thick layer of collodion and iodine (1 per cent.), applied with a third brush. When the collodion is dry, the calf may be left to the care of its mother."

Jensen, on the other hand, who believes the disease is caused by a variety of forms of colon bacilli, and that infection takes place via the alimentary tract, found in Denmark that disinfection, isolation, cleaning, etc., only led to slight and temporary improvement, and was thereby induced to provide, if possible, a serum capable of immunising against it. He considers it impossible to prepare a single serum which will be satisfactory in all outbreaks, partly because there are enzoötics with exceptional bacteriological circumstances, such as those due to aërogenes, pyocyaneus, and proteus infections, and partly because the number of different forms of the colon bacillus is very great. But he believes it is highly probable that by a judicious selection of cultures, and by using a large number of these, a serum may be obtained suitable for 90 per cent. of the outbreaks. The majority of the remaining outbreaks can, he believes, certainly be treated with a paracoli serum. During recent years he has been working with a serum which was obtained by using a dozen specially selected strains of coli bacilli, and which has given satisfactory results in about 70 per cent. of infected farms.

For the preparation of the polyvalent coli serum the horse is used. Various strains of the bacillus isolated from outbreaks are cultivated separately in bouillon at 37° C. for twenty-four hours; the cultures are then measured, mixed together, and injected intravenously into the horse at intervals of not less than fourteen days. There is no objection to beginning the immunisation with single strains, and gradually increasing the number of these. All virulent strains of the bacillus are not equally good for producing immune serum, and the serum of the horse undergoing hyperimmunisation should be frequently tested for agglutinins and bacteriolysins for the usual forms used. For paracoli infections a monovalent paracoli serum is prepared from the horse in the same manner. Although the agglutinating and bacteriolytic powers of a serum may not always appear equal, as the former may be strongly developed and the latter completely absent, still, Jensen has found that a serum capable of agglutinating cultures of the bacillus isolated from an outbreak in dilutions of 1 in 1,000 to 5,000 will, almost without exception, show itself also bacteriolytic to the organism. In other words, the serum may be used on the farm, for the animals from which the cultures were made.

The way in which the serum is employed is as follows:

Agar plates are prepared from the spleen or mesenteric glands, and at the same time gelatin plates are also sown from the intestinal contents, in order to detect any proteus infection that might be present. The isolated cultures are then examined with regard to their behaviour towards lactose bouillon in fermentation flasks, by which means paracoli infection can be recognised. The agglutination test is tried with one or several sera, using dilutions of 1 in 50, 1 in 100, 1 in 500, 1 in 1,000, or even 1 in 2,000 and 1 in 3,000. If the serum in a dilution of 1 in 500 to 1 in 2,000 causes a precipitate, it may be taken as probable that it will prove serviceable. The serum is sent out in doses of 20 c.c., and the whole dose is given subcutaneously at one time, as soon after birth as possible.

JOINT-ILL

By E. WALLIS HOARE, F.R.C.V.S., and R. HUGHES, F.R.C.V.S.,
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Synonyms.—Joint-evil; Navel-ill; Pyæmic umbilical infection; Constitutional synovitis; Specific synovitis; Specific polyarthritis; Scrofulous disease of the joints (old term).

French: Arthrite de nouveau-nés; Arthrite infectieuse des jeunes animaux; Paralysie des jeunes animaux; Polyarthrite septicémique de nouveau-nés.

German: Fohlenlähme; Kalblähme; Lammerlähme.

Definition.—A pyæmic infection of the umbilical cord, producing lesions of the joints and other structures, also marked constitutional disturbance, occurring especially in foals, less frequently in calves, lambs, and young pigs, and sometimes in young dogs.

HISTORY.

The disease is said to have been first described by Brugnone of Turin in 1781. Gmelin, however, states that reports regarding the affection were found in the records of the Marbach stud in Würtemberg in 1767. Many other Continental authorities devoted attention to it from 1825 to 1865. It was mentioned by Percivall in his "Hippopathology" (1849). He gave extracts from a paper on the disease by Bernard of Boulogne that appeared in the *Recueil de Médecine Vétérinaire*, March, 1828, in which the clinical aspects of the affection were correctly described. The disease was believed to originate from the mare, and it was said that, once it appeared, the foals in succession would be affected, unless suckled by a mare free from contamination. This paper appeared in the *Veterinarian* four years later, and in the same journal* Pritchard of Wolverhampton wrote a full account of the disease as it appeared in England. He ascribed the affection to alterations in the character of the milk, due to working the mare and only permitting the foal to suckle her when she returned from work. Percivall had little experience of the disease, and referred his readers to Pritchard's paper on the subject. He, however, thought that the malady "came fairly under the heading of rheumatic inflammation of the joints, and that in point of fact it was a form of rheumatism peculiar to foalhood."

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* *Veterinarian*, vol. v., 1832.

Hering, in 1867, divided the diseases grouped under the term "Lähme" into general weakness, joint-ill, diarrhœa, enteritis, pneumonia, etc. Other observers, including Träger, adopted various symptomatic designations of the affection, and recognised a "cerebral," an "intestinal," a "cerebro-intestinal," and a "joint" form of the disease.

In 1869, at the Government Stud, Graditz, Silesia, the disease occurred as an enzoötic, and was investigated by Bollinger; the mortality reached 72 per cent.

Bollinger studied the disease from 1873-1875, and recognised its true nature. The equine form of the affection was more particularly investigated in Normandy by Lecocq and Darrean, and in Germany by Strauss, Roloff, and others.

Walley* studied the disease especially in lambs, and defined it as an inflammatory affection of the synovial membranes of the joints of a specific character.

Robertson† regarded the well-pronounced and malignant cases of joint-ill as probably "scrofulous" in their nature, and described the disease as a "specific arthritis of a tuberculous character." This view, however, was not supported by any bacteriological evidence.

In later years the malady has been investigated in England by Penberthy and others, and by Gmelin in Germany.

ETIOLOGY.

In former times various views were held with reference to the etiology of joint-ill.

According to Fleming, the septic or pyæmic nature of the disease was not recognised in England in 1878.

Bollinger's view, that the affection was a purulent omphalo-phlebitis due to local infection of the umbilicus and umbilical vessels by pyogenic organisms, causing a metastatic pyæmia, was confirmed by further researches.

The bacteriology of the disease is not as yet definitely settled. According to some authorities, the *Staphylococcus pyogenes aureus* and the *Streptococcus pyogenes* are the micro-organisms that are responsible for the affection, while other observers think that the specific micro-organism has yet to be discovered. Again, some are of opinion that the above micro-organisms may, by passing through a series of cases, assume a specific character. Others assert that an organism of the pasteurella or fowl cholera type is the causal agent in many outbreaks of the disease.

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* *Veterinary Journal*, 1876.

† Robertson, "Equine Medicine," second edition, 1890.

According to Lignières, the pasteurella has a predilection for the synovial membranes.

Gmelin* found in some cases cocci surrounded by a fine transparent capsule, occurring singly or arranged in pairs or in short chains. These corresponded very closely in their morphological characters with the pathogenic *Micrococcus tetragenus*, and Gmelin believed they were possibly identical with it. They were found in the degenerated thrombus of the umbilical artery, and in moderate numbers in the blood and tissue-juice of the heart, also in the fluids of the joints and in the pus therein, and in small numbers in the fibrino-purulent exudate on the brain. According to this observer, it is not definitely known whether the pyæmia is due to a single or to a mixed infection.

Infection takes place at birth or shortly afterwards, the infective material probably gaining access to the unclosed umbilicus by the animal lying on a dirty floor or on infected litter, or it may be conveyed by the hands of the attendant when applying a ligature to the umbilical cord.

The condition of the dam is not known to exert any influence in the etiology of the disease, except that she may be a "carrier" of the infecting agent.

Cases of intra-uterine infection are recorded. Penberthy observed that the subjects of joint-ill were frequently born before their time, and showed "knuckling over" of the limbs, inability to stand, and swelling of the joints, a few hours after birth, and death occurred within a few days. In cases dying soon after birth the joints showed the presence of a gelatinous material both in and around them. In one instance a colt foal died two days after birth, and an abscess was found in the stifle-joint. Injection of cultures from this abscess into the jugular vein of a healthy foal caused polyarthritis. The same authority observed simultaneous metritis in ewes and joint-ill in lambs, and was of opinion that, as many cases of the disease show the earliest symptoms so soon after birth, it was rational to conclude that infection may probably occur either before or during parturition.

The commonest mode of entrance for the micro-organisms is through the thrombus that naturally forms in the umbilical vessels after the umbilical cord has been ruptured or severed.

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* *Journal of Comparative Pathology and Therapeutics*, March, 1892.

The chief thrombi form in the umbilical arteries, as the umbilical vein is generally collapsed and the thrombus therein is insignificant. The micro-organisms induce disintegration of the thrombi; they multiply therein, and gain entrance to the blood-stream, and frequently locate themselves in joints or in the vicinity of the latter, but may colonise in any region of the body.

There are other modes of entrance. The micro-organisms may enter the connective tissue of the part and induce suppuration therein, or they may pass directly to the peritoneum and give rise to peritonitis; again, they may extend along the urachus and reach the bladder.

The effects produced will vary according to the number and virulence of the micro-organisms, their location in the body, and the susceptibility of the animal. Hence generalised forms of the disease are met with, in which the course is very acute and the termination rapidly fatal; while in other instances the affection may assume a latent type, and symptoms are not developed until a variable period after birth.

Again, the micro-organisms may induce suppurative conditions in the body, without involving the joints.

The disease is said to occur more frequently amongst well-bred and thoroughbred foals; but some observers suggest that as more attention is devoted to the affection when it appears in the above class of animals than in the common breeds, the apparent greater frequency in the former is explained.

The prevalence of the disease in some breeding establishments is due to the presence of large numbers of mares and foals from various parts at such places, which may be carriers of the infection. Infected buildings are also a well-known source of infection.

Insanitary surroundings and dirty buildings are generally regarded as predisposing causes, but it must be remembered that on many farms where the surroundings are of the worst possible description the disease may never be met with. This is well exemplified in Ireland, where in some districts the malady is rarely or never seen.

As already remarked, the disease was found by Penberthy to occur frequently in foals born before their time. His observations showed that in twenty-seven out of sixty-one cases the foal was born before the completion of the normal period of gestation, and on three occasions the foal showed swelling of the umbilical cord at birth.

It is generally found that more colt foals are affected than fillies. This is attributed to the slower healing of the umbilicus in the former, due to contamination of the part by the urine, in consequence of the close proximity of the umbilicus to the prepuce. According to Gmelin, 69 per cent. of the cases that came under his observation were in the male sex.

A pervious urachus must be regarded as a predisposing cause of considerable gravity, as the trickling urine keeps the umbilicus moist, and the surrounding skin and coat are soiled with decomposing urine and adherent dirt. In such a condition the tissues of the umbilicus, with their cells saturated and reduced in vitality, offer a ready and fertile soil for any pyogenic organisms that may chance to come in contact with the part.

MORBID ANATOMY.

The lesions are of a varied character ; in some instances a large number of organs and structures are involved, while in others the pathological changes are limited to certain regions.

Bollinger found the following lesions in autopsies of two foals, one aged three weeks and the other five weeks :

The umbilical vessels showed inflammation and thrombosis; the thrombus was in a suppurative condition and extended into the portal vein, which suffered from thrombosis as well as its hepatic ramifications. In one case thrombosis of the pulmonary artery was present, also double pleurisy with pericarditis, purulent arthritis affecting several joints, and large intramuscular or subcutaneous abscesses. In the other case pulmonary abscesses were observed, also circumscribed pleurisy, tumefaction of the bronchial and mesenteric glands, and ophthalmic lesions consisting of an iridocyclitis.

According to Bollinger, the development of the disease occurs as follows :

1. Inflammation of the umbilical vessels, especially of the umbilical vein.

2. A decomposing and softened thrombus communicating with the portal vein. This forms the source of a metastatic pyæmia, and various structures are attacked, including the synovial membranes, and a septic condition of the blood is produced.

According to Ostertag, the tissues surrounding the umbilicus are swollen and suppurating, and the umbilical cord in a similar condition; the margins of the umbilical ring show ulcerative thickenings, abscess formation has occurred in the interior of the ring, and drops of pus can be squeezed out therefrom. The external lesions may have healed, but in the deeper tissues suppurative thrombi and phlebitis are found, with thrombosis of the portal vein and its hepatic ramifications.

Examination of the affected joints shows the synovial membrane injected, swollen, and thickened, the fluids increased in amount, turbid and containing flocculi, and often of a purulent character. The articular cartilages show ulcerative degeneration, and necrotic changes may be present. The joint capsule may be perforated, and the suppurative process may extend to the bones forming the joint. Abscesses may be found between the muscles and tendons in the vicinity of the affected joints, and the suppurative process may extend to the tendon sheaths, this often occurs when the knee or hock is attacked. In some acute cases we observe in the vicinity of the joints and tendon sheaths infiltration with a yellow jelly-like material; in others the material may be thick and purulent, or fluid and sanguineous.

In the more advanced cases the ends of the bones close to the articular cartilages may be involved. Gmelin, who studied this disease very thoroughly, recognised two forms of morbid alterations—viz.:

1. *Primary*, which are located at the point of entrance of the infection—*i.e.*, the umbilicus and its vicinity and the umbilical vessels. The connective tissue of this region is specially favourable to the spread of the suppurative process, and if the outward passage of the pus be obstructed, it finds its way under the peritoneum which extends over the inner umbilical ring, and an abscess is formed, into which the umbilical vessels appear to enter.

2. *Secondary or Metastatic*, in which the suppurative process extends to various regions of the body. Gmelin found that all stages of lesions of the joints may be found in the one cadaver, and that joints in which there were no evidences of disease during life showed morbid changes at the autopsy. There was no special predilection in the joints attacked. This view is in accordance with the experience of British observers. The disease is more apparent in the stifle and hock joints, because of the more capacious joint capsules, while it is less readily observed in the joints covered by muscles.

The occipito-atloid joint was found to be frequently affected.

The arthritic lesions show various degrees of intensity, depending on the virulence of the infecting agent and the duration of the disease.

The peritoneum does not often show well-marked lesions, unless in cases where an abscess ruptures into the peritoneal cavity.

The bladder may present catarrhal inflammation of its mucous membrane, due to extension of the disease from the urachus.

In many instances the urachus is pervious, but this condition is met with independently of joint-ill; the urachus in joint-ill shows purulent inflammation.

In the nervous system evidences of meningitis may be observed.

According to Gmelin, the most frequent lesion is a deposit of fibrinopurulent exudate in the vicinity of the cerebellum and medulla.

In the lungs, according to this observer, the most important secondary changes are found, in the form of a broncho-pneumonia with abscess formation.

The liver and spleen were found to contain abscesses by some observers, but in the cases investigated by Gmelin these lesions were absent. Ophthalmic lesions were observed in some cases, consisting of slight thickening of the iris, turbidity of the aqueous humour, slight opacity of the lens, infiltration of the choroid, but no pronounced formation of pus.

Abscesses may occur in the muscles, glands, and connective tissue in any region of the body. Amongst other lesions which have been observed are pleurisy, pericarditis, abscess in the kidney, a pale flabby condition of the heart, and endocarditis.

Penberthy found that in some autopsies no morbid changes occurred in the joints, but gross lesions were present only in the umbilical artery and in the pia mater. In one instance lesions were only present in the tarsal joint and in a mesenteric gland.

SYMPTOMS.

The first symptoms generally appear during the three weeks following birth. Gmelin found that the foals which succumbed to the disease attained an average age of twenty-six days, and symptoms were apparent on the seventeenth day after birth.

In the outbreak that occurred in Silesia, out of forty foals, twenty were ill within the first eight days after birth, ten in fifteen days, and the remainder between the fourth and sixth week.

It is generally recognised that the symptoms rarely or ever appear earlier than the third day after birth, except in those cases of intra-uterine infection already alluded to. Practitioners who have experience of the disease observe that it can develop up to the period of complete drying up and cicatrisation of the umbilicus—*i.e.*, three or four weeks old.

In a small proportion of cases the development of the affection may be delayed for some months. Penberthy has observed that it is possible for infection to take place, and cause only slight dulness and unthriftiness for some weeks or months; then evidences of the disease appear, such as lameness and swelling of one or more joints.

This is in accordance with our experience, and we are of opinion that some at least of those cases of lameness accompanied by swollen joints (especially the stifle-joints) occurring in yearlings and two-year-olds are due to this cause, although often vaguely ascribed to rheumatism.

In ordinary cases the primary symptoms are those of systemic disturbance, which appear for a day or two, or even longer, before the advent of the arthritic symptoms. We observe dulness, lassitude, disinclination for movement, interference with appetite, evidenced by a reluctance to suck the dam, and a tendency to assume the recumbent position; the temperature is elevated to 104° or 106° F. The respirations are accelerated and are of a short, panting character, and lameness suddenly appears, with swelling of one or more joints.

In some cases the first symptom observed by the owner is sudden lameness and swelling of a joint, and the foal is thought to have received some injury in the affected limb.

The joints which may be attacked are the hock, knee, stifle, elbow, shoulder, hip, fetlock, and the occipito-atloid.

The affected joints are swollen, hot, tense, and painful, and the swelling extends to the surrounding connective tissue; the constitutional symptoms increase in severity, prostration occurs, and abscesses form both within and around the affected joints. In severe cases the hair falls off, and a yellow or citron-like fluid exudes from the skin over the joints; sloughing of the skin next occurs, and the ligaments are involved, the result being disorganisation of the articulations affected, and exposure of the articular surfaces.

Constipation may be present at first, and is often followed by an offensive diarrhœa, or these conditions may alternate; diarrhœa, however, is not a constant symptom. Some observers have noted that the fæces are semi-fluid, very fœtid, and of a light-coloured or clayey appearance. Emaciation and debility are well marked, and, according to the degree of the metastatic lesions, evidences of generalised pyæmia are observed.

On examination, the umbilicus may be found moist or weeping and unhealthy in appearance, or it may be swollen and suppurating. In some cases it may be closed, but an enlargement due to the presence of an abscess may be felt at its upper aspect. In many instances it may be found healed and apparently healthy in appearance. Impervious urachus, with dribbling of urine therefrom, is present in many cases, but, as already remarked, this condition is met with independently of joint-ill.

When metastatic lesions occur in various organs of the body, the symptoms presented will vary accordingly; thus evidences of broncho-pneumonia, pleurisy, pericarditis, peritonitis, etc., may be observed.

COURSE.

This is generally acute, and may continue for two or three weeks; but the disease may prove fatal in a few days in some cases, or even in twenty-four to forty-eight hours, according to some observers.

Generally the affection is very acute and the course rapid in very young foals, but in those more matured the symptoms may be subacute and the progress slower.

The average course is said to be about two weeks, but in the cases investigated by Gmelin the average duration was nine days.

PROGNOSIS.

The prognosis is generally unfavourable; especially in very young foals, the mortality varying from 40 to 60 per cent., and it may be even higher.

A small proportion may recover perfectly, but partial recoveries are more common, and the results are seldom satisfactory, as chronic joint disease and lameness result. Gmelin and others have found that foals which survive the disease remain worthless piners.

DIFFERENTIAL DIAGNOSIS.

This is based on the history of the case, the absence of any history of injury, the metastatic character of the arthritic symptoms, and on an examination of the umbilicus. With reference to the last point, however, we must remember that no evidences of any abnormal condition of the umbilicus may be apparent in some instances.

PROPHYLAXIS.

In consequence of the fatal nature of the disease, the great losses it entails in breeding establishments, and the comparative failure of therapeutic agents to give successful results, it is clear that prophylactic measures are of the most supreme importance. The results of careful and systematic prophylaxis have proved highly satisfactory. The foaling-box should be thoroughly cleansed and disinfected. When cases of the disease occur in a breeding establishment, the affected animals with their dams should be rigidly isolated and placed in the care of special attendants, who must have no contact with the healthy animals.

Sulphate of copper solution is advised by some authorities as a disinfecting agent for the floors of the stables.

Disinfection of the genital organs, tail, thighs, and udder of the pregnant mare with a solution of creolin should be carried out.

Special treatment of the umbilical cord of the foal constitutes the most important detail in connection with the prophylaxis of this disease.

Gmelin, who has thoroughly studied this disease and its prevention, has devised prophylactic measures which have proved eminently successful, not only on the Continent, but also in Great Britain.

From a translation of a paper on the subject by this authority which appeared in the *Journal of Comparative Pathology and Therapeutics*, March, 1892, we give the following extracts :

" Every subsequent new case of pyæmic navel infection serves to increase and disseminate the infective material in the stable. Ultimately in the stables, which are generally used year after year for foaling, an accumulation and general dissemination of the infective germs occurs; this serves to explain the fact that in many seasons the disease may appear in quite an epizoötic fashion in the foaling stables. . . .

" The pregnant mares and those with foals at foot are kept in loose-boxes in two different stables. The litter in the boxes is composed of peat mixed with dry foliage. Every box before it is used is cleaned out, washed, and disinfected with 3 per cent. creolin solution, and immediately before it is occupied it is bedded with fresh litter. When a long time has elapsed between the disinfecting and the occupying, a second disinfection with creolin is carried out before the stall is bedded. When the mare has foaled, she is brought, the first thing next morning, into the stalls for mares and foals. If a foal in this stable falls ill, it must, along with its dam, be put into a third stable, which serves as a sick stable and is provided with loose-boxes.

" The prophylactic measures have next to do with the navel of the foal. The object is to promote the formation of healthy thrombi in the umbilical arteries, and to protect the thrombi formed from infection and softening. All suppuration is to be prevented, as it involves a danger of infection. . . .

" As soon as the foal is born, the umbilical cord is washed with sublimate lotion, and then, about 2 centimetres under the sharply-marked transition from general integument to amniotic sheath, ligatured with a linen band which has been steeped in the sublimate lotion. Before tightening the ligatures wait until the pulsation in the umbilical arteries has ceased, or until the movements of the foal or the efforts of the mother to get up require the manipulation to be hastened. Should the mare foal standing, the foal must be caught and held by attendants. When the pulsation has ceased, draw the ligature tight, sever the umbilical cord with clean scissors below the ligature, wash the stump of the cord with sublimate lotion, and over its cut surface and the part of it included in the amniotic sheath apply some concentrated carbolic acid with a brush. On the morning after birth transfer the foal and mother to the special stable for mares and foals. Cast the foal here on a clean bed, clip off the long hairs around the navel, wash the entire umbilical region with sublimate lotion, test the ligature as to position and tightness, and, if necessary, renew it. The cut surface and epidermis-free part of the stump are again to be brushed over with carbolic acid. Repeat this daily until transverse cracks appear where the amniotic sheath joins the cutaneous navel; these cracks occur about four or five days after birth. The remnant of the navel-string acquires a parchment-like character, as the result of the brushing over with carbolic acid. Do not wait until the fissures become deep and the stump is cast off, but cut off the latter with scissors. A trifling hæmorrhage occurs as the result of this, which soon ceases. Disinfect and carefully dry the navel ring, and paint on the wound a stiff zinc-gelatine dressing containing 1 per cent. of sublimate. More consistence can be given to this dressing by mixing some fibres of wood-wool with it. If the surface has been properly dried, this dressing will usually hold until the next morning. In the case of colt foals that urinate with the penis protruding from the prepuce, it is often necessary to apply the dressing once more in the evening. The gelatine dressing is easily softened with warm water, and removed without irritating the skin. After the lapse of from one to three days an opening generally appears on the navel wound, due to atrophy of the connective tissue in the navel ring, retraction of the vessels, etc. The accumulation of pus in this opening must be prevented. Cast the foal every morning, remove the dressing, wash out the cavity, plug with a small dry tampon of iodoform wool, and again close with the gelatine dressing. The wound quickly becomes filled with healthy granulations, and by the seventh or eighth day the treatment may be discontinued. About one hour is required for the dressing of eight foals in the above manner."

Gmelin stated that by adopting the above measures at the Marbach studs he had not lost a single foal from pyæmic navel infection (1890-1892).

In cases where the umbilical cord is ruptured before the practitioner has arrived, ligaturing is useless, as this procedure would only have the effect of imprisoning the micro-organisms.

The following method of prophylaxis we have found effectual in practice, and as it is simpler to carry out, it may be preferred to the above by country practitioners :

If the attendant is not present at the time of birth or immediately afterwards, it is best not to apply a ligature, provided hæmorrhage has ceased, but the end of the cord should be trimmed of irregularities, and then painted over with pure liquid carbolic acid right up to the cutaneous navel. Afterwards the stump of the cord should be dressed with sublimate or biniodide of mercury lotion, 1 in 500, two or three times daily, until it shrivels up. It is a good plan for the practitioner to supply his client, who expects a mare to foal, with a bottle of the above dressing, containing a ligature of silk or soft twine. The owner should be instructed to have the foal dropped on to some clean straw, a sheet, or a tarpaulin, and to immediately apply the ligature, with clean hands, at a distance of ½ to 1 inch from the navel ring, and then cut the cord ½ inch lower down and dress the stump. If the cord is not broken when the foal is delivered, it should be divided between two ligatures, one above and the other below the incision.

Instead of applying pure carbolic acid, some practitioners advise sponging the stump of the cord with alcohol, drying, and then painting with a solution of salicylic acid in collodion (1 in 10), which is repeated daily until the navel dries up. If alcohol is not to hand, mopping the navel dry with cotton-wool before painting on the collodion will often suffice.

If the navel is soiled, the hair round about it should be clipped, and the part washed with soap and water ; the extremity should be cut off, and the stump then painted with tincture of iodine or collodion of salicylic acid.

If the cord should break off short at the navel ring and the vessels retract, it is difficult to apply a ligature, but the collodion of salicylic acid or tincture of iodine should be painted on, and an antiseptic lotion frequently applied.

TREATMENT.

Various forms of treatment have been adopted for this disease, but with indifferent success. When suppuration occurs in the joints the case is, as a rule, hopeless.

Gmelin only observed two cases in which recovery took place : in one a retroperitoneal abscess was surgically opened by a deep incision in the umbilical region, and in the other the umbilical wound was washed out with sublimate solution and compresses of gauze soaked in a similar solution applied to the joints, while ¼ per cent. solution of creolin was administered internally.

Biniodide of mercury with iodide of potassium has proved useful in the hands of some practitioners, from 1 to 2 grains of the former with 5 grains of the latter being administered once daily.

Amongst other agents advised are quinine, salicylate of soda, hyposulphite of soda, etc.

As a local application to the affected joints, belladonna liniment and soap liniment prove useful. Attention should be paid to the surroundings of the patient, and plenty of straw should be allowed for bedding, so as to prevent the occurrence of bed-sores.

Malloch of Kirkby Stephen* has employed acetozone in the treatment of joint-ill with marked success. He injects from 2 to 4 drachms of a 1 in 500 solution of this agent into different parts of the affected joint by means of a hypodermic syringe provided with a special long needle. A tablespoonful of a similar preparation is given every five hours in 2 ounces of the mare's milk. When an abscess has formed and ruptures, a solution of acetozone (1 in 300) is injected into the joint cavity. A weak solution of formalin similarly injected is said to give good results.

Surgical interference is indicated when there are indications of the presence of pus in the affected joint. The pus should be drawn off by means of an aspirator, and the joint irrigated with a warm solution of acetozone or of peroxide of hydrogen, followed by the injection of glycerine of carbolic acid or glycerine of iodine. Mullie advises the injection of a solution of iodine after the purulent material has been withdrawn. The joint should then be covered with a layer of medicated cotton-wool, and bandaged when possible.

So long as the patient takes food of its own accord, treatment may be persevered in; but when there is marked debility and total loss of appetite, the case is generally hopeless.

JOINT-ILL IN CALVES AND LAMBS.

According to Gmelin, infection takes place through the umbilical vein in ruminants, and not through the umbilical arteries. In these animals large thrombi are found in the umbilical vein, which vessel is double up to the umbilical ring.

The symptoms observed in lambs are stiffness in movement, arching of the back, wasting of the muscles; the hocks, knees, and stifle-joints are swollen, hot, and painful; sinuses may extend to a considerable distance from the affected joint, through the tendon sheaths, so that one joint on the same limb may communicate with another.

In some cases the lymph becomes liquefied and absorbed without any external lesion of the joint, or, in cases where the constitution is strong, organisation occurs; the exudate remains a part of the joint, producing permanent thickening, distortion, and stiffness.

Erysipelatous inflammation of the skin may occur in various regions of the body, especially at the orifices of the genito-urinary organs and at the postero-inferior region of the abdomen around the umbilicus, these lesions being evidences of pyæmia.

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* *Veterinary Record,* June 24, 1911.

In calves the symptoms observed simulate those occurring in foals, and prophylaxis and treatment are to be carried out on similar lines to those already suggested.

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BACILLARY PYELONEPHRITIS IN CATTLE

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Mycotic pyelonephritis.

French : Pyélonéphrite Bacillaire du Bœuf ; Pyélite chronique.
German : Pyelonephritis bacillosa bein Rinde.

This disease is a form of pyelitis and nephritis of a chronic type, accompanied by abscesses and the presence of various morbid alterations in the kidneys, urinary passages, and urine. According to Leblanc,* it has been recognised by French veterinarians since 1848, and by German observers since 1867. Its microbic nature, however, was not demonstrated, nor did the disease receive much attention, until the year 1888, and from this period onwards various observers studied the etiology and pathology of the affection.

Bacteriology.—The bacillary character of the disease was, however, suspected prior to the above date, as Siedamgrotzky (1875) found bacteria in pus obtained from the pelvis of the kidney; Pflug (1876) met with a case that followed retention of the placenta in a cow, and suspected micro-organisms to be the causal factor of the renal lesions; Dammann (1887), Hess (1888), Schmidt (1889), Bang (1890), and Friedberger (1890), discovered bacilli of various kinds in the lesions. From 1888 to 1892 Guillebeau and Hess published a series of articles on the disease, and described the pathogenic micro-organism, which they named the *Bacillus renalis bovis*. They also drew attention to the frequency with which the disease followed parturition in the cow, to the ascending course of the infection, and to the morbid phenomena in the kidneys and urine.

In 1891 Höflich drew attention to various points in connection with the disease, including the lesions and the morphology of the organisms discovered in the renal abscesses, the urine, and the purulent material in the pelvis of the kidney. He named the organism the *B. pyelonephritidis bovis*, and observed that it was often present in pure culture in the softened foci of the renal parenchyma. It was a small rod-shaped organism 1 to

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* "Pathologie Interne," par C. Cadéac.

3 μ in length and 0·7 μ in thickness, easily stained by Gram's method, and in the pelvis of the kidney these organisms were always mixed with cocci and streptococci. The bacillus was pathogenic only for the ox. Graf found a probably identical organism in a case of pyelonephritis in man.

In the same year Enderlen discovered in the softened foci in the renal parenchyma a small bacillus from 2·1 to 2·8 μ in length and 0·7 μ in thickness, occurring in a state of pure culture, capable of cultivation, staining readily by Gram's method, and still better by that of Weigert. A variety of organisms were present in the pelvis of the kidney.

Bollinger, in 1891, discovered the *B. renalis bovis,* and stated that it showed no tendency to generalisation, but was constantly localised in the parenchyma and pelvis of the kidney. He formed the opinion that, next to tuberculosis and hepatic distomatosis, pyelonephritis was the most common disease of cattle.

Liénaux and Zwaenepoel (1902), as the result of examinations of various strains of Höflich's bacillus, have been led to associate this micro-organism with the bacillus of Preisz or Preisz-Nocard.

Ernst (1905) in the majority of cases found the bacillus of Höflich, but never in pure cultures. He placed it in the group of *coryne*bacteria, and named it the *Corynebacillus renalis bovis.**

As the bacillus of Höflich, Enderlen, and that discovered by Ernst himself, are practically without pathogenic power for the small laboratory animals, Ernst came to the conclusion that they only played a very minor part in the production of pyelonephritis. The same observer found quite frequently streptococci, bacilli of the colon type, and the *B. pyogenes bovis* of Künnemann, in addition to the *Corynebacillus renalis bovis*.

Semmer (1906) does not recognise the *B. pyelonephritidis bovis* as the specific cause of the disease, but believes that this micro-organism may play some part in the production of the affection. He isolated the above bacillus in a state of purity from three different centres in an affected kidney, but in other cases he obtained a pure culture of a bacterium of the paratyphoid group in one and a mixture of colon bacilli and streptococci in another. Leblanc regards the affection as a polymicrobic one, while Moussu thinks the bacillus of Höflich is the causal agent.

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* German authorities have created a group of bacilli, which they have denominated *Corynebacteria,* characterised by the presence of intraprotoplasmic granulations, swelling of the extremities, and having the property of giving ramified forms in old cultures. Included in this group are—the bacillus of diphtheria (*Corynebacterium diphtheriæ*), bacillus of pseudo-diphtheria (*C. commune*), bacillus of xerosis (*C. conjunctivæ*), and bacillus of glanders (*C. Mallei*), etc.

Many other observers have studied the bacteriology of pyelonephritis, and various micro-organisms were discovered in the lesions. For a full description of these the reader is referred to an excellent article on the disease by Ritzenthaler, of Courgenay, Switzerland, a translation of which appeared in the *Journal of Comparative Pathology and Therapeutics*, March, 1910.

To this article the writer is indebted for the information contained in the present sketch of the disease. He is unable to discover any recorded cases of the disease by British veterinary authorities, and is not aware that the affection occurs in the British Isles.

It will be gathered from a consideration of the above results that the *B. pyelonephritidis* cannot yet be accepted as the specific cause of the disease.

Ritzenthaler drew attention to the importance of Ernst's observation that the bacillus of pyelonephritis showed a great predilection for urine, and possessed the power of splitting urea into ammonia and carbonic acid; also it induced the formation of crystals of ammonio-magnesium phosphate, so that he considered it should be added to the list of urinary ferments.

Modes of Infection.—Authorities differ as to the mode of infection, and up to the present the question has not been decided. Guillebeau and the majority of French authors believe that the bacilli reach the bladder by way of the urethra, multiply there, and slowly reach the kidneys by passing up the ureters. On the other hand, German authorities, including Bollinger, Kitt, Hutyra and Marek, and Ernst, are of opinion that infection takes place by way of the blood-stream. Ritzenthaler believes in the theory of ascending infection, and is of opinion that the disease is generally a sequel to parturition. In such instances the infection probably occurs in connection with septic processes in the uterus, especially the retention of a putrid placenta. The bacilli may reach the vagina from the uterus, and then reach the kidney by way of the urethra, bladder, and ureter. According to Friedberger and Fröhner, the disease occurs mostly in cows, and usually appears soon after parturition. Nocard and Leclainche state that it rarely occurs in the male.

Pyelonephritis has also been observed in oxen, bulls, and calves, and this was one of the arguments brought forward by the exponents of the hæmatogenous theory of origin.

As regards the period elapsing between parturition and the discovery of the disease and the slaughter of the affected animals, Ritzenthaler found that in twenty cases it varied, the shortest being twelve days and the longest twelve months. Hess has observed periods of fifteen and eighteen months. In exceptional cases the disease appeared towards the end of the period of gestation, but in such it is thought that infection resulted from the previous gestation, and that no symptoms were observed until the condition was aggravated by the gestation following.

Morbid Anatomy.—*Kidneys.*—Either one or both organs may be affected, but not to the same degree in each. They are enlarged and increased in weight, but this increase is not equally divided, and one kidney may be far heavier than the other. During the height of the disease the enlargement may be extensive. The right kidney is said to be more frequently and more seriously affected than the left. The capsule is in many instances easily detached ; occasionally it is adherent to the kidney. In some cases it is thickened, its surface shows ecchymoses, and a sero-fibrinous exudate separates it from the kidney. The surface of the kidney is generally paler than normal, it presents ecchymoses, and numerous opaque salient spots of a yellowish-white or brownish-yellow colour, surrounded by a reddish zone.

In some cases the surface of the kidney shows, after removal of the capsule, ulceration and the presence of pus, also superficial softened foci. Sometimes an entire lobule may be converted into an abscess. In the early stages the renal tissue is soft and congested, but later on it becomes hard and dense. On section, white streaks of varying size extend from the pelvis to the surface of the kidney, and correspond with the pale areas on the surface of the organ. The cortex shows similar streaks, and often small abscesses. Softened centres and ecchymoses are found scattered through it. In some cases lobules may be found, which are converted into cavities containing a sanguineous liquid, in which urinary deposits, shreds of fibrin, etc., occur. These cavities communicate with the pelvis of the kidney, and the putrid contents flow from the former into the latter. The kidney may be converted into an enormous cyst with thin walls, due to the boundaries of the cavities breaking down.

The pelvis of the kidney in the early stages shows catarrhal inflammation, but it soon becomes enormously dilated, the dilatation extending to several of the calices. The mucous membrane of the pelvis and calices is thickened, œdematous, and ecchymosed, and may show irregular areas of necrosis or a covering of fibrinous false membrane. The pelvis of the organ contains pus, generally in large amount. The latter may be thick or fluid, and is often mixed with blood, and clots of fibrin are found floating in it. A microscopical examination shows the presence of crystals of ammonio-magnesium phosphate, epithelial cells, etc., and large numbers of the bacteria already described.

The ureters generally resemble thick cords. Their walls are thickened ;

the mucous membrane is œdematous, hypertrophied, and may show superficial ulceration. The lumen may be constricted at some places and dilated at others. In advanced cases large quantities of thick red pus may obstruct the ureter close to the kidney, and cause hydronephrosis.

The bladder shows congestion of its mucous membrane, which may be limited to the orifices of the ureters in some cases. It may be thickened, ecchymosed, and in some instances necrotic patches may be observed. The walls of the bladder may be thickened, and may contain abscesses. The urethra shows thickening of its mucous membrane, ecchymoses, and swelling of the vaginal orifice. The vaginal mucous membrane is often covered with a muco-purulent deposit. It is œdematous, swollen, and of a diffuse red colour, or has a streaked appearance.

The uterus (in cases occurring shortly after parturition) is increased in size and weight, the os uteri is swollen and inflamed, the cavity of the organ contains a purulent sanguineous material, and all the evidences of metritis may be present. Only in rare cases does the disease extend to the Fallopian tubes and ovaries.

In calves several instances of involvement of the urachus in cases of the disease have been recorded, and Cadéac records a case of ascending pyelonephritis in a calf, associated with omphalophlebitis. A case of pyelonephritis in a bull calf seven months old is recorded by Wyssman and Hess. In addition to the usual lesions, ulceration of the prepuce was observed.

According to Ritzenthaler, coexisting lesions in other organs are found fairly often at post-mortem examinations on cows destroyed on account of pyelonephritis.

Symptoms.—The disease is generally regarded as chronic in character, although Leblanc describes an acute form.

The symptoms described by those who have observed the disease are disturbance of the general health, lessened appetite, irregular rumination, emaciation, slight colics, pain evinced on pressure being applied over the loins. The urine is passed in small amounts, it is of a yellow, reddish, or deep red colour, and contains flocculi of a greyish-red tint. On examination, white and red blood-corpuscles, epithelial cells, hyaline casts, pus cells, and various micro-organisms, are found. The reaction is said to be always alkaline. The course is very slow, but ultimately death results from debility and exhaustion in from two to five months (Lucet, Hess).

In the acute form described by Leblanc, in addition to constitutional symptoms, the animal is observed to lie down constantly, the muzzle is hot and dry, the eyes are retracted in the orbits, micturition is painful, the urine often turbid, albuminous, sometimes of a reddish tint; fever is slight or may be absent, progression is painful, and later on the animal is unable to maintain the standing posture. Death may occur in from five to nine days, or the case may develop into the chronic type.

It must be remembered that the disease may be complicated with metritis, cystitis, or vaginitis.

Cases of recovery from the affection are said to be unknown. Death is always the result, although the course of the disease may be very prolonged.

Differential Diagnosis.—The differentiation of the disease from ordinary cases of chronic nephritis and pyelitis presents much difficulty. According to Leblanc, the bacteriological examination of the urine, although of assistance, is not sufficient to base a definite diagnosis on. It also requires to be differentiated from cystitis, the various forms of hæmaturia, and from tuberculosis of the kidney. According to Friedberger and Fröhner, the majority of cases of chronic nephritis in cattle are in reality pyelonephritis.

According to Leblanc, in advanced cases exploration *per rectum* will enable the practitioner to recognise the presence of the enlarged left kidney, and in all cases the thickened ureter may be felt as a stout cord extending from the kidney to the bladder. The greater frequency of the disease in cows after parturition should be remembered.

Treatment.—Therapeutical measures of any kind are useless in this disease, and when a definite diagnosis can be arrived at destruction of the affected animal is the most advisable course to adopt.

Prophylactic measures should be adopted with a view to avoid possible sources of infection; hence isolation of suspicious cases should be carried out, also disinfection of premises, etc.

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MALIGNANT CATARRHAL FEVER OF BOVINES

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Synonyms.—Malignant catarrh ; Gangrenous coryza.

French : Coryza gangreneux ; Mal de tête de contagion.
German : Katarrhalfieber ; Kopfkrankheit.

Definition.—Malignant catarrhal fever may be defined as an infectious malady affecting bovines, and characterised by acute inflammatory changes in the mucous membranes, particularly those of the respiratory and digestive tracts.

General Remarks.—In prebacterial days it was believed to be glanders of the ox, and was described under that name ; but at the present day it is almost superfluous to say that it bears no relation to glanders. The disease is of infrequent occurrence in Great Britain, and relatively unimportant ; but it is constantly present on the continent of Europe, nearly all the countries of which have periodically contributed records of outbreaks.

The contagious character of the malady is seldom very evident, the great majority of the cases occurring sporadically without traceable source of contagion ; but enzoötic outbreaks, though of less frequent occurrence, are not uncommon, the disease being limited usually to a particular pasture or farm. In certain districts of France and Germany it appears to be almost constantly present, but the cases occur at irregular intervals, and rarely succeed one another in such a manner as to convey the impression of contagion. It appears to be more prevalent in the spring than at other seasons.

Etiology.—There is little doubt that the disease is of microbic origin, but it has not been shown that any particular organism is the essential causal factor. Streptococci and short bacilli have been demonstrated in the exudates and false membranes on the pharynx, larynx, and in the lungs. Nocard showed the presence of a bacterium of the fowl cholera type in the lesions on the mucous membranes of the nose and larynx, but there is no evidence to show that these organisms cause the disease. Leclainche observed the presence of a bacillus belonging to the colon group in the small intestine and related lymphatic glands of affected animals, which he regarded as the essential causal agent. He concluded from his experiments that the organisms elaborated an actively toxic substance in the intestine and neighbouring lymphatic glands, the absorption of which was responsible for the general symptoms, whilst its elimination by the mucous membranes produced the initial alterations shown by them, the necrosis and other changes in the membranous lesions being secondary, and due to the invasion of pyogenic and other organisms. Feeding experiments with Leclainche's organism were negative, whilst subcutaneous or intravenous inoculation determined a rapidly fatal intoxication. It may be said that all experimental attempts to reproduce the natural disease have failed, and that its cause remains unknown.

Exposure to cold, which has been suggested as the cause, has at most a predisposing effect, and debility occupies the same position. Insanitary conditions act similarly, and are favourable to the continuance and recurrence of the disease on the same premises. The irregular and frequently long intervals between successive cases suggest that the virus is possessed of considerable powers of resistance. Direct contagion from animal to animal by contact does not occur, and although it is generally assumed that infection takes place by way of the alimentary tract, there is no evidence to support this belief.

Morbid Anatomy.—The post-mortem appearances present variations according to the localisations of the disease and the period of illness at which the examination is made. In the earliest stages the mucous membranes show deep congestion; the blood is dark and rather thick, but fluid. The heart is dilated with blood, and its muscle is soft and flabby. The flesh presents all the appearances of a fevered carcass. At a later stage the mucous membranes show congestion, infiltration, yellowish diphtheritic false membranes, and irregular ulcerous patches, bathed in purulent material, where the latter have become detached. Complications in the form of broncho-pneumonia or gangrenous pneumonia may be observed. Acute gastro-enteritis may be present, the mucous membranes of the stomach and intestines being deep red or nearly black in colour in patches or over large areas, and they commonly present a diphtheritic appearance, particularly on the summits of the folds of the mucosa. The mesenteric glands are enlarged, congested, and dark in colour. The kidneys may be congested or inflamed, and the bladder contains an albuminous and sometimes bloody urine. The eye shows intense conjunctivitis and infiltration of the cornea; and acute iritis may also be observed, with the presence of an exudate in the anterior chamber.

Symptoms.—The disease is manifested by symptoms common to the majority of acute general affections. The temperature rises from 3° to 6° F.; the pulse is rapid, full, and bounding; the respirations are increased in frequency; the appetite is in abeyance; and rumination is infrequent or completely suspended. The secretion of milk is markedly diminished or arrested altogether, the muzzle is dry, the coat harsh and staring, and the animal stands with back arched and head depressed. The intensity of the constitutional disturbance bears a definite relationship to the severity of the membranous lesions, and since the latter may present material variations, both in degree and in localisation, several forms of the disease have been recognised and described as a result. These do not differ essentially from one another, but merely in their respective severity.

The mucous membranes of the mouth and of the nose show the earliest and most pronounced changes. On examination in the earliest stages of the attack they are bright red and congested, but they soon become covered in patches with a yellowish or yellowish-green exudate mixed with epithelial débris, and assume a deep red or purple tinge. These exudates or false membranes are soon detached from the mouth, leaving behind ragged, irregular ulcers. Their presence gives rise to profuse salivation, difficulty in prehension, mastication, and deglutition, and the mouth quickly acquires a fœtid odour. On the nasal mucous membrane they remain attached for a longer period, and are more readily seen. They give rise to a profuse sero-mucous, and later a purulent fœtid, discharge, and they bleed readily if handled. The swollen tumefied mucous membrane and the adherent exudates mechanically obstruct respiration, which becomes sniffling or stertorous, and sometimes dyspnœic, owing to extension of the condition to the larynx and trachea. The membranes of the horn cores and of the facial sinuses are similarly affected; the horns are hot and painful on handling, and at a later stage may be cast. The head is carried low, and continuously rested on any available support. A cough develops, frequent and painful, and may result in the ejection of masses of the exudate, and not uncommonly in epistaxis. The respiratory symptoms may be conspicuously intensified by the development of broncho-pneumonia, and marked dyspnœa follows the spasmodic fits of coughing. Similarly, the implication of the gastric and intestinal mucous membranes leads to a modification in the symptoms. Constipation is in most cases present at the commencement of the attack, but it is succeeded by diarrhœa, profuse and fœtid, with blood-tinged motions, and accompanied by much pain and straining.

Ocular symptoms are usually pronounced, and are manifested at an early stage of the disease. The eyelids are swollen and partially or completely closed, the conjunctiva is intensely congested, and the cornea acutely inflamed and opaque. In cases of longer duration ulceration of the cornea is not infrequent. Pus very soon appears, mixed with the tears, which flow profusely over the cheeks.

Skin lesions are almost constantly present. They are to be found on the neck, inside the thighs, on the teats, udder, and generally on the thinner parts of the skin. They assume the form of clearly circumscribed hard nodules the size of a pea, or slightly larger, with loss of hair and desquamation of the surrounding skin. They sometimes soften and become converted into pustules.

The urino-genital mucous membrane sometimes, though infrequently, manifests changes very similar to those seen in the mouth and nose. Nephritis may develop, with suppression of urine, or the diminished secretion may contain albumin, blood, and inflammatory products from the kidneys.

During the course of the disease the animals rapidly lose condition and strength, and spend the greater part of the time lying, or completely take to the ground. In the more acute cases a fatal termination supervenes in the course of from three to seven days, but in those of a less severe type the disease may have a duration of three or four weeks, and ultimately end fatally. Not infrequently the less severe cases are somewhat erratic in their course, and show alternate improvements and remissions of the symptoms.

Diagnosis.—Rinderpest is the disease to which malignant catarrh bears most resemblance, and it is with that affection that confusion is most likely to arise both during life and on post-mortem examination. The greatest doubt may exist with a first case, where the disease is not of frequent occurrence, and more particularly if several animals are simultaneously attacked and the intestinal symptoms pronounced. The very decidedly contagious character of rinderpest, its short incubative period, its rapid spread to in-contacts, and its epizoötic occurrence, serve to distinguish the two affections. In malignant catarrh the ocular and respiratory symptoms are usually predominant, whereas in rinderpest the symptoms of alimentary disturbance are always most pronounced.

Prognosis.—The condition is very grave, and few of the affected animals recover. The mortality has been variously estimated at from 50 per cent. to 95 per cent., and probably the latter figure more nearly approaches the mark than the former. A prolonged convalescence is necessary, and the animals which reach the convalescent stage are generally economically worthless. They are emaciated, the milk secretion is arrested, and they improve very slowly in condition. Loss of the horns and sometimes of the hoofs, or of both, are not infrequent sequelæ, and blindness may follow the ocular lesions.

Treatment.—Treatment of this affection has not hitherto been productive of satisfactory results. There are no specific remedies, and treatment must be entirely symptomatic. Bleeding in the earliest stages has been recommended, in the belief that it exerts abortive effects, but there does not appear to be any evidence that it is capable of limiting the severity of the disease, and the malady is one in which bleeding would appear to be contra-indicated. In the early febrile stages antipyretics in the form of quinine, salicylates, acetanilid, etc., in combination with stimulants, may be prescribed. Frank, Hink, and Zundel, quoted by Friedberger and Fröhner, have claimed successful results from the administration of creolin and carbolic acid in doses of 5 to 10 c.c., in combination with the local application of these agents in the form of washes, inhalations, and ointments. Collargol has been employed in Germany in doses of ½ to 1 gramme, administered intravenously in 1 per cent. solution daily, but without producing very satisfactory results, and the same remark applies to the use of intratracheal injections of iodine. Moussu strongly advocates the injection of physiological saline solution. Attention must be paid to the comfort of the animal and the hygiene of its surroundings. The strength must be maintained by the exhibition of nutrients, and demulcents, astringents, and stimulants, may be administered as the case indicates.

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LIGNIÈRES' MALIGNANT ŒDEMA OF OXEN

By HENRY GRAY, M.R.C.V.S.

Synonym.—Mancha (Argentine).

Definition.—A disease of cattle termed in the Argentine "mancha," signifying a stain, characterised by a severe lameness and an intense swelling of one of the fore or hind limbs, and due to a bacillus which readily grows in bouillon exposed to the air; biologically the organism stands midway between that of black-quarter and that of malignant œdema.

History.—This malady was studied by Lignières and Bidart in 1901 and 1902 in Argentine cattle, in which, according to these authorities, black-quarter is unknown. Probably it occurs in other countries. Many workers in animal pathology in various parts of the globe have, from time to time, met with micro-organisms in œdematous diseases of the ox not corresponding to the usual biological character of the bacillus of black-quarter.

Bacteriology.—The bacillus of mancha is found in various forms in the tissues or in cultures. The rods are short, being 6 μ to 8 μ long and 1 μ wide, and usually single. The vibriones or mycelia are 40 μ or more in length, sometimes composed of unequal articles. The thick fusiform forms resemble many yeasts. Some bacilli show a spore, either in their centre or rather at one or both of their extremities. When recently drawn from the serosity of the still warm carcass, and in cultures of eight to ten hours' duration, the bacillary or mycelial forms are endowed with movements of translation, especially undulatory and serpentine. Atmospherical oxygen soon arrests the movements of these microbes.

The simple methods of staining, as well as those of Gram-Nicolle or Claudius, colour the microbial protoplasm very well, but leave the spores clear. After coloration of the cilia these are found to be very numerous, and distributed at the extremities, as well as on the sides of the bacilli.

The bacillus easily kills the guinea-pig, young rabbit of one to three months, rat, mouse, cat, sheep, goat, calf, horse, pigeon, and more rarely the fowl. The adult rabbit, ox, pig, and duck, are usually resistant, but always show, especially with the muscular juice, more or less severe lesions.

The muscular juice constantly exhibits a greater virulence than that of the best cultures.

Susceptible Age and Mortality.—The disease exclusively attacks calves of six to ten months, whilst adults remain healthy. It affects 2 to 4, rarely 6, per cent. of the total. Recoveries are quite exceptional.

Symptoms.—The first symptom to attract attention is an intense lameness of one of the fore or hind limbs. In this case, when one approaches the animal to examine it, one finds the upper part of the limb, especially the shoulder or thigh, more or less swollen. At the commencement this swelling is ill-defined, œdematous, hot, and painful, of a fairly homogeneous consistence, and the animal is still able to walk. Later on the swelling increases in size, and, without losing its diffuse appearance, the tumour becomes crepitant; in its centre it sometimes gives on percussion a peculiar tympanic sound. There is an evident formation of gas—a true fermentation of the tissues. At this period the sensibility and heat of the tumour have diminished, and the lesion seems to be slightly improved. However, the animal is absolutely powerless and unable to walk; it remains standing in one position, or lies extended to its full length on the ground. Its general condition is far from being normal; it is dull, depressed, and has no appetite; the pulsations and respirations are accelerated, and the temperature attains or even goes beyond 106° F. Shortly before death tympany and muscular tremors appear.

The tumours may also make their appearance on the neck, sides, or even on the loins, where they always rapidly develop. They may, furthermore, be deeply situated in the ilio-spinal region, and cannot be detected.

Course.—The disease generally runs its course and ends in death within twenty-four to forty-eight hours. Before death the animal remains on the ground, and cannot get on to its limbs when an attempt is made to do so. The respiration is jerky, and the temperature sometimes descends below 98·6° F., death usually taking place without a struggle.

Lesions.—When the skin is removed little abnormal is noted beyond the areolar tissue over the tumour being infiltrated with a red gooseberry-like serosity, in the form of a more or less abundant gelatinous œdema. The whole surface of the tumour leaves on the superimposed skin a large dark stain: hence the name "mancha" given to the malady.

When the tumour is cut into, it is crepitant on pressure of the section, and the muscles situated on the circumference are found to be infiltrated and of a brownish-red colour. Towards the centre the muscles are rather dry and friable; the fibres separated by the gas have a purplish-brown, almost blackish appearance, indicating that they have undergone the vitreous degeneration of Zencker. The whole cadaver gives off a strong odour resembling that of rancid butter. On exposure to the air, the dark colour lessens and becomes redder on the sections of the muscles, save towards the centre. The neighbouring glands are hypertrophied and markedly infiltrated. The muscles and glands in other parts of the body do not appear altered. The spleen is larger and softer than usual, the kidneys are dark, their capsule is easily stripped off, and on section one can distinguish the greyish tint of the cortical layer. The urine, although always dark in colour, contains neither hæmoglobin nor red corpuscles.

The liver and also the intestines appear normal. Sometimes, on the posterior portion of the latter organs, however, a sero-sanguinolent infiltration is observed; the omentum is then punctated with a few hæmorrhagic spots, as if the infection took place this way. The peritoneal, pleural, and pericardial serous membranes are normal, or sometimes show ecchymoses. The cardiac muscle has a boiled appearance, is friable, and of a greyish tint; it often exhibits hæmorrhagic patches. The blood, which is generally black when it escapes from the vessels, coagulates and becomes lighter in colour on exposure to the air. The lungs are normal.

Diagnosis.—The symptoms and lesions differ from those of anthrax. Examination of the blood and spleen will determine the diagnosis.

The cultures of mancha, black-quarter, and malignant œdema, have no distinctive value. In mancha, however, the culture is easier obtained in bouillon, when exposed to the air, than in the other two diseases. In the various anaërobic media the bacillus of mancha is also grown with little difficulty; it generally multiplies in greater abundance than in the case of black-quarter. In other respects the cultural characters are common to the bacilli of the three diseases.

The course of the malady, the character of the lesions, the rancid butter odour given off by the tumour, and the appearance of the microbes, would lead one to conclude the presence of black-quarter, but the experimental evidence demonstrates the difference.

The microbe of mancha, like that of malignant œdema, readily grows in the system in the form of long filaments, sometimes having unequal articles. On the surface of the liver they are always found to be very long in mancha, whereas they are short in black-quarter.

The virulence of the bacillus of mancha is greater than that of black-quarter for the rat, dog, young rabbit, fowl, horse, and even ox. For the adult ox the bacillus of black-quarter is incomparably more virulent than that of mancha and of malignant œdema. Subcutaneous injections of the bacillus of black-quarter often kill adult cattle, whereas the microbe of mancha or of malignant œdema hardly ever causes death under these conditions.

The bacillus of mancha is not very virulent for the adult rabbit, and in this respect it resembles that of black-quarter. On the other hand, the bacillus of malignant œdema is much more virulent for the fowl than mancha. The lesions are more often generalised in malignant œdema than in mancha.

Animals vaccinated against mancha are immune to malignant œdema, and *vice versâ*. It is not always the same in the case of mancha against black-quarter.

Immunity against black-quarter brought about by the black-quarter virus does not confer immunity against mancha. This has also been observed by Vallée; but immunity against mancha conferred by the mancha virus immunises against black-quarter.

The specific serum of mancha is very efficacious against the bacillus and virus of malignant œdema; less so for those of black-quarter.

The bacilli of mancha and malignant œdema are immediately agglutinated by one-thirtieth of the specific serum of mancha. With this degree of dilution it is very slow for black-quarter. With one-three-hundredth dilution, agglutination is slow and somewhat slightly pronounced for mancha and malignant œdema, but nil for black-quarter.

Treatment.—Immunity can be brought about on similar lines to those adopted in the case of black-quarter. A vaccine may be prepared according to the methods of Arloing, Kitasato, Kitt, or Leclainche and Vallée. A serum may also be obtained on the lines of Kitt, Leclainche and Vallée, or Arloing. Sero-vaccination is, furthermore, realisable. This study of Lignières and Bidart* is important, as it may lead the way for the investigation into the failure of black-quarter vaccinations in several parts of the world. In Chili, vaccination against so-called black-quarter has been an absolute failure, so much so that the Chilian Government has commissioned M. Lucet of France to proceed there and study the cause.

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* "Contribution à l'Étude de la Maladie connue en Argentine sous le nom de 'Mancha,'" par MM. J. Lignières et R. Bidart. Copious Bibliography (*Arch. de Méd. Expérim. et d'Anatomie Pathol.*, No. 4, Juillet, 1903).

SWINE FEVER

Synonyms.—Pig typhoid ; Pneumo-enteritis ; Red soldier ; The rose ; The purples ; Pig distemper, etc.

French : Pneumo-entérite du porc ; and Choléra hog.
German : Schweinepest.
Danish and Swedish : Svinpest and Svine diphteritis.
American : Hog cholera.

Definition.—Swine fever is a highly contagious eruptive fever peculiar to the pig, and caused by an ultra-visible virus.

HISTORY.

Although swine fever was not until comparatively recently isolated as a distinct disease of swine, it is most probable that it was represented in one or other of the deadly porcine epizoötics of even early times recorded in the history of animal scourges. There is definite knowledge that it existed on the continent of Europe almost a century ago. From Europe it spread to Ohio, in America, in the year 1832. The exact date at which the disease appeared in Great Britain and Ireland and the source of origin are not known ; but available evidence points to the disease being introduced prior to the year 1858 from the Continent. In that year cases of "red soldier" were frequently met with in swine imported from European countries. The disease was recognised in England by Professor Simonds in 1862, and by Professor Brown in 1863. In 1865 Dr. William Budd of Bristol investigated an outbreak at the Clifton Workhouse, and in a paper, read before the Royal Agricultural Society, minutely described the ulcerative and necrotic changes found in the alimentary canal, comparing the button ulcers to slices of Calumba root. About this time Professor Gamgee met with the disease in Edinburgh. From this date onwards the disease received considerable attention from English, American, and Continental authorities, the chief investigators in England being Axe (1875), Klein (1877), Walley (1885), Brown (1866), McFadyean (1888, 1891, and 1897), and in America, Law, Detmers, Salmon, Theobald Smith, Osler, Billings, Welsh, De Schweinitz, Dorset, Bolton, and McBryde; on the Continent, Harms, Bruchmuller, Bollinger, Zundel, Schütz, Galtier, Nocard, Bang, Jensen, Silberschmidt, etc.

The name "swine fever" was first given to the disease by the late Sir George Brown, and in a Parliamentary Report on the subject in Great Britain, in 1886, he described the symptoms and post-mortem lesions of "swine fever." Prior to this the disease was described as "pig typhoid" by Axe and Budd, both of whom thought it was identical with typhoid fever of man. In the year 1878 it was first scheduled as a contagious disease in the Diseases (Animals) Act. From 1878 to 1893 various orders were made, empowering local authorities to deal with the disease by slaughtering diseased and in-contact swine, disinfecting premises, restricting movements, and regulating the pig traffic. In 1893 the Board of Agriculture in England, and the Veterinary Department in Ireland, took over control of the disease from the local authorities, and are still engaged in an earnest endeavour to banish it from the British Isles.

GEOGRAPHICAL DISTRIBUTION.

It is a very widely spread, if not universally distributed, disease. It is certainly the most widely spread of epizoötic swine diseases, and causes enormous loss in all European countries, America, and Great Britain and Ireland. Cape Colony and New Zealand and the Isle of Wight are reported free from it at the present time.

ANIMALS SUSCEPTIBLE.

The disease is entirely peculiar to the pig. Not only is it never seen in any other animal naturally, but all attempts to transmit it experimentally have failed.

PORTALS OF INFECTION.

The disease can be set up by inoculation and ingestion, and also readily by association with diseased animals and from infected premises, litter, etc. It is highly contagious and absolutely never sporadic. There is no evidence to show that the virus ever multiplies outside the animal's body. The fæces are virulent, and it is probable that they are mainly the vehicle of infection.

ETIOLOGY.

The evolution of the etiology of swine fever forms a history which is probably unique in the annals of veterinary literature. Investigations into the etiology of the disease, although fairly extensive, had been practically barren of results until the year 1885, when Salmon, in America, announced the cause to be a small bacillus, which he called the *Bacillus choleræ suis.* In this country the organism is generally called the *swine fever bacillus.* The work of Salmon and Theobald Smith connecting this bacillus with the disease was corroborated by many other competent authorities, and it was regarded as settled beyond dispute that the *B. choleræ suis* was the sole cause of swine fever. McFadyean in 1895 investigated the disease in England, and found the causal organism to be a bacillus very similar to the American *B. choleræ suis.*

In 1903, however, Dr. E. A. de Schweinitz, of the American Bureau of Animal Industry, while endeavouring to produce an antitoxin for the disease by hyper-immunising swine with cultures of the *B. choleræ suis*, was led to question the correctness of the prevalent view that swine fever was caused by Salmon's bacillus, as he found that organism did not protect animals against the disease. He instituted and carried out experiments which were directed to test the claims of the *B. choleræ suis* as the sole cause of the disease. His death, however, before his experiments were complete prevented further research on his part.

The work was then taken up by Dorset, Bolton, and McBryde, of the Bureau of Animal Industry, by whom a series of important experiments were carried out in the years 1903 to 1905. The result of these experiments went to show that genuine contagious swine fever is caused by a filtrable virus, which exists in the blood of infected swine, and that the *B. choleræ suis*, although present in most cases of the disease, probably only plays the part of a secondary invader. These conclusions regarding the etiology of swine fever, being of a revolutionary character, attracted widespread attention among scientific workers throughout the world, many of whom carried out experiments, which confirmed the American results.

It is now universally accepted that a filtrable ultra-visible virus is the true cause of swine fever, and that the so-called swine fever bacillus, or B. choleræ suis, when present in the blood and tissues of pigs suffering from the disease, is probably a secondary invader, but an important accessory factor, most probably responsible for the characteristic lesions of the disease.

The rôle which the so-called swine fever bacillus plays in the disease has been the subject of much discussion. It is still a fact that the bacillus is constantly associated with the disease, and can usually be demonstrated in the lesions, mesenteric glands, and sometimes the blood. It is also possible by feeding with a fairly large quantity of cultures to set up a disease indistinguishable from genuine swine fever in lesions, but differing from it in the fundamental fact that it is not contagious, and that recovery from it does not confer immunity for the filtrable virus.

The most generally accepted explanation of the part played by the bacillus is that it is a normal inhabitant of the alimentary canal (a fact which has recently been demonstrated by Dr. Uhlenhuth, of the Imperial Sanitary Office, Berlin), but that ordinarily it is devoid of pathogenic power, and that it is only when the body is invaded by the filtrable virus that pathogenic powers are conferred on it. The point against this is that feeding cultures of the bacillus will set up lesions, without the aid of the filtrable virus, but it should be remembered that in these culture-feeding experiments a comparatively large quantity of pure culture was used, and that it was probably the immensity of a single dose that enabled the bacillus to become pathogenic.

BACTERIOLOGY.

The so-called swine fever bacillus, as isolated from lesions from affected pigs in this and other countries, although apparently identical in morphology, appears to differ somewhat in culture and virulence for the small laboratory animals. According to McFadyean, the organism accountable for the lesions in this country is a small motile bacillus, from 1 to 2 μ long, and about 0·5 μ broad, with rounded ends. It does not form filaments or spores. It stains readily with the aqueous solution of the basic aniline dyes, but not by the method of Gram. It is an aërobe, and grows well in bouillon, forming a moderate turbidity with no surface scum, and on agar forming a thin translucent film. The most certain place to find the organism is the mesenteric glands of a pig, killed at the height of the disease. The virulence of this so-called swine fever bacillus appears to vary a good deal. It is usually fatal to guinea-pigs in doses of 1 to 2 c.c. broth culture intraperitoneally, but not subcutaneously in moderate doses. It is less pathogenic for the rabbit than the guinea-pig. If a quantity of virulent culture is given to a fasting pig, death is a common result after a period which varies with the quantity of culture administered.

PERIOD OF INCUBATION.

The period of incubation is about five to ten days. A rise of temperature will generally be found about the fifth day, and visible signs of illness appear about the eighth or tenth day.

MORBID ANATOMY.

The carcass of a pig dead from swine fever may or may not show emaciation and skin discoloration. In general the lesions may be divided into *three* types—viz., the *hæmorrhagic,* the *ulcerative,* and the *diphtheritic,* which represent different forms of the disease, but may all be found present in the same animal. As a rule, however, the hæmorrhagic lesions are characteristic of the acute form, and the ulcerative and diphtheritic ones of the less acute and chronic forms.

ALIMENTARY CANAL.—The alimentary canal is the constant seat of lesions. In acute cases the only lesions seen may be an intense gastro-enteritis, or petechiæ and ecchymoses may be observed on both the serous and mucous membranes. In less acute forms and in chronic cases the lesions are confined to the large bowel, but are often found in the small intestine, and less frequently in the stomach and mouth. Sometimes the whole of the large bowel is involved, but the parts most frequently attacked are the cæcum, the ileo-cæcal valve, and the first foot or two of the colon. Both the ulcerative and diphtheritic type of lesions are found here, but the ulcerative is by far the commoner of the two.

The so-called ulcers are not, strictly speaking, ulcers, but circumscribed areas of necrosis ; their surface is flat, and they are often concentrically ringed. The colour of the ulcers may be grey, yellow, or black, and the wall of the bowel is generally more or less thickened in their neighbourhood. In cases which survive, the necrotic tissue is ultimately shed, leaving a true shallow ulcer, which in time becomes covered over with epithelium, leaving a permanent smooth scar devoid of secreting glands.

The microscopic picture is that of necrosis of the mucous, and often of the submucous, coats of the bowel. Ulcers very similar to those seen in the bowels are sometimes met with in the stomach, pharynx, and tongue, and similar lesions are quite common in the skin of the legs, and lips in chronic cases.

In the *diphtheritic* lesion, which is usually preceded by enteritis, a greyish or yellowish exudate is formed on the surface of the mucous membrane of the cæcum and colon, either as isolated spots, patches, transverse ridges, or as a continuous layer. It becomes firmly adherent to the mucous surface, which undergoes necrosis. Frequently the wall of the bowel becomes thickened. Under the microscope the diphtheritic deposit is seen to be composed of a croupous or fibrinous exudate, accompanied by more or less necrosis of the underlying mucous membrane.

LYMPHATIC GLANDS.—In acute cases the glands in the abdominal and thoracic cavities are enlarged and hæmorrhagic. In the less acute cases the intestinal, mesenteric, and inguinal glands may be those principally affected. In chronic cases the lymphatic glands may not exhibit any inflammatory change.

Heart.—In acute cases the epicardium generally shows petechial spots. In chronic cases no lesion is seen.

Lungs.—In acute cases petechiæ are seen on the surface of these organs, and areas of broncho-pneumonia and pulmonary collapse are often met with. In nearly all outbreaks in this country cases of lobar pneumonia with large centres of necrosis in the hepatised areas are seen.

Spleen.—In acute cases the spleen is somewhat enlarged and engorged with blood, and petechiæ are seen on its surface. In less acute cases it may contain areas of necrosis. In old chronic cases it is generally smaller than normal, and its connective tissue is increased.

Liver.—This organ may exhibit areas of miliary necrosis.

Kidneys.—In acute cases hæmorrhagic changes are nearly always seen. In the chronic form the kidneys appear normal.

Bladder.—In acute cases hæmorrhagic lesions in the shape of petechiæ and ecchymoses may be found in this organ.

SYMPTOMS.

The symptoms vary according as the disease shows itself in the acute, subacute, or chronic form. An outbreak may be ushered in with cases of an acute type, killing off the most susceptible animals, followed by the subacute and chronic forms, with a fairly large proportion of recoveries, Or, on the other hand, an outbreak may begin and end with acute cases and a high death-rate. It seems to depend on the virulence of the virus and the susceptibility of the animals. Young swine are more severely and rapidly attacked than old ones, which often escape.

In *acute cases* the animals refuse food, bury themselves in the litter, or lie huddled up in the corner of the sty. There may be a catarrhal discharge from the eyes, and the eyelids are often swollen. The temperature is high, running up to 106° or 108° F. Constipation is at first present, and may, if the animal lives long enough, be followed by diarrhœa. Discoloration of the ears, abdomen, and thighs may or may not appear. There is great prostration, and the animals gradually become comatose, and die after an illness of from two to four days.

In *subacute cases* the more typical clinical symptoms are seen. Only a proportion of the herd is ill at any time, and the period of illness is much longer thani n the acute type. A rise of temperature from 103° to 107° F. is the first symptom observed; this is followed quickly by more or less prostration, shivering, and almost complete loss of appetite. When the lungs are affected, there are the usual pneumonic symptoms—accelerated respiration, etc. The animals lie most of the time huddled up in their beds; they frequently shiver and show an irritable disposition, never resting quietly for long, but continually shuffling and squealing. When they do emerge from their bed, the gait is unsteady, the hind-quarters swaying from side to side, indicating a loss of muscular power, which occasionally ends in partial or complete paralysis. Thirst is a constant and well-marked symptom; the animals can be noticed sucking up any available fluids from the floor of the sty. A sticky discharge from the eyes is a frequent symptom, the eyelids often being glued together.

Although at the outset, when the temperature is high, constipation is present, and in some cases may persist throughout the course of the disease, it generally gives way to diarrhœa, which is a very common symptom. The fæces have a very offensive smell, and in colour may be yellow, brown, black, grey, or green. They are thick and viscid at first, but later on, as the disease progresses to a fatal termination, they become thin and watery. Vomition, although sometimes seen, is not very common. Abortion, however, is a common occurrence in affected sows. A short husky cough is also a constant symptom, and is best detected by entering the sty and waking up the affected animals, which will generally excite them to cough. This cough does not depend upon lung trouble for its origin, but most probably originates in a reflex manner from the stomach. The cough is of the laryngeal type, and often resembles an attempt on the part of the animal to clear a foreign body from its throat. An attack of coughing very often ends in vomition or in retching.

As to skin symptoms, a rash may be present or absent. It is, however, very common to find inside the thighs and along the abdomen a pin-head rash, caused by small petechial hæmorrhages, which do not coalesce, and upon which small brown or black scabs, or even ulcers, may ultimately appear. In prolonged outbreaks in unsanitary surroundings it is quite common to find "ulcers," or circumscribed areas of necrosis, on the skin of the lips and legs, especially on the outside of the hocks and knees; but these lesions, which resemble in appearance those found in the bowels, may be observed on any part of the body.

In the *chronic* form the symptoms are usually not very marked; the animals have a hard, stunted, unthrifty look, and the skin is of a pale ashy colour. The appetite is generally capricious, and there may be an intermittent diarrhœa. A husky cough may often be present; and the eyelids may exhibit a sticky, somewhat swollen appearance. Chronic cases have been met with where extensive ulceration of the bowel was revealed on post-mortem, although the animals showed no noticeable symptoms of the disease during life.

PROGNOSIS.

The attack is more severe and the death-rate far higher in young pigs and young "stores" than in old swine and sows. Death is the usual result in very acute cases. In less acute cases many may succumb fairly early in the outbreak, but a proportion always linger on for weeks, while others may become chronic cases and live for months. Generally in subacute outbreaks a fairly large percentage recover, while a number of animals exposed to infection show immunity. On the average, the death-rate in the acute outbreaks reaches 80 per cent., and in the subacute and chronic ontbreaks it is never higher than 40 per cent. From inquiries made by the Board of Agriculture in the year 1907, it was found that the average death-rate, taking one outbreak with another, was about 30 per cent.

DIFFERENTIAL DIAGNOSIS.

It is almost impossible to definitely diagnose the acute disease from clinical symptoms alone. A post-mortem examination, and often a bacteriological investigation, is necessary to distinguish it from acute *swine erysipelas*. Inoculation of susceptible swine with 2 to 5 c.c. each of "filtered" blood from the suspected animal is really the best test for the discovery of the disease. The expense attached to it, however, practically debars it from general use. In subacute cases a diagnosis in the living animal can be made with a fair degree of accuracy, although some cases in the early days of an outbreak may not show the usual clinical symptoms and characteristic lesions; but as the outbreak develops the symptoms become more pronounced, and some of the fatal cases reveal the characteristic bowel lesions, thus placing the diagnosis beyond doubt.

There is absolutely no diagnostic symptom; indeed, mature experience generally leads one to regard swine fever as a most protean disease. Still, a suspicious history, with a number of pigs ill, and symptoms such as more or less prostration, thirst, staggering gait, diarrhœa, or obstinate constipation, cough, gluing up of the eyelids, and a petechial rash on the under-surface of the body, justifies a positive diagnosis.

Diffuse skin discoloration is by no means a diagnostic symptom; it is frequently met with in minor ailments of the pig, and little importance should attach to it in forming a diagnosis.

Cough may also be present in tuberculosis and parasitic bronchitis.

In *tuberculosis*, however, the cough is deeper and stronger, and the affected animals do not show the same prostration as in swine fever.

In *parasitic bronchitis* the cough is sonorous and paroxysmal in attack, and the affected animals, although they may be anæmic and emaciated, do not show pronounced signs of severe illness.

In chronic cases it is most difficult at times to distinguish the disease from *tuberculosis, cirrhosis of liver, intestinal parasites*, etc. In all these cases the history of the illness must be carefully gone into, and a diagnosis arrived at by the process of exclusion. In this respect a microscopic examination of the fæces for parasites and ova, and the application of the tuberculin test will be helpful.

Generally speaking, one ought to suspect chronic swine fever if the history of the illness indicates that the animals were in any way exposed to infection, or had passed through a mild attack of the disease, and if they exhibit an unthrifty appearance, with a pale ashy colour, capricious appetite, intermittent diarrhœa, and a catarrhal condition of the eyelids, with or without a cough.

The Board of Agriculture believes that swine owners may reasonably be expected to suspect the existence of the disease under the following circumstances: (1) When a number of animals are dying; (2) when a number of animals are sick or unthrifty; (3) when periodic deaths are taking place, even if the other pigs appear healthy; (4) when a high mortality is noticed in sucking or newly weaned pigs; (5) when a number of pigs are sick or dying, with symptoms of pneumonia, diarrhœa, or what may appear to be acute swine erysipelas; (6) the fact of the suspicious symptoms appearing first in pigs which have been recently purchased, or in a sow which has been to the boar, or in pigs recently castrated, or in those which have been taken off the premises to a market, and, being unsold, have been brought back, should always increase the suspicion.

TREATMENT.

Good nursing is a far more important factor in the treatment of this disease than the administration of drugs. A comfortable warm bed, a well-ventilated sty and sloppy, milky food in small quantities and often, will certainly prolong life, and greatly assist in bringing about recovery. In the United Kingdom, however, the disease is scheduled under the Diseases of Animals Acts, and swine owners, their agents and *veterinary practitioners*, are legally bound to notify the existence of affected or suspected animals to the police. Under these circumstances curative treatment is practically ruled out, as the authorities generally slaughter off the affected and in-contact swine.

Treatment with serum from hyper-immunised swine has been recently tried in America, but so far without definite results. It seems probable, however, that large doses of serum injected in the early stages of the disease may have some curative effect.

PROPHYLAXIS.

By only purchasing and breeding from herds which are known to be free from the disease, avoiding markets, and by good sanitation and careful attention, it is possible to considerably reduce the risk of infection, and, if the disease does appear, to prevent it spreading. The Board of Agriculture recommends the following observances on this point to owners of swine:

1. Pigs which have been recently purchased, or which have been off the premises to a market and brought back again, should be kept isolated from the others for about a month, and carefully watched. The same rule applies to sows which have been to the boar, and to pigs which have recently been castrated by a practising castrator.

2. Sows which have survived an outbreak—that is to say, those which are known to have suffered from swine fever and recovered—should not be bred from, as there is a considerable amount of circumstantial evidence to show that they may possibly infect their young.

3. In the event of swine fever or suspicious symptoms appearing in any part of the premises, the pigs therein should, if possible, be kept rigidly isolated from the others, and have separate attendants, who should wear special boots and overalls in and about the sties. The experimental inquiries conducted by the Board show that sick and healthy pigs can be kept in close proximity without the latter becoming infected, if the sties are completely separated from each other, and if the attendance is performed by separate persons. It is true, of course, that under such conditions it will be difficult at any time safely to assert that the pigs in the other sties are not sickening for swine fever, and that they can be sent to market without risk. But inasmuch as it is advisable to stop breeding on infected premises, it will greatly tend to lessen the losses while this is in progress if proper measures for internal isolation are taken.

4. Once an infected sty has been cleared of its occupants, it should be thoroughly disinfected and left empty for about three weeks.

5. If there are rats on the premises, an effort should be made to reduce their numbers by the use of one of the viruses which are harmless to pigs.

This is advisable, not because rats suffer from swine fever, but because it is possible that they may convey infected material from one sty to another.

6. When affected pigs have been the subject of post-mortem examination, care should be taken that no material from the carcass comes in contact with healthy swine, and all assistants at the autopsies should properly disinfect themselves.

Vaccination.—The discovery that swine fever is not caused by the so-called swine-fever bacillus, or *Bacillus choleræ suis*, has naturally discredited any attempts to produce immunity by its use. If, however, as seems probable, this bacillus is the cause of the intestinal lesions, a vaccine which would protect against it would apparently be of great advantage in modifying the course of the disease, although, of course, not being able to prevent it.

Protective Inoculation with the "filtrable" virus has recently been introduced by Dorset, McBryde and Niles, of the American Bureau of Animal Industry, and since 1908 has been used fairly extensively in the United States. There are two methods employed, known as—(1) The "*Simultaneous*"; and (2) the "*Serum-alone*" method.

The *Simultaneous* method, which is specially recommended for herds which have not been exposed to infection, consists in injecting subcutaneously on one side of the body a suitable quantity of serum, and simultaneously on the other side of the body a small quantity of virulent blood from a pig affected with swine fever. By this method an active immunity lasting several months is set up.

The *Serum-alone* method consists in simply injecting a dose of the protective serum without the simultaneous use of virulent blood, and confers only a passive immunity lasting for about a few weeks, unless the inoculated animals are exposed to swine fever infection a short time after receiving the serum, in which case an active immunity is conferred.

The serum is obtained by injecting large doses of swine-fever blood into pigs which have natural or acquired immunity. After a week or so, when the inoculated pig has recovered from the effects of the injection, blood is drawn from the animal by cutting off the end of the tail. The blood drawing is repeated three or four times at intervals of a week, after which the hyper-immune pig is usually bled to death by section of the carotid artery. After each drawing the blood is defibrinated, mixed with a suitable antiseptic, and stored in sterile bottles. It is said to retain its potency for years.

According to Niles, the approximate dose of serum subcutaneously is, for sucking pigs, from 10 to 15 c.c.; for pigs weighing 30 to 200 pounds, from 20 to 30 c.c.; for old pigs, from 40 to 60 c.c. The dose of virulent blood is 1 to 2 c.c. During the autumn of 1907 Niles vaccinated about 2,000 hogs on fifty different farms. A large number of pigs were left unvaccinated to act as "controls." Both methods of vaccination were used, and the local conditions varied widely. The herds consisted of—(1) those in an infected district, but themselves free from disease; (2) those known to have been exposed by contact with sick animals, but which had not developed disease at the time of inoculation; (3) herds in which swine fever existed, and pigs were sick and dying at the time of inoculation. Attempts at isolation or disinfection were entirely disregarded. The results were that in herds where the disease appeared subsequent to inoculation practically all of the inoculated animals remained well, while more than 65 per cent. of the "control" animals died. In herds which had been exposed, but were apparently well at the time of inoculation, 4 per cent. of the inoculated animals died, while approximately 90 per cent. of the "controls" succumbed. In herds where the disease existed at the time of inoculation, and where much success was not expected, 13 per cent. of the inoculated animals were lost, whereas 75 per cent. of the "controls" died.

It is important to note that as the result of their experiments the American authorities believe that pigs vaccinated by the serum-simultaneous method do not communicate disease to others unless they themselves become sick, and that it is not necessary to produce illness by this method in order to secure an immunity lasting for at least three months. Indeed, if a sufficient dose of serum is given, pigs are not injured by the serum-simultaneous vaccination.

PUBLIC HEALTH.

All carcasses showing skin discoloration, congestion of the lymphatic glands, or hæmorrhagic lesions, are unfit for food, and should be condemned, buried, or cremated. The carcasses of mild, chronic, and recovered cases often present a normal appearance, and there is no reason why they should not be consumed. The disease is not communicable to human beings.

SWINE PLAGUE

Synonyms.—Pig pneumonia ; Swine septicæmia.

French : Pasteurellose du porc.
German : Schweineseuche.
American : Septicæmia hæmorrhagica suis.

In Continental countries and in America this disease has for years past been represented as a most serious contagious disease of swine, responsible for immense losses. Its cause is ascribed to a small bipolar bacillus indistinguishable morphologically and culturally from the fowl cholera bacillus, although its virulence for animals is said to differ somewhat from that organism. The disease is represented as occurring in acute and subacute forms. In the acute form it is septicæmic in character, killing the animal after a very short illness, and leaving no characteristic lesions. In the subacute form the animal may survive for weeks, and in this case there is always extensive lobar pneumonia, with necrosis or caseation. In a large percentage of cases it is admitted there is also congestion, petechiæ, ulceration, or diphtheresis in the bowel. The symptoms are indistinguishable from those of swine fever with pneumonia complications.

Experience in this country goes to show that this so-called swine plague does not exist as an independent disease. It appears to be met with only in association with swine fever. American and German opinion is at present also advancing towards this view, and it now seems fairly generally held that swine plague is probably caused by the filtrable virus of swine fever, and that the bipolar organism only plays a secondary rôle in producing lung lesions similar to that played by the *Bacillus choleræ suis* in producing bowel lesions. Like the *B. choleræ suis,* the swine plague bacillus is a normal inhabitant of the healthy pig, and its pathogenic power, like the former organism, seems to depend on the invasion of the body by the filtrable virus.

It often happens in this country that at the beginning of an outbreak of swine fever, or during its course, cases are met with which exhibit the pneumonic symptoms and reveal the lung lesions of swine plague, but invariably in such outbreaks typical swine fever bowel lesions are also found in a large percentage of the victims. However, if there is ever any doubt about the diagnosis, subcutaneous inoculation of a susceptible pig, with 1 or 2 c.c. of filtered blood from the suspected animal, will settle the matter definitely.

SWINE ERYSIPELAS

Synonyms.—Red soldier; Blue disease; The purples; Swine distemper; Swine typhus; The rose; Nettle-rash; Diamonds; Pig measles, etc.

French : Rouget du porc.
German : Schweine-Rothlauf.
Danish : Anthracoid erysipelas, or red sickness.

Definition.—It is an infective disease of swine caused by a specific bacillus, and characterised in the acute form by fever, discoloration of the skin, hæmorrhagic visceral lesions, and in the chronic form by verrucose endocarditis and arthritis.

History.—Swine erysipelas was at one time confounded with swine fever, but for a great many years past they are recognised by authorities as distinct diseases. It is said to have been first regarded as a specific disease of swine in France about the year 1846. It was not, however, until the early eighties that the investigations of Löffler made its etiology perfectly clear, and firmly established its claims to be recognised as an independent disease.

Swine erysipelas may have existed in this country for ages, but it was until comparatively recently mixed up by some practitioners with swine fever. There seems, however, little doubt but some of the outbreaks of what were called "red soldier" were cases of swine erysipelas. Cartwright, in 1847, was the first in this country to describe the chronic form, without, of course, knowing the true nature of the disease. Bang, in 1888, and McFadyean, in 1891, found the causal bacillus in the chronic heart lesions, and thus were able to connect these lesions with the disease.

Distribution.—On most parts of the Continent the disease is prevalent in epizoötic form, and is responsible for a serious mortality among pigs. It is scheduled as a contagious disease in Austria, Denmark, France, Hungary, etc. In these countries and in Germany many thousands of pigs succumb to its ravages annually. Although in this country the disease is not constantly prevalent in the epizoötic form, still serious outbreaks of a very acute type are not infrequently met with. The most serious of these occurred in the Chatteris district of Cambridgeshire in the year 1905, when the disease attacked most of the pigs in a strip of Fen country four miles long by one mile broad, with a fatality of 80 per cent. The chronic form is constantly met with in this country. The disease is said not to exist in the United States.

Animals Susceptible.—It is essentially a disease of the pig. So far as is known, the pig is the only domestic animal to contract the disease naturally. Many cases have been recorded in man, who is susceptible by inoculation. As a rule, however, its effects on man are not very serious. The disease in him is usually contracted through a wound at a post-mortem examination, and it generally remains confined to an inflammatory lesion of the hand or arm.

Young pigs under the age of three months, and old swine are comparatively insusceptible, the vast majority of the cases occurring in animals from four to eight months old. In outbreaks where the fatality runs high among pigs of this age, it is not uncommon for the young pigs and brood sows to entirely escape. At the same time it should be noted that swine of any age are not immune, and cases have been recorded in brood sows and sucking-pigs.

The susceptibility is also said to vary somewhat with the breed, as Continental authorities assert that highly-bred, imported English pigs are more susceptible than the native breed. This, however, is just as likely to be due to the virulent nature of the disease in Continental countries as to any innate susceptibility in breed.

Although the disease is not seen naturally in other animals than the pig, the mouse is very susceptible to inoculation, death usually taking place in from three to five days; similar remarks apply to the pigeon. Subcutaneous inoculation of the rabbit usually induces a local inflammatory lesion, and rarely death. The guinea-pig is very resistant. Horses, cattle, sheep, dogs, and cats are immune.

Portals of Infection.—Ingestion is commonly regarded as the natural method of infection, but the results of experimental feeding or inoculation are very uncertain, especially the results of feeding. The bacilli recovered from natural cases in the pig seem to vary very much in virulence, as tested experimentally on mice or pigs. As a rule it is most difficult to infect pigs with bacilli from the chronic cases which are so common in this country.

BACTERIOLOGY.

The causal organism is a slender cylindrical bacillus, with rounded ends, measuring about 1·5 μ in length, and about 0·2 μ in breadth, and is morphologically identical with the bacillus of mouse septicæmia. It *stains* excellently with Gram, Gram-Weigert, Claudius, and any of the simpler stains, especially dilute carbol-fuchsin. It is a facultative parasite, but probably most of the outbreaks are of a sporadic nature, infection arising from saprophytic bacilli in soil or manure. In the blood it does not form filaments, but in culture and in the heart lesions of chronic cases threadlike forms are found. It is non-motile, and does not form spores.

For a non-sporulating organism it shows a rather high resistance to the usual destructive influences, such as disinfection and putrefaction. According to Kitt and Petri, it is difficult to kill in large pieces of meat, such as hams, by ordinary cooking or pickling. Prolonged drying, and heating for five minutes at 60° C., usually kills it. Putrefaction has little effect on it, and the bacillus flourishes in filthy fluids. In *culture* it grows readily at summer temperature and in the presence of air, but better as an anaërobe, and on all the ordinary media, except potato. In gelatine stab cultures the growth is characteristic, occurring along the needle track with a multitude of delicate outgrowths, giving the mature culture the appearance of a bottle-brush. In acute cases the disease is septicæmic in character, and the bacilli are everywhere present in the blood. Although not in great numbers, they are usually to be found within the polynuclear leucocytes. They are also seen in the spleen, kidneys, lymphatic glands, skin lesions, fæces, and urine. In the chronic heart cases the bacilli are not found in the blood, but they are present in the cardiac vegetations in immense numbers, and they are also found in the spleen in some chronic cases.

ACCESSORY CAUSES.

Bad sanitation and overcrowding constitute accessory causes.

PERIOD OF INCUBATION.

The period of incubation is about two to three days, and the duration of visible illness two to five days.

MORBID ANATOMY.

Swine dead from an *acute* attack usually show purple or violet discoloration of the skin all over the body. A dressed carcass appears as if it had been dipped in vermilion. In such cases the autopsy usually reveals a small quantity of blood-stained fluid in the abdominal and thoracic cavities, hæmorrhages and petechiæ are common on the serous membranes and organs generally. The lungs are congested; the heart-wall and epicardium and endocardium show ecchymoses and petechiæ.

Frequently there is some degree of gastritis and enteritis present, the latter involving the small rather than the large bowel, but never becoming ulcerative or diphtheritic. The solitary glands are often swollen and congested. The kidneys show petechial hæmorrhages on the surface, and also on section. The spleen is somewhat enlarged and hyperæmic. The lymphatic glands throughout the body are swollen, dark in colour, and sometimes hæmorrhagic.

In animals dead from the *chronic* form the carcass is generally more or less wasted. The skin discoloration is not so diffuse as in the acute cases, being usually confined to the ears, perineum, and under surface of the abdomen. On opening the chest there is often found blood-tinged hydrothorax, and nearly always pronounced œdema of the lungs, causing them to appear much larger than normal, with the interlobular septa very plainly marked. The heart on section shows vegetations of a cauliflower-like appearance, and various dimensions on the tricuspid, mitral, and aortic valves. The mitral valve is, however, the commonest seat of the lesion. The mucous membrane of the large bowel is congested and bile-stained. The liver and spleen are usually enlarged.

SYMPTOMS.

In all temperate countries swine erysipelas has a seasonal occurrence. Comparatively few acute or subacute outbreaks are seen in winter, but are most prevalent in the summer months, declining in the autumn. This seasonal incidence is probably due to the higher temperature of the summer months being more favourable to the growth of the bacillus in the soil or manure of the sty. In this country three forms of the disease are known, and may be classified as follows: (1) *Mild*; (2) *acute*; (3) *chronic*.

Mild Form.—The *mild* or *urticarial* form is commonly met with, during the summer in forward "stores" and fat pigs. The early symptoms, which appear suddenly, are great dulness or prostration; the animal lies buried up in the straw in an almost comatose condition. There is complete anorexia and some loss of power of the hind-quarters. The temperature runs up to 107° or 108° F. Constipation is always present, and may be followed by diarrhœa.

In a short time the characteristic skin lesions appear on the neck, back, shoulders, and buttocks in the shape of raised diamond-shaped markings, which may remain discrete, but usually some of them tend to run together, forming large irregular raised patches; the markings at first are of a bright red colour, passing on to violet, and finally to a dark purple colour. The hock and knee joints are often swollen, and if the animal is made to leave its bed it evinces pain by walking on its toes and squealing, and resumes the recumbent position as soon as possible. This form is rarely fatal, and does not seem to spread by contagion, only a small minority in a herd being simultaneously attacked.

Usually about the fourth day of the illness the skin-marking disappears, and the pig makes a remarkably quick recovery. Often, however, pigs which recover from this form remain more or less lame or crampy, and may ultimately succumb to the chronic form.

Acute Form.—In the *acute* form the symptoms are not very characteristic, being the usual signs of acute illness in the pig. The attack is sudden and rapidly fatal. The disease is ushered in with a high temperature, accompanied by rigors; the animal refuses food, and lies buried up in the straw; extreme prostration, with loss of muscular power, quickly follows. Skin discoloration appears in a few hours' time, beginning as small raised patches on the throat and under surface of the body, which rapidly spread and become confluent; in a short time the entire surface of the body often becomes dark rose or purple in colour.

In prolonged cases sloughing of portions of the discoloured skin may take place, especially the tips of the ears and tail. Vomition may occur, but it is not a common symptom. There is generally a tendency to constipation, which is often followed by blood-stained diarrhœa.

As death approaches the temperature falls, the respirations are accelerated, and the ears and snout become cyanotic. The duration of the disease varies from twelve hours in a very acute case to four or five days in the less acute forms.

Chronic Form.—In the *chronic* form it is usually found on close inquiry that the animal had previously a mild attack from which it had apparently recovered, or the history may be that the animal had been lame or crampy.

The first symptom is increased respiration, with or without a soft cough, followed in a day or two by skin discoloration, beginning at the tips of the ears and perineum, and spreading to the cheeks, neck, and along the ventral surface of the body. The appetite is capricious, and constipation is present.

As the disease progresses the respirations become greatly laboured, of an abdominal or " pumping " character, and the animal rests itself on its sternum. The skin discoloration, which is often "spotty," becomes dark purple or cyanotic. Death usually supervenes in about eight to sixteen days after the symptoms have become established. Occasionally in these chronic cases the animal may be found dead without showing any symptoms of illness.

PROGNOSIS.

In acute cases the mortality is fully 80 per cent. In the mild form the disease runs a course of about four days, and recovery is the usual result. The chronic form is invariably fatal.

DIFFERENTIAL DIAGNOSIS.

Acute cases of swine erysipelas resemble in a marked degree acute swine fever, and, without a post-mortem examination and often a bacteriological investigation, it is impossible to decide which disease is present.

As a rule, however, swine erysipelas attacks fat pigs, is more rapid in its onset, and kills more quickly than swine fever does at the present time in this country. The characteristic diamond-shaped skin-markings may also be noticed on some of the milder cases, the appearance of which should go a long way to decide the diagnosis in favour of swine erysipelas.

The mild form is readily diagnosed by the skin lesions and course of the disease. It is most probable that the majority of cases showing rheumatic symptoms, with swollen joints and crampy walk, are due to swine erysipelas.

The chronic form is recognised by the "pumping" character of the respiration, and by the skin discoloration beginning at the ears and perineum.

Bang has shown that all cases of vegetative endocarditis in swine are not due to swine erysipelas bacilli, and that a similar lesion is sometimes produced by a streptococcus. In these latter cases Bang states that the vegetations have not so pronounced a cauliflower-like appearance, but are smoother and firmer in character.

THERAPEUTICS.

In the mild form a warm bed should be supplied. A purgative, such as castor oil, Epsom salts, or sulphate of soda, if taken by the animal in the food, will do good. If otherwise, it is very doubtful whether it is prudent to upset the patient, and run the risk of choking by drenching. In acute cases serum from a hyper-immunised animal in large and oft-repeated doses is the best remedy. Salicylate of soda may also be tried. Treatment is useless in the chronic form.

PROPHYLAXIS.

Affected animals should be isolated, and disinfection of infected premises, litter, etc., should be rigidly carried out. Swine erysipelas was one of the diseases against which Pasteur introduced a vaccine. It consisted of a broth culture attenuated by "passage" through rabbits. Two vaccines of different degrees of virulence were used. Vaccination by this method was used on a large scale in France for some time, but its results were never satisfactory, and in recent years it has fallen completely into disuse, its place being taken by a "mixed" method, in which serum and living cultures are employed. This method was first introduced in Germany by Lorenz of Darmstadt, and afterwards modified in France by Leclainche.

Lorenz uses injections of serum and culture on opposite sides of the body, and applied at the same time; Leclainche injects a mixture of serum and culture, followed some days later by an injection of culture alone. The serum is now obtained from horses which have been hyperimmunised by repeated and increasing doses of virulent swine erysipelas culture. This serum, when injected into a pig, confers immediately a passive immunity which lasts only about ten days. The immunity set up by serum and pure culture, however, lasts from six to twelve months.

Vaccination should not be practised on non-infected premises, except the herd is in imminent danger of infection from surrounding sources.

Leclainche's Method.—When swine erysipelas appears in a herd, and is positively diagnosed, no time should be lost in applying Leclainche's method. He advises that in herds where the disease has already appeared all the pigs should receive as soon as possible a dose of serum, varying from 10 to 20 c.c., according to weight. This preliminary dose of serum prevents the immediate spread of the disease, and may have some curative effect in early cases; it also minimises the risk of vaccination proper which is to follow.

About ten days afterwards the vaccination proper is performed. It is divided into two operations. The first consists in injecting subcutaneously at the root of the ear or inside of the thigh a mixture of serum (the dose being 1 c.c. per 20 pounds live weight, with a maximum dose of 10 c.c. and a minimum of 5 c.c.), and 0·8 c.c. of pure culture. Twelve days later the animal receives 0·8 c.c. of pure culture without any serum.

Mr. Franklin, of Stevenage, who has had a large experience of this method of vaccination in England, recommends the injection of serum alone for the first operation, in doses of 1 c.c. to each 20 pounds live weight, followed in six or seven days later by the injection of 0·5 c.c. pure culture.

The serum-virus method of vaccination just described has been practised largely, and has proved entirely satisfactory.

PUBLIC HEALTH.

In acute cases the carcasses are unfit for food, and should be cremated or buried intact. The carcasses of chronic cases, if slaughtered before symptoms are established and where the disease is entirely localised in the heart, need not be condemned.

NOTE BY EDITOR.

Transmission of the disease to man has been observed by several investigators. Infection may occur from inoculation wounds received during the slaughter, or post-mortem examination of affected pigs, or during the employment of inoculation syringes.

"The local changes consist chiefly of an erysipelas of the seat of infection (finger, hand), with painful swelling and sometimes a formation of vesicles; in some cases, high fever and swelling of the neighbouring joints are observed. Recovery generally takes place after eight to fourteen days."*

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* Friedberger and Fröhner, "Veterinary Pathology."

CONTAGIOUS BOVINE PLEURO-PNEUMONIA

By J. F. CRAIG, M.A., M.R.C.V.S.,
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Synonyms.—Pleuro-pneumonia contagiosa ; Lung plague of cattle.
French : Péripneumonie contagieuse.
German : Lungenseuche viehseuche.

Definition.—A contagious febrile disease, confined naturally to bovines. It is usually chronic in its course, and the specific lesions are developed in the lungs and pleuræ.

HISTORY.

The first definite report of the appearance of this disease as an epizoötic emanated from Switzerland in the early portion of the eighteenth century. Later the disease spread over the whole of Germany, France, and Italy, and at the commencement of the nineteenth century it extended throughout all the countries of Western Europe. In 1839 it was introduced to Ireland as the result of the importation of some Dutch cows from the Hague. Thence it spread all over Ireland. England and Scotland were also infected with the disease in 1842 from the Continent and Ireland. In every part of Great Britain the disease appeared except the Welsh and Scottish Highlands, into which no foreign cattle were imported. It was introduced into the United States in 1843 by the purchase of a ship's cow by a dairyman in Brooklyn, and spread rapidly. It reached Chicago in 1887. In 1854 bovine pleuro-pneumonia was imported into South Africa, and in 1859 into Australia ; later to Tasmania and New Zealand. The British Isles are now free from the disease. The last outbreaks occurred in 1898. The disease was said to be exterminated from the United States in 1892. Contagious bovine pleuro-pneumonia still prevails in Austria, France, Belgium, some parts of Germany, Africa, Asia Minor, and Australia.

ETIOLOGY.

The cause of the disease is a micro-organism, which is almost ultravisible, and appears as a brilliant refrangent point with a magnification of 2,000 times. It is probably only about $\frac{1}{5}$ to $\frac{1}{10}$ μ in diameter, and passes through the pores of a Berkefeld filter. It is present in the lesions, but can only be identified in culture. The organism was discovered and first isolated by Nocard and Roux in 1898. These investigators first cultivated the microbe in broth inoculated with pleuro-pneumonia lymph or exudate from the interlobular connective tissue of a bovine dead of pleuro-pneumonia. This broth was enclosed in a collodion capsule, and inserted with aseptic precautions into the peritoneal cavity of a rabbit. The reason collodion was used as a capsule was that it permitted diffusion of liquids, but was impermeable to leucocytes or other cells. At the end of fifteen or twenty days the rabbit was killed, and the capsule removed from the peritoneum. The contents had become slightly opalescent, and were found on examination to be teeming with the organisms.

Later, the organism has been grown in Martin's peptonised broth, containing one-twentieth of its bulk of blood-serum from the ox or rabbit, incubated in a test-tube at body temperature. It may also be cultivated on solid nutritive media.

The reasons for accepting this organism as the cause of the disease are—(1) That cultures inoculated subcutaneously into a susceptible animal will produce a lesion at the seat of inoculation identical with those in the interlobular connective tissue in the lung of a natural case of the disease; (2) that cultures of the same microbe can be recovered from the lesions so produced; (3) that animals inoculated with these cultures acquire an immunity against the disease and against the lymph from a pleuro-pneumonia lesion. Experimentally, the disease showing the typical lesions in the lungs has not been set up by cultures of the microbe.

Animals affected are bovines. The yak and buffalo are said to be susceptible.

SUSCEPTIBILITY AND IMMUNITY.

Warm climates and hot seasons accelerate the spread of infection and increase its virulence. It is believed that 25 per cent. of cattle have a natural immunity against the disease. Recovery from a natural attack of the disease confers a lasting immunity.

TRANSMISSION OF THE DISEASE.

The disease is ordinarily contracted by cohabitation or immediate contact of healthy and diseased animals. It is believed that contagious bovine pleuro-pneumonia is transmitted by inhalation, but there is no experimental evidence to show by what path the organisms enter the body. It is estimated that the disease may be transmitted to a distance of forty yards through the air, and still farther if carried by the medium of dust. Even in the early stages, before any symptoms are observed, infected cattle can spread the disease. When the disease is at its height the infection is most virulent. Animals apparently recovered, but having necrotic foci or encapsuled lesions in the lungs, can transmit the disease even for long periods after the attack. Walley estimated in such cases that the virulence may be retained for fifteen months. This was the experience in dealing with outbreaks in the British Isles, and proved one of the several drawbacks to the use of preventive inoculations. Dr. Theiler states that these old cases, the so-called "lungers," did not cause much trouble in applying protective measures in the Transvaal. Probably the reason for this difference of experience is that infective chronic cases are much more numerous in temperate or cold than in warm climates. The lungs of affected animals are said to retain the power of transmitting infection for a year if kept at a temperature of 5° or 6° C. In exceptional cases the contagion is carried from the pregnant animal to the fœtus. Less frequent means of spread of the disease are infection through contaminated food, by attendants, by common watering-troughs or ponds, through infected empty cowsheds, or, very occasionally, through the manure from affected cattle spread on lands grazed over by healthy cattle. Law states that buildings may retain the infection for a long period—up to a year.

PERIOD OF INCUBATION.

The period of incubation is indefinite, because of the insidious nature of the disease and the want of evidence from artificial transmission. It may be regarded as the interval which elapses between contact with the diseased animal and the first rise of temperature. This period may be taken as varying between three weeks and three months. Most frequently the symptoms appear within a month. The local swelling resulting from the inoculation of cultures or of the lymph from an infected lung usually appears in from six days to three weeks. Cases are recorded in which three or four months have elapsed after contact before any definite symptoms of the disease were shown. The period of incubation is shorter in hot weather than in cold, and as the result of adverse conditions, such as fatigue or bad feeding.

MORBID ANATOMY.

The lesions are confined to the lungs and pleuræ. The pneumonia always precedes the pleurisy, and hence a post-mortem examination of an animal killed in the early stages of the disease may reveal lesions confined entirely to the lungs. The lungs may be involved to a considerable extent before the pleuræ become affected. In the initial stage small circumscribed inflammatory centres, about the size of a hazel-nut, form in the lung. In these areas the interlobular connective tissue becomes congested and infiltrated with serum, and the lobules become reddened, well marked off, and saturated with a serous fluid.

In the advanced stage a large proportion of one or both lungs is hepatised. When the hepatisation reaches the surface, the overlying pleura becomes involved. The pleurisy is sero-fibrinous, and a yellow fibrinous layer forms on the parietal and visceral pleura. The exudate in some cases is very great. Adhesions sometimes result between the lung and the chest-wall. In cases of old standing these adhesions may become permanent.

The hepatised parts of the lung are very prominent, very firm and heavy, sink in water, and do not crepitate on manipulation. A hepatised lung has weighed as much as 50 to 100 pounds. On section, the cut surface of the affected lung presents a characteristic marbling. The interlobular connective tissue is very much thickened, may be as much as ½ inch thick, and is of a light yellow colour. The network formed by this tissue corresponds with the veins of the marble, and the lung parenchyma with the groundwork. The thickening of the interlobular septa is due to the distension of the connective-tissue spaces and lymphatic vessels with a yellow inflammatory fibrinous fluid, the so-called "pleuro-pneumonia lymph," which has been used for the purpose of producing immunity against the disease. In the older lesions and in the sequestra the septa are dry and firm, the fluid having been replaced by new fibrous tissue. The lobules within the septa may present different colours, varying from dark red, red, yellow, to grey. Probably the lesions in the lobules are secondary to those of the septa. The reddish colour of the lobules is very often due to collapse from pressure of the neighbouring swollen septa. In many of them it is due to red hepatisation—*i.e.*, to the presence of a fibrinous exudate.

Various stages in the inflammatory process may be present in the lung, because the lesions are of different ages. The grey-coloured lobules indicate grey hepatisation. In the yellow lobules catarrhal changes are taking place. Black hæmorrhagic infarcts are frequently observed as black or dark red areas, and are believed to be brought about by an antecedent thrombosis of portion of the pulmonary artery. In extensive lesions some parts may undergo necrosis. This necrosis is the result of interference with the blood-supply, and is brought about by pressure of the distended septa or the contraction of new fibrous tissue formed in the septa of old lesions. These necrotic areas, "the sequestra," vary in size from a nut to a child's head. They are dry, and a characteristic feature of them is that they are never putrid. In old lesions they become detached and surrounded with a capsule of fibrous tissue, forming the so-called "pleuro-pneumonia cysts." Sometimes these sequestra are softened and absorbed, and leave empty capsules or vomicæ, or the affected portion of the lung presents a honeycombed appearance on section. Sometimes in such areas all that is left consists of the thickened branched bronchial tubes, all the other neighbouring tissues having become necrosed, softened, and absorbed.

With the pleurisy, occasionally the pericardium is involved. The bronchial, pectoral, and mediastinal lymphatic glands are often enlarged, congested, and infiltrated with exudate.

SYMPTOMS.

The disease is usually insidious in its onset.

In the first stage the only symptom observed is a cough, at first slight, later becoming more pronounced and frequent. It is commonly short, dry, or husky and weak, occasionally hard. It is particularly noted when the animal is rising or feeding or drinking, or after coming out of the stall into the open air. Later the cough is accompanied by arching of the back, extension of the head, and protrusion of the tongue. On auscultation at this time very little or no abnormality may be detected, save, perhaps, an exaggeration of the bronchial sounds.

In the second or more advanced stage the animal is dull and is not inclined to move about, and its condition becomes impaired. The hair stands erect and the coat becomes hide-bound. The pulse and respirations are increased in frequency. Grunting may be noted, especially after feeding. The temperature is raised, and varies from 104° to 108° F. The external temperature is uneven. The appetite is capricious, rumination is irregular, and the secretion of milk in cows is diminished. Thirst is increased, and there is a tendency towards constipation. Pregnant animals frequently abort. Manipulation of the chest may cause flinching or moaning. Auscultation and percussion reveal the presence of consolidation of certain parts of the lung, and even of pleurisy. This condition has been noticed by some investigators to frequently commence first in connection with the apical lobe of the right lung. Hence one should not omit to auscultate in front of the right shoulder. In the majority of cases a portion only of one or both lungs is affected. Over these solid areas there is an absence of vesicular murmur, an exaggeration of the heart or abdominal sounds on auscultation, and increased dulness on percussion. At the margins of these areas crepitation sounds may be heard. In the pleurisy of this disease the friction sounds are never well marked. When the exudate has formed, absence of lung sounds will be revealed on auscultation of the lower portion of the thorax, and occasionally a tinkling sound at the upper margin of the liquid.

In the last stages these symptoms become exaggerated. Emaciation becomes extreme. The animal stands with the elbows turned out and the forelegs wide apart; the head is extended, and the tip of the tongue protruding. There is marked respiratory distress, heaving of the flanks, and each respiration is accompanied by a loud moan.

A muco-purulent discharge comes from the nostrils; the muzzle becomes dry and hot. The appetite, rumination, and lactation (if in a cow) are in abeyance, and constipation may give place to a profuse diarrhœa. Auscultation and percussion reveal large areas of consolidation and extensive pleuritic effusion. The animal ultimately dies from asphyxia or exhaustion.

COURSE AND MORTALITY.

The mortality ranges from 50 to 70 per cent. The course varies very considerably. In some cases the ox recovers in the early stage, and soon improves in condition. In the large proportion of cases the first stage may last for weeks or months, but the disease ultimately advances. The further development is hastened by certain conditions, such as high condition, hot weather, exposure to chills, long journeys on land or sea, an attack of bronchial catarrh, or some special susceptibility to the disease. In young, strong, well-fed bovines, and on the first appearance of the disease in a district, the course is frequently rapid. In severe cases, with both lungs involved, death may occur in from five to eight days, and in some instances without any marked loss of condition. In ordinary cases death usually occurs in from two to four weeks from the commencement of the acute stage. An apparent complete recovery occasionally takes place within a few days after the acute stage has set in, but then recovery is protracted, and the animal remains debilitated and in poor condition for a long time.

Apparent recovery may occur even in some comparatively advanced cases. Here chronic lesions remain, and the affected animals, commonly called " lungers," are still able to infect their fellows. These bovines may remain unsuspected for a long time in a herd, and have been the means of introducing the disease into other localities or countries. Relapses with these cases, however, are frequent, and not infrequently they remain in poor condition and succumb in the end to the disease.

SEQUELÆ AND COMPLICATIONS.

Tympanites may complicate the disease. Abscesses depending on other infections sometimes develop in the lung, the pleuræ, bronchial glands, and joints. Sero-fibrinous effusions into the tendon-sheaths and dewlap may also appear. Finally, contagious bovine pleuro-pneumonia may be complicated with tuberculosis.

DIFFERENTIAL DIAGNOSIS.

It is difficult to obtain conclusive evidence of contagious bovine pleuro-pneumonia *intra vitam*, especially in the early stages. The symptoms are never pathognomonic, and at most only point to chronic lung trouble. During its development the disease may not even point to an affection of the lungs. During the life of the animal the disease can only be suspected. These suspicions are aroused when it is understood—(1) That the disease is prevalent in the country; or (2) that the cattle have been introduced from countries where the disease is known to exist; or (3) that these cattle have been recently in contact with the animals that are being examined. The slow and insidious onset and course of the disease, and a succession of similar cases, are all indications which require attention. But a post-mortem examination of an affected animal is essential before an exact diagnosis can be made. At that examination the marbling of the lung, the distension of the interlobular connective tissue, the varying colour of the lung parenchyma, pointing to the difference in age of the lung lesions, are points of special importance. In doubtful cases cultures of the pleuro-pneumonia organisms may be made from the exudate in the lung lesions.

Diseases with which Contagious Bovine Pleuro-pneumonia may be confused.

1. **Tuberculosis** is difficult to differentiate during life, because the two diseases are often associated. In cases of contagious bovine pleuro-pneumonia where the temperature is normal or not raised above 103° F., such as occurs in the early stages, one is enabled, by the use of tuberculin, to ascertain the presence or absence of tuberculosis, but it does not negative contagious bovine pleuro-pneumonia. In investigations in this country 50 per cent. of the cases of the latter disease were complicated with tubercle. Here the post-mortem examination is decisive.

2. **Ordinary or Sporadic Pneumonia** is differentiated clinically by its acute invasion, by the cases being more frequently isolated, by the rapid rise of temperature, and the short, sharp, painful cough. On post-mortem examination there is little or no thickening of the interlobular connective tissue, and the lesions are nearly all of the same age, and present an almost uniform colour. Exceptionally very acute cases of contagious bovine pleuro-pneumonia may be mistaken for sporadic pneumonia. The isolation of the organism by culture may then be resorted to.

3. **Verminous Bronchitis** is usually easily distinguished. It most commonly affects young bovines up to about two or three years old. It is prevalent mostly in the autumn. The symptoms are chiefly bronchial; the cough is hard, husky, and spasmodic, and the nasal discharge contains the eggs or embryos of the parasite. No doubt could arise at a post-mortem examination.

4. **Epizoötic Pneumonia of American Cattle.**—Nocard describes an infective pneumonia among American cattle imported into France and slaughtered at Paris. In these cases the lesions were very similar to those of contagious bovine pleuro-pneumonia. This epizoötic pneumonia is distinguished by the presence of a short ovoid motile organism in the lung tissue and bronchial tubes. The disease may be transmitted to calves and pigs (see p. 418).

5. **The Pulmonary Form of Septicæmia Hæmorrhagica of Bovines** is differentiated by its acute onset and the rapidity of its course. The affected animals seldom last for more than eight days. The disease is associated with skin and intestinal complications. The lung lesions are all of the same age, and in the blood a pasteurella has been found which is pathogenic to deer, swine, and rats. In America this is sometimes known as "cornstalk disease," and it has been suggested that the condition which Nocard describes as epizoötic pneumonia is a chronic form of the disease (see p. 416).

6. **Aspergillosis of the Lung** is distinguished by the more limited areas of consolidation, by the absence of marked respiratory symptoms, and by the presence of the aspergillus in the bronchi and lung tissue. The disease may be transmitted to smaller animals of other species.

TREATMENT.

No successful curative treatment for contagious bovine pleuro-pneumonia has been found. Even were treatment successful, the disease is so infectious, so insidious, and has such a long period of incubation, that for economic reasons it would not be wise to preserve affected animals. In this, as well as many other countries, the disease is scheduled, and all the measures employed are put in force to eradicate the disease and prevent its recurrence in the country.

PREVENTIVE TREATMENT AND PROPHYLAXIS.

The best method of dealing with outbreaks of the disease consists in isolation of affected herds, destruction of affected and in-contact animals, and disinfection. The neighbouring herds should be kept under close supervision for a period of three months after the outbreak, and movement in the affected area restricted. The in-contact animals must be destroyed, because it is impossible to ascertain whether any or all of them are in the incubation stage of the disease, or have only partially recovered from an attack and have some old lesions in the lungs. These animals would still be capable of spreading the disease. In this country, not only was that done, but the cowsheds, discharges, and contaminated fodder were disinfected; and the affected carcasses carefully disposed of, either by burying 6 feet deep in quick-lime or subjecting to a very high temperature in a destructor. In order to prevent the recurrence of the disease, the source of the outbreak must be traced and prevented. Importation of bovines from affected areas must be stopped, a most successful safeguard, which has prevented the reappearance of the disease in the British Isles since 1898. A restriction of less value would consist in the importation of cattle subject to a three or four months' quarantine. A less important point in the prevention consists in the prohibition of fodder, litter, etc., from infected countries, unless thorough disinfection has been carried out.

In certain countries, such as South Africa, it has been found impossible to carry out these measures. Efficient isolation cannot be enforced, and even the destruction of affected animals cannot be insured. The latter measure, however, is imperative in order to eradicate the disease. But even supposing those bovines that are clinically affected are destroyed, it is by no means certain that the disease will ultimately be got rid of. The "lungers," the partially recovered animals, and the animals in the incubation period of the disease, are left alive. and are a serious source of danger to their fellows.

The "lungers," however, have not proved such a very serious bar to the eradication of the disease in the Transvaal as in the British Isles.

Under such conditions an attempt may be made to immunise, by means of certain inoculations, in-contact animals or herds threatened with the disease. In this way the disease has been successfully dealt with and eradicated in the Transvaal.

Methods of Preventive Inoculation against contagious bovine pleuro-pneumonia have been practised since the beginning of last century, and in all cases the agent used contains the specific organisms.

The natural disease with lung lesions is not set up by the inoculations, but a local lesion is produced which resembles in appearance those in the interlobular tissue of the lung. The inoculation is a unique example of a vaccine in which no attempt is made to modify the virus.

The methods employed are the following:

1. *The Old Method.*—The material here used is the yellow fibrinous exudate from the interlobular connective tissue of a hepatised portion of an affected lung from a natural case of the disease. This may be obtained by incision and pressure over the affected parts of the lung, due antiseptic precautions being taken in the process. The fluid pressed out is received into an aseptic receptacle, and then filtered through a piece of clean linen to remove any solid particles. The site of inoculation is the dorsal aspect of the tail, about 3 or 4 inches from the tip. The hair at this point is clipped and the skin cleansed, and about ½ to 1 c.c. of the lymph or exudate injected subcutaneously with a hypodermic syringe, or a piece of woollen thread is soaked in the lymph and inserted as a seton under the skin. Seventy-five to ninety per cent. of inoculated bovines react. The reaction takes place in from one to four weeks, and consists of an inflammatory swelling, not usually larger than a hen's egg, accompanied frequently by slight fever and increased respirations. If the lymph be contaminated, excessive swelling, necrosis of the tail, and even pyæmia, may result. The mortality following this inoculation is generally put down as between 1 and 3 per cent., and necrosis of the tail occurs in from 5 to 15 per cent. of cases.

2. *Pasteur's Method.*—The only difference in this method consists in the source of the inoculating material. It is obtained from a local subcutaneous swelling, produced artificially by injection of the pleuro-pneumonia lymph from a natural case. The subject used is a healthy calf, about three or four months old, and the injection is carried out in the dewlap or behind the shoulder. On the appearance of the swelling the calf is destroyed, and the lymph collected in sterilised glass tubes, which are afterwards hermetically sealed until required. In this way the lymph may be kept active for six to eight weeks. A few minims constitute a sufficient dose for a full-grown bovine. It has the advantage over the old material of being free from contamination, but it would appear that the virulence of the lymph is attenuated in some way. In Prussia it has not met with such success as the old method.

3. *Intravenous Injections* of ℥ss. of lymph into the jugular vein have been employed. The results are said to be very good, and more certain than those previously mentioned. Greater care, however, must be taken that the lymph is not contaminated, and therefore this method has not met with much favour.

4. *Method of Nocard.*—As a result of the discovery of the causal organism of the disease, and its later cultivation outside the animal body, cultures have been used in place of the lymph from a naturally affected case. The cultures are applied in the same way as the lymph in the older method. They have the advantage of being much more convenient to obtain, and in every way better adapted for the purpose. The effects are less severe, and the results obtained are very encouraging, and more certain than with the older methods. Of 970 cattle so inoculated, none died; in three only loss of the tail resulted.

VARIOLA

By E. WALLIS HOARE, F.R.C.V.S.

I. GENERAL REMARKS ON VARIOLOUS AFFECTIONS.

The term "variola" in human medicine signifies small-pox. The connection between this disease and the variolous affections met with in animals is a subject on which marked differences of opinion exist amongst authorities. Some believe that bovine and equine variolous affections are in reality a form of human variola, modified by transmission through animals; others are of opinion that these diseases are separate and independent.

Chauveau inoculated a number of calves with the virus of human variola, and found that small reddish conical papules were developed, which rapidly disappeared, and no eruption resembling cow-pox was manifested. An unvaccinated child was inoculated with the contents of an original papule from one of the above calves, and confluent small-pox developed. On the other hand, there are authorities who claim to have demonstrated that typical cow-pox may be produced in the calf by inoculation with the virus of human variola. All variolous affections in animals resemble each other in certain particulars; they all present the febrile stage preceding the appearance of the typical eruption.

Variola affects cattle, sheep, horses, goats, pigs, dogs, buffaloes, camels, and monkeys. With the exception of sheep-pox, concerning which there is a difference of opinion, the variola of one species can be conveyed to another by accidental infection or by inoculation, and also to human beings.

While cow-pox and horse-pox are mild affections, sheep-pox, on the other hand, is a serious disease, accompanied by a high mortality, and resembling in many respects human variola. Again, the contagion of cow-pox and horse-pox is conveyed from one animal to another by actual contact, while in sheep-pox infection may take place at some distance from the affected animal. Sheep-pox is said not to be transmissible readily to man—indeed, Voisin and Nocard failed in their attempts to convey the disease from sheep to man. Vaccination of sheep with cow-pox does not produce immunity to sheep-pox.

Variola in all animals presents a characteristic eruption which passes through certain stages. Although there are certain peculiarities in the eruption and in its location in the various animals, one description of these stages will suffice for all:

1. *The Papular Stage.*—This commences with the appearance of small red spots resembling flea-bites. These, by proliferation of cells into the papillary layer and *rete mucosum*, are formed into small papules or firm nodules.

2. *The Vesicular Stage.*—In this the proliferation of cells increases. They collect in clusters or groups, separated from each other by septa or walls formed chiefly by the epidermic cells. Exudation next takes place, and the lymph collects in these spaces. The vesicle thus differs from that produced by an ordinary vesicant, in which there is but a single sac formed. The summit of the vesicle becomes clear, and in that of cow-pox there is a slight depression or umbilication in the centre. The exact cause of this umbilication is not definitely known. Some authorities believe that it is caused by the hair follicles and orifices of the sweat glands lying in the centre of the vesicle, and firmly connected with the underlying true skin, which prevent the epidermis from rising externally under pressure of the contents of the vesicle. Others state that it is due to the septa in the interior of the vesicle, which prevent the centre from rising up. During the next stage—viz., that of suppuration—the septa break down and the umbilication disappears.

3. *The Pustular Stage.*—In this the vesicle becomes a pustule, its contents become turbid and purulent, and its summit assumes a globular form and a greyish-yellow colour. An areola of congestion occurs around the pustules, and the skin between them becomes swollen.

4. *The Stage of Desiccation.*—In this the pustule dries up, at first in the form of yellowish scales, which later on become a dark brown crust. Desquamation occurs, and a white shining cicatrix or a brownish-red spot may be left, which is known as a "pit." This results from destruction of the superficial layer of the dermis.

II. COW-POX (VARIOLA VACCINIA).

Synonyms.—Vaccinia.

French: Cow-pox; Vaccine.
German: Kuh-pocken; Pocken Krankheit Kühen.

Definition.—An acute contagious and inoculable disease affecting chiefly milch cows, characterised by slight febrile disturbance and the appearance of an eruption on the surface of the mammary gland and teats, which passes through the stages of papule, vesicle, pustule, and crust.

GENERAL REMARKS.

It is readily conveyed from the cow to the horse, and also to man, by actual contact or by inoculation. The virus inoculated into man (vaccination) affords protection more or less permanent from small-pox.

Cow-pox and horse-pox resemble each other in many respects. The variola of the cow can be conveyed to the horse, and that of the latter to the cow. They differ, however, as regards the seat of the eruption and in certain minor details. Cow-pox has been known in England and in many parts of the Continent for centuries. Special attention was directed to the disease in 1796, when Jenner discovered that vaccination of man with the virus of cow-pox afforded protection against small-pox. It is said that even previous to this date it was a common notion with breeders and dairymen that cow-pox protected against small-pox. Numan (1839) reported numerous observations on the disease, and realised its transmission from the cow to the horse, ass, sheep, goat, camel, etc. He also proposed the cultivation of vaccine in cows and its collection in glass tubes, sealed by means of a lamp, and finally showed that vaccine thus preserved was, after a period of ten weeks, as active as the fresh virus. About this period the disease was observed many times in England and in Germany.

Hering (1839) studied it and differentiated it from eruptions on the udder of a similar appearance. He pointed out that it was readily transmitted to the cow and calf by direct contact or by various intermediaries. Shortly afterwards Ceely published a description of cow-pox, giving its clinical characters. The disease has been observed since this period in every country, especially in Denmark, Prussia, Holland, etc., and also in France.

In the present day it is not a common affection in Great Britain, but sporadic cases appear from time to time.

Nature of the Contagion.—The contagion of cow-pox is conveyed by actual contact or by accidental inoculation from one animal to another. No specific micro-organism has yet been discovered for the disease. Guarnieri (1892-1894) and others discovered in human variola, and also in the variolæ of animals, protozoön-like bodies in the skin lesions and in the lymph, which were termed *Cytoryctes variolæ*. Councilman and his colleagues found similar bodies in the lesions of cow-pox, and termed them *Cytoryctes vacciniæ*. These parasites were found chiefly in the cells of the *rete mucosum;* other observers discovered them in the vaccine lymph and in the surrounding tissue. It was claimed that cultivations of these organisms and subsequent inoculation of calves produced vaccinia in these animals. But according to Nocard and Leclainche, attempts to make cultures have not given any results, and the isolated cultivable microbes are powerless to reproduce the vaccinal eruption.

Various investigators have demonstrated that the virus of cow-pox is filtrable, hence the disease is now regarded as depending on an ultra-visible virus. According to McFadyean,* the facts obtained as the result of numerous experiments "render it highly improbable that the so-called Guarnieri bodies constitute the actual micro-parasites of the disease. On the other hand, the constancy of these bodies in the epithelial cells of vaccinia points strongly to their being of the nature of cell alterations caused by, and probably including, the actual microbes of the disease. . . . The so-called 'initial bodies' described by Prowazek as occurring in the epithelial cells, either in the bodies of Guarnieri or external to these in the cell substance or nucleus, must also be considered too large (1 to 1½ μ long) to traverse the pores of a Berkefeld filter, and, like the Guarnieri bodies, they ought probably to be interpreted as 'reaction products' of the virus. On the other hand, it is possible that the much smaller bodies recently described by Volpino may constitute the actual virus. These bodies, which did not exceed $\frac{1}{5}$ μ in size, and were very actively mobile, were detected in the protoplasm of the epithelial cells and also in the intercellular substance of the rabbit's cornea, after inoculation with vaccine lymph."

ETIOLOGY.

The disease is chiefly conveyed from affected animals to the healthy by the hands of the milker. Freshly vaccinated human beings may also infect cows if their hands become contaminated with the virus. Attendants who have dressed horses suffering from horse-pox may get their hands contaminated with the virus, and if they milk cows without adopting suitable precautions lesions similar to those of cow-pox will occur on the udder and teats of the animals.

As already stated, there are marked differences of opinion with reference to the analogy of human variola and vaccinia. Osler† states: "The weight of evidence favours the view that cow-pox and horse-pox are variola modified by transmission." According to Copeman, "small-pox and vaccinia are both of them descended from a common stock—from an ancestor, for instance—which resembled vaccinia far more than it resembled small-pox."

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* *Journal of Comparative Pathology and Therapeutics*, September, 1908.

† "Principles and Practice of Medicine."

PERIOD OF INCUBATION.

When the disease is contracted by accidental infection, about six or seven days is the average period of incubation; when due to inoculation, about two days.

SYMPTOMS.

Cow-pox is a mild affection, usually preceded by a trivial increase of temperature and slight interference with appetite and rumination. The milk is to a small extent lessened, of lighter specific gravity, and thinner than normal.

The udder and teats become hot and sensitive; the latter are slightly swollen, and at their bases especially, isolated pale red papules appear, varying in size from a lentil to a pea. Similar papules appear on the surface of the udder. In the course of one or two days the papules increase in size and develop into vesicles. These are depressed or umbilicated in the centre, elliptical in form on the teats, but circular on the udder. They have a swollen, firm margin, and when the skin is non-pigmented, a red areola is observed surrounding them. By the eighth or tenth day the vesicles increase in size, the contents become purulent, pustules being formed. These gradually dry up, forming dark brown shining crusts. Up to the fourteenth day the crusts dry and thicken, and by the twentieth day they usually become detached, leaving pale red, smooth, shallow depressions, which form permanent "pits" after the skin has healed. The eruption on the teats seldom passes through these regular stages, as the vesicles usually become ruptured by the hands of the milker, and slow-healing sores may result.

In rare cases the eruption may spread to other parts of the body, such as the head, inner surface of the thighs, the back, throat, chest, etc. Cases are recorded in which the eruption has appeared on the scrotum of the bull.

Bulls, oxen, and young cattle are rarely affected, but occasionally the virus may be conveyed to them by contaminated litter. In calves the eruption is generally situated on the muzzle, lips, and around the nostrils. The pustules have the same characters as those found on the udder.

SEQUELÆ AND COMPLICATIONS.

The vesicles ruptured by the hands of the milker may become infected from outside sources, and mammitis may result, with its sequelæ. In some instances the constitutional disturbance is more marked, and the eruption may appear on the mucous membranes, especially in the buccal region, salivation and difficulty in mastication resulting.

In hot climates the disease may assume a more serious character, and become complicated with abdominal pain, emaciation, diarrhœa, and it may even prove fatal.

COURSE AND PROGNOSIS.

As a rule, the course of cow-pox is mild, the duration of the eruption being about twenty-one days. A succession of vesicles may, however, appear, and the various stages of the eruption may be observed on the udder at the same time. Thus the duration of an attack may be prolonged.

Loss may occur through diminution in quantity and an alteration in quality of the milk-supply; also the cow may prove difficult to milk after recovery.

DIFFERENTIAL DIAGNOSIS.

From Epizoötic Aphtha (Foot and Mouth Disease) cow-pox is distinguished—

1. By the character of the eruption. In cow-pox the vesicle is multilocular—*i.e.*, it is divided into compartments by septa. In epizoötic aphtha the vesicle is a single undivided cavity. In cow-pox the vesicle is umbilicated in its centre. In epizoötic aphtha the centre of the vesicle is rounded and convex.

2. By the location of the eruption. In cow-pox the udder and teats are the usual seats of the eruption, the mouth and feet being unaffected; while in epizoötic aphtha the lesions are situated in the latter regions. Also, there is an absence of thickening of skin in epizoötic aphtha.

3. In epizoötic aphtha there is more constitutional disturbance than in cow-pox, the disease spreads rapidly in a herd, and is also conveyed to sheep, pigs, and goats.

Cow-pox only spreads by actual contact, or the virus is conveyed by the hands of the milker, and heifers, oxen, and other animals usually escape the infection.

From the Eruption of Rinderpest cow-pox is distinguished by the presence of lymph in the skin lesions, the skin eruption in rinderpest being of the nature of an epidermic concretion. The virulent infectious nature of rinderpest, the marked constitutional symptoms, and the high mortality, also serve to distinguish the two diseases.

From Eruptions on the Udder and Teats due to Streptococcic Infection cow-pox is distinguished by the character of the eruption. In streptococcic infection the eruption consists of pustules and abscesses of various sizes, and these tend to invade the deeper structures of the udder. The method of healing is by granulation, instead of the formation of a thick crust, as in cow-pox.

From False Cow-Pox.—Many mammary eruptions exist which resemble cow-pox. These have been grouped under the name of "false cow-pox" by Ceely and Hering. Gamgee described varicella or chicken-pox as occurring in cows on the Continent.

The points in the differential diagnosis are said to be as follows: In varicella the vesicles vary in size, their interior is undivided, no areola is present, the pustules form and dry up rapidly, and are covered with a thin crust; also the eruption tends to appear in successive crops, so that the duration of the disease may be prolonged to several weeks.

According to Malcolm,* false cow-pox or varicella is not infrequently met with, and while resembling true vaccinia in some respects there are certain distinguishing features as follows: The eruption in varicella is of a vesicular rather than a pustular character, and no distinct papules precede the formation of the vesicles; the latter are small, superficial, raised on the surface or acuminated rather than umbilicated, and not surrounded by a tumefied raised areola. The crusts formed are thin, and when cast off, no pit-mark is left. Except when the eruption appears in successive crops, the duration of the affection is short, being about seven or ten days.

The disease seems capable of transmission from cow to cow by the hands of the milker.

Some authorities state that these eruptions are probably true cow-pox observed in its last stage, and are transmissible from one animal to another. In some cases a differential diagnosis is difficult, and can only be carried out by inoculating a calf with the contents and scrapings of a pustule. In the case of true cow-pox a typical vaccinal eruption will result. Ehrhardt observed varicella (false cow-pox) to occur as an enzoötic in cows, with transmission of the disease to the milkers of the animals. Similar cases of transmission of the disease to the milkers are recorded by other authorities.

We frequently observe isolated cases of a pustular eruption on the teats of milch cows, but in most instances the pustules are ruptured early by the hands of the milkers, so that it is not possible to determine their character. Sometimes two or three cows in a dairy are so affected, and spontaneous recovery follows without further cases occurring. We have never observed the milkers to become affected in such cases.

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* *Journal of Comparative Pathology and Therapeutics*, March, 1901.

PROPHYLAXIS.

As the disease is spread chiefly from the affected to the healthy by the hands of the milker, affected animals should be kept by themselves, and milked by special attendants, who should not be permitted to milk healthy cows. Unless the milkers have been previously vaccinated, they are liable to contract the disease.

The milk of affected animals should not be used by human beings, as there is a risk that it may become contaminated with the pustular exudate, and might possibly transmit the disease.

The horse is more susceptible to vaccinia than either man or the ox. In the horse it is very liable to become generalised.

TREATMENT.

Cow-pox being a mild affection that runs a definite course, little medicinal treatment is necessary. A saline laxative may be given, and soft diet ordered. The surroundings should be kept clean, and the milkers should be cautioned to avoid rupturing the vesicles; but this is seldom possible when the eruption affects the teats.

When the vesicles are ruptured and the parts become irritable, they should be dressed with boric or hazeline ointment. The hands of the milkers should be washed in an antiseptic solution, and every precaution taken to prevent secondary infection of the affected parts. Should mammitis supervene, suitable treatment for that condition must be carried out. If it becomes necessary to employ a teat-tube, strict aseptic precautions must be observed.

III. ON ANIMAL VACCINATION IN MAN.

Since the time of Jenner the true lymph of cow-pox has been employed in the vaccination of man as a preventive of human small-pox. "Vaccination is not claimed to be an invariable and permanent preventive of small-pox, but in an immense majority of cases successful inoculation renders the person for many years insusceptible" (Osler).

At one time humanised lymph—*i.e.*, that obtained from the vesicles of persons vaccinated—was employed, in consequence of the scarcity of genuine cow-pox lymph. In the present day institutes exist in all countries for the cultivation of the lymph of cow-pox, and glycerinated calf lymph has come into general use. For its cultivation calves from five to twelve weeks old are employed. These are inoculated with the lymph on the inside of the thigh and on the abdomen, strict aseptic precautions being carried out. The vesicles become mature in four or five days, and the lymph is carefully collected and preserved.

The necessity for careful veterinary examination of calves used for this purpose is apparent from the fact that the presence of actinomycosis in vaccine virus was discovered twenty-four times in ninety-five cultures from the virus of five producers in the United States, while the bacillus of tetanus was discovered in ninety-five cases (also in America), sixty-three of which occurred in 1901 (Osler).

Although no case of invaccinated tubercle is on record, still, common sense would dictate that precautions should be taken to insure freedom from this disease in the calves employed, because tuberculosis is known to affect even calves of this age.

In view of the possibility of calves employed for the production of calf lymph becoming affected with foot-and-mouth disease (see p. 772), precautions should also be taken to prevent the occurrence of such a catastrophe.

IV. HORSE-POX (VARIOLA EQUINA).

French : Vaccine du cheval ; Horse-pox.
German : Pferde-pocke.

Definition.—An acute contagious disease of the horse, characterised by slight febrile disturbance and the appearance of a typical variolous eruption, especially on the buccal mucous membrane and on the skin in the hollow of the pasterns.

GENERAL REMARKS.

Horse-pox is a comparatively rare disease. Towards the end of the eighteenth century it was recognised by Jenner in the valley of the Severn, but he erroneously associated it with that affection of the horse's heels known as "grease." It has appeared at various times in France and Germany. In 1877 an outbreak of the disease occurred in Montreal, Canada, and was described by McEachran in the *Veterinary Journal*, August, 1877. Several hundred horses were affected, but the origin of the disease was unknown. Small-pox was very prevalent in the city at the time. Several cases of transmission of horse-pox to man were observed during the outbreak. According to Friedberger and Fröhner, the disease is now very rarely met with.* It is occasionally seen in Great Britain, more especially amongst imported Iceland ponies. The similarity of the disease to contagious pustular stomatitis must be borne in mind (see p. 586). The majority of French authorities, and also Hunting, believe that equine variola and contagious pustular stomatitis are identical diseases.

ETIOLOGY.

The virus is conveyed from affected animals to the healthy by various ways. The buccal mucous membrane may become infected as the result of eating food contaminated by the virus. Infected litter, harness, clothing, bandages, brushes, halters, sidelines, hobbles, etc., may infect a wound or abrasion of the skin, and thus produce the disease. The stables lately occupied by affected horses also prove a source of infection. The hands of the groom attending affected animals may convey the disease, especially to the skin in the hollow of the pastern, where abrasions and slight cracked heels so frequently occur, and offer a ready entrance for the virus. The mucous membrane of the lips, mouth, etc., may become infected by the animals licking or biting affected animals having lesions in these parts. The nasal mucosa may also become the seat of the disease, if the virus gains access thereto. Infection may take place by coitus when the penis or the vagina is the seat of the specific eruption. By this means an affected stallion may occasion an enzoötic of the disease.

According to Nocard and Leclainche, the contagion may possibly be spread for a short distance by the intermediation of dust containing the virus, as desiccation does not alter the virulence of the latter, and the disease can be produced experimentally by introducing the virus into the respiratory passages. According to Friedberger and Fröhner, recently vaccinated human beings may infect the horse and produce in the latter typical horse-pox. The same authors state that the variolæ of cows, sheep, and man, can be transmitted to horses.

SYMPTOMS.

The febrile symptoms and constitutional disturbance may be so slight as to escape observation. The eruption may be located on the limbs, or on the mucous membrane of the mouth and lips, or on the nasal mucosa, or it may appear on the skin in various parts of the body, or in rare cases it may become generalised. In the limbs it generally commences in the hollow of the pastern ; the skin becomes swollen and very sensitive, and, if non-pigmented, bright red spots are seen, on which the papules appear. In this region the regular transition of papule to vesicle and pustule may not be observed, but instead the surface is covered by an exudate which forms into a thick yellowish crust, causing agglutination of the hairs, and an ammoniacal odour is given off from the parts. In from fifteen to twenty days the crust falls off, leaving a granulating surface.

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* This is probably due to the fact that the Germans do not consider stomatitis pustulosa contagiosa to be a form of variola, while the French hold the opposite view.

The disease may be limited to one hind-limb or to one fore and one hind limb ; it may also extend to the anterior surface of the metatarsal region. Occasionally the swelling of the limb may be considerable, and the aspect resembles that seen in cases of "grease."

When cleanliness is neglected or irrational treatment is adopted, complications occur; the pustules become confluent, and sloughing of the skin or deep fissures in the heels may result. Suppuration of the subcutaneous tissue has also been observed.

On the Buccal Mucous Membrane the eruption in the early stages consists of vesicles of a smooth pearly appearance, with an absence of a central depression. They occur on the mucous membrane of the lips, the inferior surface of the tongue and on the borders of its free portion, on the mucous membrane of the cheeks, and on the gums. They rupture as the result of friction during mastication, and small depressed erosions of the mucous membrane are produced, having a deep surface of a dark red colour. Cicatrisation generally occurs rather rapidly. Profuse salivation and some difficulty in mastication accompany the eruption. In most cases pustules appear at the same time on the skin of the lips and around the nostrils.

On the Nasal Mucous Membrane transparent vesicles of various sizes may be observed in some cases. After about twenty-four hours their contents become turbid, and they appear of a yellowish colour. They rupture, and small circular superficial erosions result, which rapidly heal without leaving any cicatrix. A thick yellowish muco-purulent nasal discharge may accompany the eruption ; also a slight tumefaction of the submaxillary glands. Petechiæ may be observed in some cases.

In rare cases the eruption appears on the conjunctiva, and is accompanied by conjunctivitis, turbidity of the cornea, lachrymation, and photophobia. Although the symptoms appear serious, the termination is generally favourable.

On the Genital Mucous Membrane.—In the stallion the eruption is situated on the mucous membrane of the penis and the terminal portion of the urethra. The pustules are of various sizes. They become ruptured by coitus, and superficial erosions result, which heal so rapidly in some cases that the presence of the affection may be overlooked. If the pustules, however, are numerous and become confluent, suppuration may occur in the vicinity of the erosions, and in some grave cases secondary infection produces extensive swelling of the free end of the penis, tumefaction of the sheath, and paraphimosis as a result.

In the mare the eruption occurs on the vaginal mucous membrane, and generally also on the skin in the vicinity of the vulva, anus, perineum, udder, and internal aspect of the thighs. The lips of the vulva are swollen, and the pustules appear rapidly.

On the Skin, the eruption may be confined to certain regions, or may be generalised over the surface of the body. It may in some cases occur in successive crops, so that pustules of various ages may be seen on the same animal. The eruption on the head is usually found on the inferior aspect of this region, and coincides with the buccal eruption. Complications may occur when the pustules become confluent; the entire face may be invaded, and secondary infections are produced, resulting in the formation of superficial abscesses or diffuse lymphangitis of this region.

COURSE AND PROGNOSIS.

Horse-pox is a mild affection that runs a definite course. If, however, irrational treatment be adopted, or the animals be kept under insanitary conditions, complications are likely to occur, and the course of the disease becomes protracted.

DIFFERENTIAL DIAGNOSIS.

The following diseases may be confounded with horse-pox.

Grease.—This is distinguished from horse-pox by its non-contagious nature, by the absence of the typical eruption and of the buccal lesions. When, however, horse-pox affects the limbs only, and the pustules become confluent, with occurrence of free secretion, considerable difficulty will arise in the differential diagnosis in isolated cases. But grease is a very common affection, while horse-pox is a very rare one.

Glanders.—For the differential diagnosis, see Glanders (p. 88).

Stomatitis Pustulosa Contagiosa.—As already remarked, many observers consider this affection to be identical with horse-pox. When horse-pox affects the mouth and the skin of the lips and nostrils, and not the heels, a differential diagnosis may be impossible (see Contagious Pustular Stomatitis, p. 596).

Dourine.—For the differential diagnosis, see section on Dourine.

Strangles.—The exanthematous form of strangles may simulate horse-pox. For the differential diagnosis, see p. 815.

PROPHYLAXIS.

Isolation of affected animals should be carried out, and their attendants should not be permitted to come into contact with healthy horses. Precautions should be adopted in the disposal of contaminated litter, fodder, etc., and the feeding utensils, brushes, bandages, halters, harness, clothing, etc., should be thoroughly disinfected. The attendant should be cautioned as to the contagious nature of the disease to human beings, and the necessity of adopting precautions to prevent accidental inoculation.

TREATMENT.

Attention to the sanitary condition of the stables is of importance. Little medicinal treatment is required. Soft diet and the administration of saline diuretics in the food constitute all that is necessary in ordinary cases.

Poultices and fomentations to the lesions in the heels are contra-indicated, as they tend to render the pustules confluent, and so induce a protracted course. An application composed of zinc oxide with boric acid gives good results.

When salivation is profuse and the mouth so tender as to interfere with feeding, a mouth-wash composed of chlorate of potash, belladonna, honey, and water, may be prescribed with benefit. The diet in such cases should consist of oatmeal gruel, linseed tea, milk, etc.

V. SHEEP-POX (VARIOLA OVINA).

French : Clavelée ; Claveau ; Picotte.
German : Schaf-pocken ; Schaf-blattern.

Definition.—An acute infectious and inoculable affection, occurring as an epizoötic peculiar to sheep, characterised by a typical variolous eruption on those parts of the body either devoid of wool or scantily covered with it, and by serious constitutional symptoms, high mortality, and the intense and diffusible nature of its infection.

GEOGRAPHICAL DISTRIBUTION.

Sheep-pox appeared in Great Britain in 1847, having been brought thereto by infected sheep from Denmark. The outbreak lasted for four years and occasioned great losses. It again appeared in 1862, but was stamped out in four months by the adoption of effective measures. Since that time no cases of the disease have occurred in Great Britain and Ireland. It still exists in the Balkans, Italy, Spain, the South of France, Algiers, India, and other parts of Asia.

America, Australia, Tasmania, and Japan have remained free from the disease. An outbreak occurred in German South-West Africa in 1910, and was investigated by Ostertag.

GENERAL REMARKS.

There is considerable difference of opinion amongst authorities as to whether sheep-pox can be transmitted to other animals. According to Nocard and Leclainche, it cannot be conveyed to other animals either by natural contagion or by experimental inoculation. On the other hand, Friedberger and Fröhner state that it may be transmitted to cattle, goats, pigs, and horses, both naturally and experimentally.

As already remarked, cow-pox can be transmitted to the sheep, and produces a local reaction or eruption, but it does not confer the least immunity to sheep-pox.

With reference to the possibility of the transmission of sheep-pox to man, definite evidence is not forthcoming. According to Nocard and Voisin, the cases recorded of accidental infection of sheep-pox in man are at least doubtful. It is said that no case of transmission of the disease to man has occurred amongst the attendants of large numbers of affected sheep.

No definite knowledge exists as to whether human variola can be transmitted to the sheep. Küchenmeister and Tappe are said to have produced a general variolous eruption in the sheep by direct introduction of the virus of human small-pox into the blood.

According to Nocard, the blood of variolous sheep is not virulent at any period of the disease. Ostertag states that sheep-pox in South-West Africa was transmissible by blood inoculation. Further investigation is desirable in connection with the subject.

Nature of the Contagion.—A specific micro-organism for sheep-pox has not been discovered. The experiments of Borrel (1902) tend to demonstrate that the disease depends on an ultra-visible virus. "When material from the pustules was largely diluted with water, and the mixture was passed through a Berkefeld filter, the filtrate was sometimes infective. On the other hand, the Chamberland F filter always arrested the virus. A series of other graduated porcelain filters, all more porous than the F pattern (F^4 up to F^{10}), almost always allowed the virus to pass." *

The infective matter is contained in the structure of the specific pustules, in their contents, and in the crusts. According to some authorities, including Nocard, it is limited to the above, the blood is not virulent, and the products of secretion or excretion are only virulent when they have been contaminated by the contents of the pustules or by the crusts. Other authorities state that the milk proves virulent, and that the blood becomes infected in severe cases. As already mentioned, Ostertag found that the disease was transmissible by blood inoculation.

The infective matter is both fixed and volatile; it possesses marked powers of diffusion, and can be carried to animals at some distance from those affected either by the air, or by dust, or by intermediary bearers. Its vitality is well marked, and in the dried condition, when protected from air and light, it is said to remain virulent for an indefinite period, and to be unaffected by the action of cold. In ordinary sheds it has remained active for five or six months. Animals that have recovered from the disease remain capable of transmitting the infection for a period of six weeks after convalescence. According to Ostertag, cases were capable of conveying infection for three months after recovery, the infecting agent being retained in the wool.

Exposure to sunshine, high temperature, putrefaction, and to the action of various antiseptic agents, destroys the virulency of the infecting agent.

ETIOLOGY.

Sheep-pox always arises from contagion, either direct or indirect. Thus, infected sheep, or those recently convalescent or subjected to preventive inoculation, also infected wool, sheep-skins, fodder, litter, manure, railway-trucks, sheds, etc., may be sources of infection. The attendants on affected sheep, and even dogs in contact with the latter, may convey the disease. Congenital infection is said to occur, and death of the fœtus *in utero* from the disease is possible; also lambs may show evidences of the affection when born.

IMMUNITY.

One attack is said to confer permanent immunity from the disease. Preventive inoculation confers immunity for a year or longer (see p. 588). Newly-born lambs, or those living exclusively on a milk diet, are said to remain immune for a short time, or to prove somewhat refractory to infection. Lambs born of ewes that had been inoculated, or had suffered from an attack of the disease towards the later stages of gestation, are also said to prove immune. According to Nocard, in countries where the disease has been prevalent for long periods, such as in Algeria and Brittany, certain breeds of sheep appear to be in a large measure immune.

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* McFadyean, "The Ultra-visible Viruses," *Journal of Comparative Pathology and Therapeutics*, June, 1908.

MORBID ANATOMY.

The lesions depend on the severity of the attack. Decomposition sets in early. Gelatinous exudation into the subcutaneous and intermuscular connective tissue is observed in the affected regions. The nasal mucosa, larynx, trachea, and bronchi, may show congestion, ecchymosed spots, vesicles, pustules, and ulcerations, and the presence of a tenacious muco-purulent exudation. Pustules may also occur on the buccal mucous membrane, the pharynx, and in some cases on the cornea, and the eruption may be generalised on the skin.

Pulmonary lesions may be present, such as croupous or broncho-pneumonia; also lesions of the cerebral meninges. In some instances erosions and ulcers may be found in the gastro-intestinal mucous membrane. Evidences of septicæmia or pyæmia may be present; also lesions of the joints. Extensive suppuration, swelling, and even gangrene of the affected areas of the skin, may occur in the malignant form of the disease.

PERIOD OF INCUBATION.

The average period is said to be from four to seven days, the minimum two to three days, and the maximum twenty days. Ostertag found the incubation period to be from eight to twelve days.

SYMPTOMS.

Three forms of the disease are generally described—viz., (1) *The Discrete, Regular, or Benign*; (2) *the Confluent, Irregular, or Malignant*; (3) *Variola Hæmorrhagica.*

1. *The Discrete or Regular Form.*—In the early stages mild constitutional disturbance occurs, such as a rise in temperature to 105° F., dulness, interference with appetite and rumination, and slight stiffness in progression. (A well-known authority on the disease, the late Professor Simonds, stated that he had never observed the fever to precede the eruption.)

The initial symptoms are followed by high fever; the temperature may reach 107° F., and rigors, arching of the back, accelerated respirations, frequent pulse, congested conjunctivæ, and a serous nasal discharge, may be observed. Ostertag observed in the outbreak mentioned, catarrhal symptoms, diffuse swelling of the lips and of the pituitary mucous membrane. The eruption appears on those parts of the body which are free from wool or but scantily covered with it, such as the axillæ, the sternum, the abdomen, the inner surface of the thighs, the head, eyelids, nostrils, and lips.

Cutaneous congestion occurs prior to the appearance of the eruption, but this is only visible when the skin is white. In about two days small deep-seated red spots are observed in the congested areas of skin, and in twenty-four hours they become firm rounded papules, which in two or three days more assume a vesicular character. The vesicles in sheep-pox are rounded or flattened on their summits, seldom umbilicated, and are usually surrounded by red areolæ. On the fifth or sixth day they change into pustules, their summits become of a yellowish colour, and the contents are turbid. They increase in size, and the swelling in the surrounding skin becomes more marked, especially in the region of the head.

Desiccation next occurs, and a greyish crust forms on the surface, which alters to a thick scab of a dark brown colour. After five or six days more the crust falls off, and small depressions or cicatrices are left, which may remain devoid of wool. In some cases the crust is thrown off in the form of scales. The eruption may be found in various stages in the same animal.

The fever may abate on the first appearance of the eruption, but it rises again during the pustular stage. When the pustules are numerous and become confluent, hyperpyrexia is observed. In severe cases the eruption invades the conjunctivæ, the nasal mucosa, the mouth, pharynx, larynx, bronchi, stomach, and intestines. In such cases salivation, cough, dyspnœa, dysphagia, diarrhœa, swelling of the head, and a muco-purulent nasal discharge, are observed. The eruption may invade the cornea, and blindness may result.

In favourable cases the fever and constitutional symptoms subside when desiccation occurs. Occasionally a secondary eruption appears, consisting of papules, which do not proceed to the pustular stage.

2. *Confluent or Malignant Form.*—This is characterised by continued hyperpyrexia, severe constitutional symptoms, marked prostration, total loss of appetite, laboured respirations, inability to assume the standing posture, and by the presence of extensive œdematous swellings on the head, limbs, sternum, and abdomen. The eruption becomes generalised,

the vesicles become confluent, large pustules are produced containing fœtid pus, extensive areas of suppuration result, gangrene of the skin may occur in parts, and the body exhales a fœtid odour. Occasionally death may take place in the absence of any eruption, probably depending on toxins acting on the nervous system. Fœtid diarrhœa sets in when the gastro-intestinal canal is involved. In pregnant ewes abortion generally occurs.

3. *Variola Hæmorrhagica.*—In this form hæmorrhages occur in the skin and mucous membranes in various parts of the body, and also into the vesicles. The latter undergo gangrenous disintegration, due to hæmorrhagic infiltration. Hæmaturia is also observed (Friedberger and Fröhner).

COMPLICATIONS AND SEQUELÆ.

Septicæmia or pyæmia, with the formation of metastatic abscesses in the joints and in the cerebral meninges, may occur. Shedding of the feet and sloughing of the entire digital structures have been observed in some cases. Suppuration of the lymphatic glands and subcutaneous abscesses may occur in various parts of the body. Ulceration and sloughing of mucous membranes, septic pneumonia, ulceration and gangrene of the nasal bones and cartilages, and chronic lameness, have also been observed.

COURSE AND MORTALITY.

In the discrete form the duration of the disease is about three weeks, and the mortality about 7 per cent.; while in the malignant form the mortality may average from 20 to 30 per cent., or even 50 to 90 per cent.

In those that recover convalescence is very prolonged, and permanent lameness or blindness may result. In very hot or in damp, cold weather, with insanitary surroundings, and in the case of old, feeble, or very young animals, the mortality is greatly increased. The losses due to the disease include not only those resulting from deaths, but also those depending on the occurrence of abortion, shedding of the wool, diminution of body-weight, and other permanent after-effects.

DIFFERENTIAL DIAGNOSIS.

The diagnosis is based on the typical character of the eruption, the severe constitutional symptoms, and the marked infectious nature of the disease. Pustular eczema, contagious pustular dermatitis, and urticaria of the head, simulate sheep-pox to a certain extent.

On the Continent it is found that the small punctures of the inferior abdominal wall resulting from contact with the stems of the *Juncus* *acutus* may simulate the eruption of sheep-pox. Continental authors draw attention to some enzoötic eruptive diseases occurring in sheep, which bear some resemblance to sheep-pox, but constitutional symptoms are absent.

Sheep-pox has been mistaken for "blue-tongue" in German South-West Africa (see section on Blue-Tongue).

PROPHYLAXIS.

Sheep-pox being a disease notifiable under the Diseases of Animals Act, in the event of a case occurring, the owner or veterinary surgeon attending is compelled to report to the local authority, who will deal with the matter.

Ostertag advises the destruction of all infected and suspected animals, the quarantining of farms and areas, the inoculation of all sheep in infected areas, and the prohibition of movement within these areas. The period of quarantine advised is three months for a naturally infected flock, but this period could be reduced to one month by dipping in 2½ per cent. Jeyes' Fluid, creolin, or creosote soap solution. The carcasses should be destroyed by fire.*

PREVENTIVE INOCULATION, ALSO TERMED "OVINATION" OR "CLAVELISATION."—Preventive inoculation by means of the lymph of sheep-pox has been carried out from early times, and confers immunity for a year or longer. It is only to be recommended when the disease has already appeared in a flock, or in secluded districts where the disease is generally prevalent. Ovination produces as a rule a milder and more local affection than ordinary sheep-pox. Sheep coming from Algeria cannot be imported into France until preventive inoculation against sheep-pox has been carried out a month before embarkation (Signol and Cagny).

Technique of Ovination.—The virus may be taken from a young sheep affected with the discrete and benign form of the disease, or from the centre of the large pustules obtained by inoculating a healthy sheep. Various methods of preparing the virus are adopted. At the present time it is prepared in the Pasteur Institute at Algiers, and very large numbers of sheep are inoculated yearly.

In Brémond's method the inoculations are made on the under surface of the tail or at the extremity of the ear, and a subepidermic puncture with a hollow needle or a lancet assures the penetration of the virus. Not more than one puncture should be made, as otherwise a generalised eruption might be produced; and the subcutaneous tissue should not be penetrated.

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* Paper read before the Transvaal Veterinary Association by Professor Ostertag, December, 1910.

Soulié and Emery have adopted the intradermic inoculation of diluted virus by means of a hypodermic syringe, and obtained good results in Algeria.

The result of ovination is to produce a local eruption and very slight general disturbance. Occasionally a discrete generalised eruption may result. Inoculated sheep are capable of spreading the disease.

Inoculations should not be practised on very young lambs, as grave results may follow. Pregnant ewes towards the end of gestation may abort as the result of ovination.

In some countries ovination is said to prove very safe, the mortality not exceeding 1 to 5 per 1,000. Ostertag gives the proportion of fatalities after inoculation as 1 per cent. In the centre and north of France the mortality may reach 1 to 3 per cent., and in a few instances 10 to 20 per cent. has been recorded.

The duration of immunity is not definitely determined. It generally lasts longer than a year. The after-treatment of inoculated sheep consists in protection against unfavourable weather and attention to diet. If after ten or twelve days the inoculation has not been successful, re-inoculation should be carried out.

TREATMENT.

This can only be advisable in benign cases, and as the disease is a notifiable one, it can only be carried out by permission of the authorities. Attention to diet and to the surroundings is of first importance. Cleanliness and the application of antiseptic lotions, etc., constitute the local treatment. In severe and malignant forms treatment of any kind has little or no effect.

VI. GOAT-POX (VARIOLA CAPRINA).

By H. GRAY, M.R.C.V.S.

French: Variole de chèvre.
German: Pockenkrankheit bei Ziegen.

The goat is subject to a variola peculiar to its own species. It is independent of vaccinia and sheep-pox, and is never transmitted by natural contagion to the cow, horse, or sheep. On the other hand, there is an abundance of experimental and clinical evidence to show that sheep-pox is not transmitted to the goat; but, according to Hervieux, cow-pox is readily conveyed to the goat, and this animal may be utilised for the production of vaccine (cow-pox virus).

This disease has often been encountered as a special enzoötic in Norway. G. Hansen, who described it in 1879, observed in Central Norway outbreaks in 1867-1869 and 1874-1875. In 1865 eleven cases of variola serosa (water-pox), and in 1868 four cases of goat-pox, were reported in South Norway. In 1880 Dr. Cæsar Boeck, of Christiania, published a summary of forty cases observed by him in 1871 in Thelemarken, South Norway. Hering says it is a rare disease in Germany. Hertwig describes an outbreak and gives illustrations of the eruption. In 1832 it occurred in Geislingen, attacking ten out of a herd of fifty-four. In 1847 Spinola observed a similar outbreak (Fleming). Miquet and Jousseaume observed in 1884 an epizoötic of it in the regions of Mascara and Fuendah, Oran, Algeria. Bonvicini of Pisa (1898) and Marcone of Naples (1900) studied it respectively in the provinces of Modena and Reggio de Calabra. It has also been observed in Spain in the province of Murcia, and Perez Blasco, in 1881, studied an extensive outbreak occurring in Descarga-Maria (Nocard and Leclainche). Cases have also occurred in Persia.

It attacks without exception the males, kids, and those in milk.

The period of incubation is within a week. The duration of the disease is about one to three weeks. The mortality is not very great—about 2 per cent.; but in one flock of 150 the mortality was thirteen. It occurs principally during the summer months.

Symptoms.—The initial stage is manifested by a slight elevation of temperature, the coat lacks lustre and is staring, the animal is dull and off its feed. After a brief period the lips become swollen, and there is a profuse whitish muco-purulent discharge from the eyelids and nostrils. Sometimes a cough occurs. In the milch goat there is a diminution of the milk-supply, the act of milking causes great pain, and the milk is rather viscid and sometimes contains blood. The eruption appears on the mouth, mammary gland, abdomen, inside the thighs, occasionally on other parts, and in the male on the scrotum.

It first appears as a reddish spot, and then as a hard, flattened papule with a red areola, having the diameter of a lentil to that of a split-pea, on a thickened skin. About the third to the fifth day the pock contains a thin serous fluid, which two or three days later becomes purulent, and ultimately dries up, forming a thick, brownish, dry crust or scab. The resulting cicatrix is more or less stellate and of a light hue.

A phlegmonous mastitis is sometimes produced, and not infrequently suppurating furunculi form on the udder.

Lesions.—The eruption is found in various parts, and sometimes becomes generalised ; occasionally it is confluent. Pustules occur on the mucous membrane of the stomach and intestines, such as are often witnessed in malignant sheep-pox.

Treatment.—The same means should be adopted as in the case of the variolæ of the other domesticated animals.

Transmission to Man.—According to Hansen and others, it may be transmitted to man.

VII. SWINE-POX (VARIOLA SUILLA).

By H. GRAY, M.R.C.V.S.

Swine-pox is a contagious and inoculable eruptive disease that, like the variolæ of other animals, runs a regular or irregular course, and is benign or malignant in its effects. The eruption is discrete or confluent. One attack confers a relative immunity.

It is a very contagious malady, resembling in its course, complications, and infectivity, sheep-pox. Young pigs are most susceptible, although old swine develop the disease. Its mortality is sometimes very high, reaching as much as 25 per cent.

It is said to be transmissible to man, the sheep and goat. It has not been conveyed to the horse or cow. According to Gerlach and Bollinger, sheep-pox may be transmitted to the pig ; but Nocard and Leclainche state that inoculation fails to convey ovine variola to this species. Vaccinia and, according to some authorities, human variola may be transmitted to the pig.

According to Felix, Grêve, Spinola, Pozzi, Hering, Gerlach, Bollinger, and other authorities, swine-pox has been observed in Austria, Denmark, France, Germany, Hungary, and Italy. Fleming says, on the authority of "a very observant and competent veterinary surgeon," it was a common disease in the county of Kent. In all probability some writers may have confounded swine erysipelas and the eruption of swine fever with swine-pox. Poenaru of Bucharest studied this disease in 1906 (*vide Archiva Veterinarâ*, April, 1907 ; and *Journal of Comparative Pathology and Therapeutics*, vol. xx., p. 159).

Symptoms.—In the initial stage there is an elevation of temperature, the animal is dull, off its feed, and seeks shelter as if cold. This goes on for some days, when an eruption appears on the back, abdomen, chest, neck, head, and inner surface of the thighs. This eruption is first observed in the form of petechial spots, which soon become papules, and on the fifth or sixth day vesicles, and on about the ninth or tenth day pustules form. Their contents begin to dry up and form crusts or scabs, which are thrown off in a few days, leaving a well-defined cicatrix.

Treatment.—Similar means should be adopted as for the other variolæ.

VIII. WATER-POX (VARIOLA SEROSA; WASSER-POCKEN).

This disease has been occasionally observed in young suckling pigs. The pock on the sow's teats quickly bursts, and the discharge is ingested along with the milk by the young animal, which a few days after develops a yellowish-brown eruption around the eyes and nostrils, and then all over the body.

If the young pigs are removed early from their dam the consequences are not serious, but otherwise the entire body becomes covered with the eruption, and the young animals die very rapidly.

In Germany the peasants bathe the sick animals in bran water or chamomile-tea.

IX. DOG-POX, PUPPY-POX (VARIOLA CANINA).

By H. GRAY, M.R.C.V.S.

The question might be asked, "Has the dog a variola of its own?" Old writers, such as Fournier, Barrier (1803), Escovar, Vilalba, Prinz, and Waldinger, considered that distemper resembled variola. Jenner even went further, and asserted it was a variola, and highly recommended for its prevention inoculation with cow-pox virus or vaccine. Viborg (1795), Saccho, Hamont, Nauche, and others, were firm believers of Jenner. Trasbot resuscitated in 1879 this old theory, and maintained it up to his death. He says, in answer to his critics, "that because cow-pox virus does not give immunity to distemper it does not establish that canine distemper is not a variola; the variola of the sheep is not inoculable to the majority of animal species." Blaine (1817), speaking of distemper, says: "In some the disease will make its attack by a universal eruption over the body, particularly under the belly and down the inside of the thighs. This eruption is pustular, and the pustules are not very dissimilar to those of the small-pox. In one year in particular almost every case that fell under my notice was accompanied by this appearance." Youatt is silent upon this eruption in distemper, but described small-pox as a different disease from distemper, no doubt being influenced in this view by the observations of Gohier made in the Lyons Veterinary School in 1809.

Symptoms.—As described by Youatt, " the skin of the abdomen, the groin, and the inside of the forearm, becomes of a redder colour than in its natural state, and sprinkled with small red spots irregularly rounded. They are sometimes isolated, sometimes clustered together. The near approach of this eruption is announced by an increase of fever. On the second day the spots are larger and the integument is slightly tumefied at the centre of each. On the third day the spots are generally enlarged, and the skin is still more prominent at the centre. On the fourth day the summit of the tumour is yet more prominent. Towards the end of that day the redness of the centre begins to assume a somewhat grey colour. On the following days the pustules take on their peculiar characteristic appearance. On the summit is a white circular point corresponding with a certain quantity of nearly transparent fluid which it contains, and covered by a thin and transparent pellicle. This fluid becomes less and less transparent, until it acquires the colour and consistence of pus. The pustule during its serous state is of a rounded form. It is flattened when the fluid acquires a purulent character, and even slightly depressed towards the close of the period of suppuration, and when that of desiccation is about to commence, which ordinarily happens towards the ninth or tenth day of the eruption. The desiccation and the desquamation occupy an exceedingly variable length of time, and so, indeed, do all the different periods of the disease. What is the least inconstant is the duration of the serous eruption, which is about four days, if it has been distinctly produced and guarded from all friction. If the general character of the pustules is considered, it will be observed that, while some of them are in a state of serous secretion, others will only have begun to appear.

" The eruption terminates when desiccation commences in the first pustule, and if some red spots show themselves at that period of the malady, they disappear without being followed by the development of pustules. They are a species of abortive pustules. After the desiccation the skin remains covered by brown spots, which by degrees die away. There remain no traces of the disease, except a few superficial cicatrices on which the hair does not grow.

" The causes which produce the greatest variation in the periods of the eruption are the age of the dog and the temperature of the situation and of the season. The eruption runs through its different stages with much more rapidity in dogs from one to five months old than those of greater age. I have never seen it in dogs more than eighteen months old. An elevated temperature singularly favours the eruption, and also renders it confluent and of a serous character. A cold atmosphere is unfavourable to the eruption, or even prevents it altogether. Death is almost constantly the result of the exposure of dogs having small-pox to any considerable degree of cold. A moderate temperature is most favourable to the recovery of the animal. A frequent renewal or change of air, the temperature remaining nearly the same, is highly favourable to the patient; consequently, close boxes or kennels should altogether be avoided."

This description of supposed canine variola lengthily quoted from Youatt, together with other remarks of his, not mentioned here, must appeal to practitioners who have made a careful clinical study of canine distemper. They will probably come to the conclusion that, if it is a fair account of dog-pox, then distemper is no other disease than canine variola; with reference to the latter Fleming says, without stating his authorities, "it is a well-known disease to veterinarians." His description, like that of Youatt, corresponds in every detail to that of the eruptive phase of distemper. On the other hand, the description of the eruptive phase of distemper given by Camille Leblanc, Trasbot, and other authorities, agrees *in toto* with the clinical picture of dog-pox mentioned by Youatt and Fleming.

That the dog successfully contracts vaccinia by inoculation, although the pock may not as a rule be very characteristic, is beyond question. But it does not give immunity to distemper, as proved by the experiments of Professor William Sewell in 1799, of Youatt, Blaine, Urbain and Camille Leblanc, Hertwig, Gohier, Chauveau, and others, notably those of Dupuis, of the Brussels Veterinary School, who carried out numerous investigations with vaccine during the years 1883, 1884, and 1885, and drew from the results the following conclusions:

1. That vaccine is transmissible to dogs, with its specific characters.
2. That a first vaccinal inoculation generally preserves against a second.
3. That intravenous, subcutaneous, and peritoneal injection gives immunity against the vaccine without the development of any external manifestations.
4. Finally, *that the vaccine cannot by any means be considered a certain means of preserving the dog against distemper.*

A few instances are on record of dogs having contracted small-pox from man and other animals. Grêve is said to have inoculated eight dogs with the virus of human small-pox, and three of them became covered with pustules and died. The results of these old experiments should be received with grave doubt, because the conditions under which the experiments were carried out were not known, and it is quite possible the dogs were in reality suffering from canine distemper at the time of the experiments.

X. AVIAN VARIOLA, CHICKEN-POX, GOOSE-POX, PIGEON-POX (VARIOLA GALLINARUM, ANSERUM, AND COLUMBARUM).

The older authorities have described variola occurring in poultry, but this is in all probability the epitheliosis of the present day, which has by the recent researches of Marx and Sticker (1902) been proved to be due to an ultravisible organism, and which many observers erroneously considered was related, if not identical, to bird diphtheria.

For an account of this so-called "avian variola," see Epitheliosis (p. 323).

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CONTAGIOUS PUSTULAR STOMATITIS

By E. WALLIS HOARE, F.R.C.V.S.

Synonym.—Stomatitis pustulosa contagiosa.

Definition.—A contagious and inoculable eruptive disease of the horse, chiefly affecting the mucous membrane of the mouth and lips, also the tongue, and occasionally extending to the nasal mucosa, the conjunctiva, and the skin.

General Remarks.—Many British and French authorities consider this affection as being one of the local manifestations of equine variola. German and Italian authors, on the other hand, assert that it is a disease distinct from variola. In former times the affection has been described by various authors, and, as the lesions in some of the outbreaks were found on the nasal mucosa, the differential diagnosis between it and acute glanders received special attention (Hering, 1856). In 1876 several cases occurred in Berlin, and the pathology and clinical characters of the disease were described by Eggeling and Ellenberger. A translation of the report is contained in the *Veterinary Journal* for November, 1878.

We are not aware whether the disease has ever been met with in Great Britain. We have seen cases in imported Iceland ponies, in which the oral symptoms strongly resembled those described as occurring in contagious pustular stomatitis, but as some of these cases showed an eruption in the heels the disease was probably equine variola. Certainly the two affections resemble each other very closely so far as the lesions on the buccal mucosa are concerned. (See Horse-pox, p. 578.)

According to Friedberger and Fröhner, the differential diagnosis is based on the facts "that equine variola is exceedingly rare, and that it occurs as a rule only on the pasterns." Both these statements, however, are not in accordance with the views expressed by French authors on equine variola. Further investigations seem to be necessary before definite information on the duality of the two affections can be obtained.

In the meantime the most desirable procedure is to follow the German authors, and to give a description of contagious pustular stomatitis, as found in their textbooks and clinical reports of the disease.

Etiology.—The contagium exists chiefly in the saliva and in the mucus from the mouth. The disease can be conveyed from the affected to the healthy by contact, by accidental inoculation of an abrasion or wound of the skin or mucous membranes, and also by the media of infected litter, food, or grooming utensils, etc. It can also be conveyed by experimental inoculation. Horses prove the most susceptible animals; then cattle, sheep, and pigs; it can also be transmitted to birds.

The disease is communicable to man, and cases are recorded in which the lesions appeared on the hands, arms, and conjunctivæ. One attack appears to confer immunity. The virulence of the contagium seems to become weakened and finally disappears after inoculation from animal to animal. Nothing has been determined with reference to the bacteriology of the disease.

Symptoms.—The preliminary symptoms are a moderate degree of fever and very slight constitutional disturbance. The appetite is usually maintained at first, but later on prehension of food is difficult, and profuse salivation occurs.

On examining the mouth, the buccal mucous membrane is found congested and covered with a tenacious secretion. On palpation, small, firm, isolated nodules are felt, which after two or three days increase in number and size, and extend to the cheeks, tip of the tongue, frænum linguæ, inferior surface of the tongue, and upper lip; also to the gums. Suppuration occurs in their centres, and pustules are formed. The summits of these nodules show signs of ulceration, so that in four or five days numerous ulcers with slightly depressed centres are found on the tongue, mucous membrane of the lips, gums, etc.

The ulcers may be isolated, or may become confluent and extend to the submucosa. The eruption may extend to the skin of the upper lip, cheeks, margins of the alæ nasi; also to the nasal mucous membrane, and in rare cases to the conjunctiva and to the skin of the breast, forelegs, and anus. Ulcers may also be formed in these situations. Enlargement of the submaxillary glands, nasal catarrh, and purulent conjunctivitis, may be observed. Healing of the ulcers may commence in from six to eight days.

The average duration of the disease is from twelve to fourteen days.

Contagious pustular stomatitis has been mistaken for glanders. The chief points of distinction are: The ulcers in the former disease do not possess the characteristics of those occurring in glanders. In contagious pustular stomatitis the ulcers show sharply-defined borders; they are generally superficial, and heal in from six to eight days. The cutaneous ulcers are not confined to the course of the lymphatics. Also the disease can be transmitted to cattle by inoculating the latter in the vaginal mucous membrane.

Prognosis.—This is distinctly favourable, and no losses are recorded even in extensive outbreaks.

Treatment.—Beyond attention to hygienic and dietetic details, little treatment is required. Antiseptic mouth-washes may be employed to hasten healing of the ulcers.

Prophylaxis.—Preventive measures are similar to those advised in the case of equine variola (see p. 582).

RABIES

By CAPTAIN E. CLIVE WEBB, A.V.C.

Synonyms.—Canine madness; Rabidus canis; Lyssa; Hydrophobia (human subject).

French: Rage.
German: Tollwut; Hundstollheit; Hundswut; Wutkrankheit.

Definition.—Rabies is an acute specific infectious and inoculable disease, the virus of which affects more particularly the central nervous system, with resulting symptoms of nervous disorder.

HISTORY.

The affection has been recognised from the very earliest times—in fact, it is one of the oldest known diseases of animals. It is mentioned by Plutarch, who asserts that, according to Athenodorus, it was first observed in mankind in the days of the Asclepiadæ, the descendants of Æsculapius, the god of medicine. The disease was known to Homer, and was alluded to by Hippocrates and Democritus. We also find allusions to it in the works of Virgil, Horace, and Ovid.

The disease was described by Aristotle in the fourth century B.C., who writes: "Dogs suffer from madness, which puts them in a state of fury, and all the animals that they bite when in this condition become also attacked by rabies." Thus, even at that early period, the disease was recognised to be infectious.

In the first part of the Christian era Cornelius Celsus referred to human rabies, and employed the term "hydrophobia."

In 1271 it is related that in Franconia rabid wolves invaded the towns and villages, attacking the herds and flocks, and no fewer than thirty persons died in consequence of bites inflicted by them.

In 1604 an epizoötic of rabies broke out in Paris, towards the end of the seventeenth century in Italy, and from 1719 to 1723 in France and Germany.

It appeared for the first time in Peru in 1803. In this year also commenced one of the most remarkable outbreaks of vulpine rabies on record. It began at the foot of the Jura Alps, and continued to 1835, spreading over nearly the whole of Switzerland.

It was common in America from 1779 to 1807, and in England in 1806, according to Blaine. During the latter part of the eighteenth and beginning of the nineteenth centuries the disease spread over the whole of Europe.

GEOGRAPHICAL DISTRIBUTION.

Epizoötics of rabies have occurred from time to time in practically every country throughout Europe, also in North and South America, West Indies, India, Ceylon, Mauritius, North and South China, Cochin-China, Annam, Syria and Turkey, Arabia, North Africa, Rhodesia, and Egypt.

Hayes* gives particulars of an outbreak of disease amongst dogs in South Greenland in 1870, the features of which strongly suggest rabies.

A serious outbreak of rabies occurred in Ontario in 1907-1910. It began at Queenston in May of that year, and gradually extended over Western Ontario. According to Rutherford,† the disease was first introduced by a dog from the State of New York, which crossed the Suspension Bridge at Queenston, and after biting several Canadian dogs, returned to the United States, without at the time attracting any serious attention. Large numbers of horses, cattle, sheep, and swine contracted the disease, and some owners suffered rather serious losses. In 1909 the number of outbreaks reported was 146 ; in 1910 they reached 307 ; and up to March, 1911, 146 were reported. Under stringent regulations the disease is being successfully brought under control.

According to Rutherford, the wild animals have become infected in some of the United States, where the disease exists continually to a greater or less extent.

The disease has been kept out of Australia, New Zealand, Van Dieman's Land, Azores, St. Helena, Madeira, Sumatra, East and West Africa. Rabies may be said, however, to be confined to no particular climate, and, were it introduced into a country at present free from it, there is always the danger of its rapidly spreading. Hence no country can afford to relax in the smallest degree preventive measures, so long as the disease exists in any other country with which there is intercommunication.

In Great Britain and Ireland the disease has been stamped out, the last case occurring in England in 1902. This very creditable result was only brought about by putting into execution the most stringent suppressive measures, and reintroduction has been prevented by rigid inspection and quarantine of all imported dogs.

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* "Open Polar Sea."

† Report of the Veterinary Director-General and Live-Stock Commissioner (Canada), 1910-1911.

ANIMALS AFFECTED.

It is probable that every warm-blooded animal is susceptible to rabies. The disease is most frequently met with in the dog. Other animals of the canine species (wolf, fox, jackal) are possibly just as susceptible, but their habit of life and smaller numbers in civilized countries would not favour its spread and maintenance to the same extent as in the dog, which is under domestication.

The virus of rabies may be said to maintain its existence mainly by propagation in the body of the dog. In most countries its continued existence is bound up with this species, and if dogs ceased to exist, rabies would probably die out in other species also.

From the method of propagation of the disease it follows that it is liable to spread widely amongst animals that use their teeth as weapons of offence. Hence it is that the carnivora are the most active propagators of the disease, and herbivora are infected by them.

Man readily becomes infected, usually by the bite of a rabid dog, but he might also be infected as readily by the bite of a wolf, fox, or jackal, etc.

The disease has been actually observed in the following animals: dog, wolf, fox, jackal, cat, lion, hyena, badger, skunk, horse, cattle, sheep, goat, ass, deer, rats, mice, chickens, pigeons, rabbits, guinea-pig, monkey, and racoon.

ETIOLOGY.

Rabies has long been recognised as a specific disease due to infection alone. Claims to have discovered the causal organism of the disease have been made by a number of observers at different times, but conclusive proof has always been wanting.

At the present time no one doubts that rabies is caused by the propagation of a parasite, and this latter is probably a bacterium. There is also no doubt as to the parasite being an obligatory one, incapable of maintaining its existence outside the animal body, since there is no such thing as spontaneous rabies in any animal.

Remlinger* in 1903 showed that the virus of rabies was filtrable, although prior to this year such was not believed to be the case. He found that the virus of rabies failed to traverse the close-grained Chamberland B and F filters, but proved that under certain conditions the virus is not arrested by the more open-textured Berkefeld filter.

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* *Annales de l'Institut Pasteur.*

From these experiments it may be surmised that the hypothetical causal organism is beyond the present powers of the microscope, and it is customary, therefore, to include rabies in the group of diseases caused by the "ultra-visible viruses."

The results of Remlinger's investigations were subsequently confirmed by other observers (Bertarelli, Mazzei).

"Negri bodies" and their significance as the causal factor will be discussed later, under the heading of Morbid Anatomy.

Method of Infection.—The almost invariable natural method of infection is by inoculation, the inoculating agent being the tooth of a rabid animal and the inoculated material "saliva." It is possible, however, for infection to be brought about by the application of saliva to an abraded surface.

The disease is not transmitted by ingestion or inhalation.

Bacteriology.—Since the causal organism of the disease is still unknown, "bacteriology" is hardly the correct term to apply to a discussion of the "virus," but will suffice for want of a better one.

Distribution of the Virus in the Body.—The central nervous system, particularly the medulla oblongata, appears to be the main seat of propagation of the virus.

The virus is also abundantly present in the salivary secretion, especially that of the parotid gland.

The peripheral nerves also sometimes contain the virus, and occasionally it is present in secretions other than the salivary—*e.g.*, milk. Eckel and Lafosse are said to have successfully inoculated the blood of a goat to a sheep, of a man to a dog, and of a dog to a dog. The vast majority of observers, however, are agreed that blood, as a general rule, is non-infective.

No doubt the virus is capable of multiplying in the connective tissue anywhere, and its first multiplication takes place in the connective tissue where it is implanted.

The virus was first shown to have a special selection for brain and nervous matter by Pasteur. From its seat of implantation in the tissues the virus probably travels to the central nervous system along the nerve trunks. Hence it is that infected wounds are dangerous in proportion to their proximity to the medulla—*i.e.*, wounds about the head and face are much more dangerous and apt to have a shorter period of incubation than those about the extremities. A further factor rendering wounds in the former position more dangerous in man is the fact that these parts are usually exposed, being uncovered by clothing. For this reason there is not the same chance of the teeth inflicting the wounds being cleansed by their passage through the clothing, as would happen in the case of wounds about the extremities (in persons dressed in European clothing). The amount of infective material implanted in the wounds would therefore be greater in the former case.

Period at which the Saliva of a Rabid Animal becomes Infective.—Cases have been reported of infection resulting from the bite of a rabid dog many days before the dog actually showed clinical symptoms of the disease.

In 1890 Nocard and Roux showed that the mixed saliva is always virulent twenty-four, and sometimes forty-eight, hours before the manifestation of any change in the appearance of the dog.

In 1901 Rabieaux and Guinard showed that saliva from the submaxillary gland, removed aseptically from an opening in Wharton's duct, might be virulent for two days before the appearance of any symptoms.

Nocard, Roux, and Nicolas noted that the appearance of fever indicated the secretion of virulent saliva.

The moment of infectivity of the saliva, therefore, is variable. The question, however, is a most important one, since it must be determined what is a safe period to allow between the moment that a dog, apparently healthy, bites a person and the moment at which it shows clinical symptoms of rabies, before it is considered necessary that such a bitten person should undergo antirabic treatment. To be licked on an abrasion must be looked upon as equally dangerous as a bite, from the point of view of treatment.

The question is a difficult one to settle. Professor Galtier, of Lyons, always advised Pasteur's treatment being carried out on every person bitten within a period of eight days before the appearance of symptoms in the offending animal. This is certainly a safe period to adopt.

Resistance of the Virus.—It is destroyed by desiccation and sunlight. The virulence of the spinal cord of a rabbit exposed to desiccation is destroyed in fourteen or fifteen days. Glycerine preserves the virus.

The virus is very resistant to putrefaction, and has been found active in the central nervous system of animals after being buried for varying periods of from two to six weeks (Galtier). It is destroyed by exposure to a temperature of 60° C. for half an hour, or by exposure to disinfectants, of strengths ordinarily used, for one hour.

PERIOD OF INCUBATION.

This varies within wide limits. In the dog and human subject a common period of incubation is five or six weeks.

No definite period can be stated, but it may be taken as roughly between fifteen days and two months for the dog, cat, solipeds, cattle, sheep, swine, and the human subject. Authentic cases are on record, however, of the period of incubation in the case of the dog and human subject running into several months, and other cases, the proofs of which are not conclusive, of the period of incubation extending to one or more years.

In the case of the dog—assuming that it was known to have been bitten by a rabid animal and developed clinical symptoms of the disease a year afterwards—it is difficult to imagine how it could have been possible to keep it under such close observation as to be able to assert that it had not been bitten again at a later date, and that a subsequent but unobserved bite was not the moment of infection.

Without more conclusive evidence these reported long periods of incubation must be accepted with reserve.

Six months is probably a safe period to adopt, with our present knowledge, as the maximum period of incubation in all animals.

I. RABIES IN THE DOG.

It is with rabies in the dog that we as veterinarians are chiefly concerned. If the disease in this species could be kept under control, one need have little fear of it in other species, wild or domesticated.

The dog is the natural reservoir of the disease, from which animal it spreads to other species, and it is by the dog that man is infected in the vast majority of cases. There is, however, a certain percentage of human beings infected by species other than the dog—*e.g.*, wolves, foxes, jackals, etc. In India especially, the latter species is responsible for the infection of a number of human beings (natives). This is not surprising in view of the fact that during the hot weather months the inhabitants sleep in the open, and their clothing is scanty. They therefore fall an easy prey to a rabid animal, should one be in the vicinity.

Seasonal Prevalence.—Statistics show that in France the season of greatest prevalence is during the spring months of March, April, and May. It is suggested that the reason for this is that spring is the season during which bitches come " on heat," and that this factor brings together numbers of dogs, and causes them to fight for the favours of the former.

In this way the disease would be actively propagated at this season. In India I have seen the largest number of cases in the hills during the summer months, but this might be explained by the fact that most owners of well-bred dogs either take or send them to the hills for the hot weather, which procedure collects a large number of dogs together in a comparatively small area.

According to Fleming, there is no seasonal prevalence. The explanation of a large number of cases occurring at one time of year is not because the climate at that particular season has any influence on the development of the disease, but merely that at that particular time external surroundings are more favourable for contact, etc., and the opportunity of contracting the disease is therefore greater.

Sex Predisposition.—Both sexes are equally susceptible, although it is in males that the disease is more often met with. This is readily explained by the fact that the stronger sex are always fighting amongst themselves for supremacy, whereas they respect the weaker sex.

In the human being, males suffer more frequently than females, the explanation of this being that women's extremities are more protected from bites by a flowing skirt.

SYMPTOMS.

The disease is generally manifested in two different forms, which are distinguished as *furious*, and *dumb* or *paralytic*, rabies. There is no satisfactory explanation as to why the disease sometimes takes one form and sometimes another. Other types of the affection are also met with (see p. 607).

Furious Rabies.—The premonitory symptoms of this form are more or less identical with those of the dumb form. The first noticeable symptom is a change in the disposition of the animal. There is no disposition to bite at this stage, but, on the contrary, it is usually manifested by dulness and apathy. The change in disposition in the case of a usually undemonstrative dog may be indicated by a disposition to fawn upon its owner. A noisy dog may become suddenly quiet, whereas a usually quiet dog may take to growling without apparent cause. There is nervousness, manifested by increased sensibility to sound. Restlessness is a marked symptom. The animal cannot rest in one place or position, but is continually changing both. A depraved appetite is an almost invariable symptom, and a rabid dog will swallow all sorts of things foreign to his usual tastes—*e.g.*, straw, bits of string, stones, rags, etc. Most observers have noted a peculiar change in the sound of the voice, but this is not always easy to detect, although it might be apparent to the owner.

Martin* noted catarrh of the mucous membranes, chiefly nasal, as being one of the earliest symptoms, with loss of appetite, and sometimes very high fever (106° or 107° F.).

Intelligence is not altogether destroyed in these early stages, and an animal will recognise a human voice, especially its master's.

There may be a wound or wounds at which infection took place. Such wounds may occasionally be characterised by intense pruritus, which causes the animal to bite itself. In the majority of instances, however, the seat of infection is not apparent.

Commencing nervous derangement is usually manifested at this stage by a peculiar vacant expression, and by snapping at imaginary objects. The peculiar expression is due to dilatation of the pupils from paralysis of the optic nerve. These nervous symptoms are intensified as the disease progresses. The patient becomes more restless, reflex excitability and hyperæsthesia are increased, and the animal now becomes aggressive, will bite at any object held up to him, and finally, if at large, will dash off and go for miles, attacking any animal or human being that may cross his path. During the course of his mad career he may succumb to exhaustion, or may return to his own home, provided he escapes destruction by human agency.

It is not usual for a rabid dog to go out of his way to attack a man or other animal. He usually trots on in a more or less straight line, and only attacks anything that actually crosses his path.

Martin states that the desire to attack in the dog is confined to his own species, and that he seldom attempts to bite a man unless interfered with. This may be so in individual cases, but rabid dogs will undoubtedly make an unprovoked attack on human beings in many instances, and I have myself seen a rabid hound bitch go out of her way to attack a buffalo.

The rabid dog attacks in silence, and having "bowled over" his imagined enemy, he continues on his way. The saliva is usually thick and viscid, and the buccal mucous membrane congested. There may be wounds about the latter and laceration of the tongue. The animal has no fear of water. On the contrary, there is usually marked thirst, which he will try to satisfy. Rabid dogs have been known to swim rivers. There may, however, be difficulty in swallowing, which symptom frequently deceives the owner into thinking that there is "a bone in the throat." Exaltation of the sexual instincts is a frequent symptom of rabies. If allowed to live, these furious symptoms are followed by debility, paralysis, and death.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xviii., 1895.

Dumb Rabies.—In this form there are no preliminary furious paroxysms. The premonitory symptoms of dulness and altered disposition are the same as in the " furious " form, but they are rapidly followed by paralysis.

Usually, the first muscles to become paralysed are the masseters, as a result of which the lower jaw drops and there is dribbling of saliva. There is neither the inclination nor ability to bite. The pupils are dilated, and the expression usually appealing and pathetic. Vomiting or retching has occasionally been noticed as a symptom, and a " squint " is not uncommonly developed. Paralysis involves the pharynx, rendering deglutition difficult or impossible. Paralysis of the hind-quarters usually follows that of the masseters, then complete prostration, and death within a few days. The dropped jaw and drivelling saliva are frequently the first symptoms noticed by the owner, and are generally pathognomonic. Owners are frequently deceived into thinking that this symptom is due to " a bone in the throat," and I have known cases where the owner has poked about in the animal's mouth with his finger in an endeavour to dislodge the hypothetical bone—naturally a very dangerous proceeding.

Cases have been reported of rabid dogs recovering temporarily and living for several months, but such cases are exceptionally rare. Probably an error in diagnosis is the explanation of such instances.

Tranquil or Lethargic Type.—This was first described by Bernat, and was also observed by Lafosse. The dog pays no attention to his owner, and gets into a lethargic condition; the countenance is mournful, and both food and drink are refused. Coma gradually sets in, and general paralysis, with death, results. During the course of the affection there is no inclination to bite. In some instances a low, mournful howl may be emitted at intervals.

Modified Types of the Disease.—Cases are met with in which the early symptoms are dulness, and weakness of the hind-quarters, the latter being ascribed by the owner as due to an injury. The animals may partake of a little food, and there may be no disposition to bite, but definite symptoms of rabies may appear without any warning. Cases of this kind were recorded by St. Cyr, in which, after a brief period of illness, symptoms of rabies developed. One case in which a dog showed dulness for three days, but still ate and drank, died in the space of three days, with all the evidences of rabies. In another instance a dog was brought for treatment suffering from paraphimosis. He was said to have been ill for three days, and bit the veterinary surgeon while the latter was carrying out the necessary manipulation. The animal then showed the first evidences of rabies, and died of the disease within two days.

A case is recorded in which a dog was brought for treatment, the only symptom presented being paralysis of the hind-limbs, and no history of the animal having attempted to bite. On manipulating him, however, he snapped viciously, and bit the owner, who was attempting to pacify him. The presence of rabies in the dog was proved by the post-mortem examination, the stomach being full of foreign bodies, and presenting the usual lesions of the disease; also, when the correct history of the case was obtained, it clearly pointed to the existence of rabies. In other instances, with precisely similar clinical histories, the diagnosis was confirmed by examination of the brain at the Pasteur Institute.

These types of the disease must be regarded as very dangerous to the owners of dogs, and also to practitioners, as paralysis of the hind-limbs is a very common symptom of various affections in canine patients, while the tendency to bite may be developed suddenly.

No doubt the correct history of the case is not always given by the owner, as he is apt to overlook certain symptoms, which, if observed, would put us on our guard. For example, in one of the cases mentioned above it was found, on examining the room in which the dog had lain the previous night, that the animal had eaten away the woodwork to a considerable extent.

Course and Duration.—After the onset of clinical symptoms the course of the disease is very short, and death almost invariably takes place within seven days.

DIAGNOSIS.

It must be remembered that one rarely has the opportunity of observing all the above-mentioned symptoms. When advice is sought, it may be after the animal has been destroyed, on account of its having attacked other dogs or human beings. On the other hand, it may only be showing premonitory and very indefinite symptoms, yet the owner will probably wish the animal destroyed immediately if there is the slightest suspicion of the case being one of rabies. The recognition of rabies in its earliest stages is of the utmost importance, owing to the danger of spreading the disease by allowing a case to go unrecognised; yet it must be admitted that there are no means of making a positive diagnosis ante-mortem.

It is always advisable to err on the side of caution, and to remember that any sudden alteration in the normal disposition of a dog in a country where rabies exists should be viewed with the strongest suspicion, and if, added to this, there is the history of the animal having made an unprovoked attack on a human being, one should act in every detail as if there were no doubt as to the case being one of rabies.

When the disease is more advanced and the paroxysmal stage has set in, diagnosis is less difficult.

DIFFERENTIAL DIAGNOSIS.

There are certain conditions which might be mistaken for rabies.

Vicious Temperament.—An unprovoked attack by a dog known to have an habitually vicious temperament could not be looked upon as in any way a symptom of disease. In such a case one would have to rely on other symptoms.

Infection by Bacillus Pyocyaneus.—Harvey, Markham-Carter, and Acton, draw attention to a disease in dogs, due to natural infection by the *B. pyocyaneus.* The symptoms presented resemble to a certain extent some of those seen in rabies—viz., weakness and paralysis of the hind-quarters, cachexia, convulsions, and death. Microscopical examination does not reveal the presence of Negri bodies. It is suggested that cases of reputed cures of rabies, abortive rabies, etc., may in reality be instances of pyocyaneus infection. The question requires further investigation (see *Veterinary Record,* July 22, 1911).

A bone or foreign body fixed between the molars might give rise to symptoms simulating rabies—viz., difficulty in swallowing, drivelling of saliva, irritability, etc. There would usually, however, be other symptoms to assist in diagnosis—*e.g.*, pawing at the face to try and dislodge the object, coughing, shaking the head, and possibly attempts at vomiting, etc.

Dislocated Jaw.—In this rare affection the jaws would be rigidly fixed open, whereas in rabies the lower jaw merely drops from paralysis of the muscles, and can be easily closed with assistance.

Paralysis of Lower Jaw.—Cases of this nature have been recorded, depending on injury or on nervous lesions, and, although rare, it is necessary to remember that they may be mistaken for dumb rabies.

The history of the case must be taken into consideration ; also, there is an absence of the peculiar expression of the eyes which occurs in cases of dumb rabies.

In an outbreak of a particularly fatal form of pharyngitis in dogs, described by Williams as occurring in Edinburgh during the winter of 1878-79, one of the first symptoms observed was " dropping of the lower jaw," while discharge of a viscid, ropy saliva from the mouth, swollen glands, diarrhœa, and finally paralysis of the posterior extremities, were also present. Robertson* described similar cases in dogs, and erroneously termed the affection "diphtheria." Diphtheria in the cat may be accompanied by "dropping of the lower jaw." This symptom is occasionally observed in cases of canine distemper see (p. 684).

Such cases must be differentiated from dumb rabies, although it must be admitted that, should they be met with in a district where rabies was prevalent, considerable difficulty would be experienced in the diagnosis during the early stages.

There are other conditions, such as epilepsy, meningitis, pentastoma, cysticerci in the brain, entozoa, etc., which might give rise to symptoms somewhat resembling the nervous disorder in rabies, but are distinguished by the absence of the characteristic train of symptoms seen in rabies.

It must be remembered that dogs of a naturally irritable disposition may, when suffering from acutely painful affections, such as canker affecting the middle or internal ear, acute rheumatism, pachymeningitis of the spinal cord, etc., become vicious, and may bite even their owners. When rabies was prevalent, many errors were made in the diagnosis of such cases, and we may add that considerable doubt existed as to the presence or absence of the disease in the dog when human beings were bitten. A very serious responsibility was placed on the veterinary surgeon in these instances, as he was called on by the medical attendant of the bitten person to give a definite opinion as to the existence of rabies or otherwise in the dog that inflicted the bite. Naturally, the proper course to adopt is to secure the dog in a safe place until either definite symptoms of the disease develop or death occurs, and then to have the carcass examined by a competent bacteriologist. In some of the nervous forms of distemper, symptoms closely allied to those of rabies may be manifested (see Distemper, p. 686). For further details of the differential diagnosis see Morbid Anatomy, p. 611.

TREATMENT.

Treatment is never under any circumstances justified. The disease is invariably fatal after an animal has shown clinical symptoms.

There is no reason, however, to suppose that a number of animals which are bitten by a rabid animal and actually infected do not overcome the infection in its early stages before clinical symptoms are manifested.

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* *Veterinary Journal*, August, 1875.

MORBID ANATOMY.

Lesions in the Dog.—(*a*) *Macroscopic.*—The following are the abnormalities most constantly present in dogs that have actually died from rabies: Some degree of congestion of the gastric mucous membrane, complete absence of food materials in the stomach, and in many cases the presence of such foreign substances as straw, hay, pieces of stick, stones, rags, etc.

Sometimes the stomach is completely empty, excepting for a little brown-tinged mucus. There may be congestion of the fauces and larynx, but it is not so constant or, when present, so pronounced as to be of any diagnostic value. There are also frequently wounds about the tongue and buccal mucous membrane, and injuries either self-inflicted, or caused by the animal biting and worrying foreign substances during paroxysms of rage.

The condition of the stomach, when taken in connection with the clinical history, is of considerable diagnostic value.

To sum up: The absence of ordinary food and the presence of considerable quantities of foreign materials in the stomach of a dog that has made an unprovoked attack on a human being leave hardly any room for doubt that the animal was actually rabid. On the other hand, the presence of a considerable quantity of ordinary food is rather strong evidence against a diagnosis of rabies (McFadyean).

(*b*) *Microscopic.*—Some observers have described certain minute lesions of the nervous system as characteristic of rabies. These lesions are said to occur principally in the medulla oblongata and spinal cord. They are said to take the form of minute hæmorrhages, local accumulations of leucocytes, and degeneration of nerve cells. In 1886 Babes concluded, as the result of numerous researches in man and dogs, that the essential lesion of rabies consisted in an accumulation of embryonic cells in the neighbourhood of the central canal, and especially about the large modified cells of the motor centres of the bulb and cord.

In 1892 he reaffirmed his observations, and held that it was possible to make a rapid diagnosis of the disease by a microscopic examination of the bulb and cord. He described in the bulb what he considered the diagnostic lesion of the disease—viz., pericellular accumulations of embryonal cells. The cells of the bulbar nuclei undergo degeneration, and present various stages of chromatolysis. There is a loss of the prolongations and a progressive modification, and even total disappearance, of the nuclei, a dilatation of the pericellular space, and an invasion, not only of this space, but also of the nerve cells. Many of the nerve cells become surrounded by a large zone of embryonal cells, and when the cell is completely degenerated, these occupy the cell area, and constitute the "rabic tubercle."

Nélis and Van Gehuchten first noted what they considered to be a more diagnostic lesion of rabies than any other. They asserted that the most profound, constant, and earliest lesions are noted in the peripheral, cerebral, and sympathetic ganglia, and the changes are especially marked in the intervertebral ganglia and in the plexiform ganglia of the pneumogastric nerve. The characteristic changes consist in the atrophy, invasion, and destruction of the nerve cells brought about by new-formed cells derived from the capsule, which appear between the cell body and its endothelial capsule. These new-formed cells invade the nerve cell, and finally occupy its entire capsule.

Cuillé and Vallée found that in animals in which the disease had just become manifest, the lesions in the plexiform ganglia were slight or absent. They concluded that as a means of diagnosis the method of Nélis and Van Gehuchten has great value in the case of animals in which the disease has run its full course, ending in death. Rabieaux also confirmed these observations.

The experience of all authors who have investigated the subject shows that the practical value of this method of diagnosis varies according to the nature of the case. If the suspected animal has been killed at an early stage of the disease, the diagnostic value of the above is very restricted, for the lesions are then at the most very slight, and most frequently entirely absent. Consequently, the non-detection of the lesions never justifies one in pronouncing the case not to be rabies. On the other hand, in the case of animals that have died, the before-described lesions are constantly present, and such lesions have not hitherto been observed with certainty in animals dead from other diseases than rabies.

In March, 1903, Negri announced that in many nerve cells obtained from rabid animals it was possible to detect corpuscles having special staining properties. These, Negri described as the parasite of rabies. They appear as oval, rounded, or kidney-shaped bodies, varying from 5 to 27 or 30 μ in diameter, lodged in the protoplasm close to the nucleus. These corpuscles are strongly refractile, and appear to contain granules in their interior. The number in different cells varies, and from ten to fifteen may be present in one cell. These corpuscles can be found in all parts of the nervous system, but are most numerous and constant in the large pyramidal cells of the hippocampus.

Negri held that the presence of these corpuscles afforded a rapid method of diagnosis. He admitted, however, that in rare cases they could not be demonstrated, although the animal might be proved to be rabid by other means.

The above observations have been confirmed by other authors, and all are agreed that the corpuscles can only be found in rabid animals. The significance of these bodies is still in doubt. Evidence is against their being the parasite of rabies. In support of this conclusion the following reasons are given by Anna W. Williams and May McLowden :*

1. They have not been found in all cases of hydrophobia, notably not in the " fixed virus," nor in all parts of the nervous tissue proved to be virulent.

2. Forms small enough to pass through the coarser Berkefeld filters have not been seen.

3. The structure has not been definitely shown to be analogous to that of any known living organism.

4. No definite series of forms indicating growth and multiplication have been demonstrated.

Moore,† however, comes to diametrically opposite conclusions, and states as follows with regard to these " bodies " : " The Negri bodies are organisms belonging to the class Protozoa."

The reasons he gives for this conclusion are—

1. They have a definite characteristic morphology.

2. This morphology is constantly cyclic—*i.e.*, certain forms always predominate in certain stages of the disease, and a definite series of forms indicating growth and multiplication can be demonstrated.

3. The structure and staining qualities, as shown especially by the smear method of examination, resemble that of certain known protozoa, notably of those belonging to the suborder Microsporidia.

The question as to their significance, therefore, must be considered as *sub judice*, although the majority of observers probably do not favour the view of their parasitic nature.

From the foregoing remarks on the nervous lesions it will be gathered that, although these lesions form valuable aids to diagnosis, negative results of microscopical examinations do not justify a verdict of " not rabies."

The only absolutely certain method of proving the existence or otherwise of rabies is by experimental inoculation.

Experimental Inoculation.—The material inoculated is a piece of the spinal cord or medulla—preferably the latter—from the suspected animal, and the rabbit is the animal selected for the inoculation.

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* *Journal of Infectious Diseases*, vol. iii., 1906.

† " Pathology of Infectious Diseases of Animals."

A small quantity of an emulsion in bouillon or water of the brain of the suspected animal is inoculated subdurally, after trephining, into not less than three rabbits.

Provided that the inoculated rabbits survive the usual period of incubation (fourteen or fifteen days), and that the dog from which the brain material used for the inoculation was taken was the subject of the disease, the inoculated rabbits will infallibly develop rabies.

The period of incubation in the rabbit inoculated with material from a natural case of rabies in the dog is usually about fourteen or fifteen days, and the period of visible illness one or two days. The symptoms are those of gradual paralysis, commencing usually in the hind-quarters and rapidly extending forwards. As a rule, there is no cerebral disturbance and no propensity to bite.

The method of diagnosis is very certain when the brain used for inoculation is fresh. If partially putrid, however, the experiment may fail for several reasons, the chief one being that the associated putrefactive bacteria are almost certain to kill the rabbit from a local inflammation of the membranes of the brain within a few days, and for the success of the experiment it is necessary that the rabbit should survive at least a fortnight, and not be the subject of any other cerebral disturbance.

Method of Removal and Setting up for Despatch of the Brain of an Animal suspected of Rabies.—It will be seen from what has already been stated that there are two methods of diagnosing rabies post-mortem—viz., (1) *microscopical* and (2) *experimental*. Brain material is necessary for both these methods of examination. When destroying a suspected dog, therefore, the cranium should be mutilated as little as possible.

The Director of the Pasteur Institute at Kasauli, India, in a pamphlet entitled "Rabies and Antirabic Treatment in India" (August, 1908), gives the following instructions for the removal and setting up for despatch of the brain:

"The first procedure is to open the skull and expose the brain. First wash the head well with an antiseptic such as carbolic acid, phenyl, etc. Then take a hammer, and with a few hard, sharp blows fracture into many pieces, through the intact skin, the top and sides of the brain cavity. With a knife throw back the skin, and then remove as carefully as possible the pieces of fractured bone, in this way exposing the brain.

"For the experimental method, with a clean knife remove a small piece of brain, about the size of a bean, and place it in a small bottle of pure glycerine. On no account is an antiseptic to be allowed to come in contact with the piece of brain, nor is anything to be added to the glycerine, "For the microscopical method a special portion of the brain is desirable—viz., the hippocampus major. As this is somewhat difficult to dissect out by anyone unaccustomed to such work, it is best to remove the brain entire. It should at once be put into a wide-mouthed bottle containing a plentiful supply of the following solution:

Bichromate of potash	90 grains.
Glacial acetic acid	2¾ fluid drachms.
Water	6½ fluid ounces.

"In removing the brain of a rabid animal, the greatest care must be taken not to allow the saliva or brain substance, both of which, as we have seen, contain the rabies virus, to come in contact with any cuts or abrasions on the hands."

II. RABIES IN THE HORSE.

Symptoms.—The premonitory symptoms in all the domesticated animals and also in man are more or less analogous, and referable to morbid changes in the central nervous system.

Amongst the first symptoms noticed in all animals, including the horse, are restlessness, nervous excitability, dilated pupils, hyperæsthesia. The horse becomes hypersensitive to external surroundings, starting at the least sound, and looking upon everything with suspicion. Later there is irascibility, and the animal will kick or bite at any living being within reach. There are mental hallucinations, during which the patient exhibits furious paroxysms. During these paroxysms, which increase in intensity as the disease progresses, the patient may kick at and break down any partitions and other objects not strong enough to withstand his violence. He may also seize the manger or other object with his teeth, and attempt to tear it down.

Yawning, champing the jaws, and salivation, are also frequent symptoms. Sexual excitement is a common symptom in both stallion and mare, the latter showing symptoms of being "in season." The respirations and pulse are accelerated. A stick held in front of the animal may excite a paroxysm, and he will seize it between the teeth.

A rabid horse will frequently lacerate himself deeply, especially at the seat of infection, in which excessive pruritus seems to occur. There are periods of calm between the paroxysms, during which the faculties are partially regained; but these periods become shorter and shorter as the disease progresses. The rabid horse usually exhibits ardent thirst, and has no dread of water, although there may be difficulty in swallowing.

The disease terminates with exhaustion, paralysis of the hind-quarters, complete prostration, and death within a few days.

According to Youatt (quoted by Percivall), the disease always develops more quickly when the seat of inoculation—*i.e.*, the bite—is in the region of the head; this was confirmed by Blaine. The same author also observed that the rabid horse showed a marked antipathy to dogs.

Martin* makes the following observation with regard to rabies in the horse and mule: "The desire to bite themselves is more marked in horses and mules than in dogs. I saw a very good example of this in a Government mule at Rawal Pindi. . . . When I saw him he had been moved from his lines, where he had shown a disposition to attack his fellows on either side, and was standing quietly in a large loose-box. The temperature was 104° F., pulse correspondingly accelerated; but the most striking symptom was a grinding of the teeth. After a cautious examination I found he was chewing his tongue. . . . On post-mortem examination I found that the right side of the tongue in the vicinity of the molars was completely gone. In another case that occurred in the same place a horse tore strips of skin and flesh from his breast. . . ."

Course of the Disease.—Five or six days.

Differential Diagnosis.—In no other nervous disorders of the horse are the same symptoms of malicious intent shown.

The disease is readily distinguished by the paroxysms of fury in which the animal will kick and bite at anything within reach. Cases have been noted in which paralytic symptoms only were observed. When rabies is prevalent in a district, the fact of the horse having been recently bitten by a dog will assist in the diagnosis.

Morbid Anatomy.—In the horse the macroscopic lesions are neither definite nor characteristic. Youatt observed in every instance inflammation of the glottis, and generally also of the trachea, also evidences of gastritis and inflammation of the lungs, the latter occurring in patches. The meninges covering the medulla oblongata, and also the substance of this organ always showed marked injection.

Zahn, of the Vienna Veterinary School (quoted by Fleming), found the spinal cord greatly softened, acute inflammation of the spleen, and a non-coagulated, thick (*sic*) condition of the blood.

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* *Journal of Comparative Pathology and Therapeutics*, 1895.

III. RABIES IN CATTLE.

Symptoms.—These are similar to those met with in the horse. There is restlessness, manifested by pawing and stamping on the ground, prominent and staring eyes, with dilated pupils, depraved appetite, nervous excitability (especially if after dark a light of any kind is brought near the animal), bellowing, salivation, muscular tremblings, exaltation of the sexual appetite. There may be some degree of pharyngeal paralysis, with consequent difficulty in deglutition. Hallucinations are constant. The animal may become very excited and try to break away. In the cow there is suspension of the milk.

Violent paroxysms ensue, during which the animal uses its natural weapons of offence—viz., the horns—for attack. Severe injuries and broken horns are a common result of these paroxysms, from the animal butting at hard obstacles. There is usually constipation at first, followed by diarrhœa. Tenesmus may be so great as to cause eversion of the rectum. The infection wound, if observable, may be irritable and itchy, causing the animal to continually lick it.

Emaciation, exhaustion, paraplegia, and general paralysis, are the final stages. It is also recorded that purely paralytic rabies in cattle is not uncommon.

Course of the Disease.—Four to six days.

Differential Diagnosis—*Meningitis.*—Some cases of meningitis in cattle are characterised by uncontrollable excitement, the animal rushing wildly against walls and doors, and running about in a mad manner. The origin of many of these cases is obscure, but should rabies be prevalent in a district, a differential diagnosis will present considerable difficulties. Some vegetable poisons, such as rhododendron, may cause similar symptoms.

IV. RABIES IN SHEEP AND GOATS.

Symptoms.—The general course of symptoms is practically the same as in cattle—viz., loss of appetite, cessation of rumination, pruritus at the seat of infection, hyperæsthesia, genital excitement, staring eyes and dilated pupils, stamping the feet, grinding the teeth, aggressiveness. In their normal condition sheep are usually in terror of dogs, but rabid sheep will actually attack these animals.

These symptoms are rapidly followed by emaciation, exhaustion, and paralysis. Goats have a greater propensity to bite than sheep.

Fleming states that rabid sheep have been known to drink blood.

A striking characteristic of rabies in the sheep is the genital excitement. The animals "mount" each other by turns, and this may continue for a day or two, and they may feed at intervals. The rams and wethers may sometimes show more or less ferocity, in which the ewes occasionally participate, but there is no tendency to bite. The animals nibble at a stick when it is presented to them; they "butt" with their heads when anyone approaches them, and also press their heads against the walls, fences, etc., with considerable force, so that the skin becomes abraded at the region of the forehead. Youatt observed that in rabid sheep the bleating is higher in tone than normal, and more plaintive. Also, the head is elevated and turned to one side, and one ear and one eyelid may be drooped. Also, they may swallow foreign bodies, nibble wood, and show a discharge of ropy saliva from the mouth. He described a dumb or subacute form of the disease, and observed that lambs showed a succession of convulsive fits, and died in a day or so.

Course of the Disease.—This varies from five to eight days.

In adult sheep death may take place in some instances on the second or third day, in others on the fifth day, or later.

The disease generally appears between the close of the second to the fourth week after being bitten by a rabid dog, but it may be delayed to the eleventh week.

Morbid Anatomy of Rabies in Ruminants.—The lesions are neither constant nor definite. In cattle, meningitis and congestion of the mucous membrane of the abomasum and of the intestines have been observed, and a dark red, imperfectly coagulated condition of the blood (Steel). In the sheep Youatt observed inflammations of the tongue, pharynx, trachea, and abomasum, in some cases, while in others evidences of meningitis only were found. The same author describes instances in which the rumen contained a large number of foreign bodies, and the lining membrane of this viscus was inflamed in patches and showed small spots of extravasation of blood. In some cases Youatt did not discover lesions of any kind.

V. RABIES IN THE PIG.

Symptoms.—Irritability at the seat of the wound, restlessness, fixed stare, with dilated pupils, excitability, hyperæsthesia, hurried respirations, salivation, grinding of the teeth, difficulty in deglutition. Rabid pigs will attempt to hide themselves under straw or litter. Then follows aggressiveness; the animal will gnaw at any inanimate object. The sow may suckle her young during remissions. At other times she may devour them. Sometimes partial paralysis of the lower jaw is seen.

The appetite is usually depraved. The disease terminates in the usual way, in paralysis and death.

Course of the Disease.—Two to six days.

VI. RABIES IN THE CAT.

Symptoms.—These are more or less the same as in the dog. The disease is not often seen in the cat. Whether this is due to a greater resistance to infection, or to their habit of life being unfavourable to infection, it is difficult to say.

It is easy to appreciate the great danger from a rabid cat. This species being less domesticated than the dog, there is not the same loving instinct towards their master to act as a control over their viciousness. Fleming gives the following picture of a rabid cat:

"Its great eyes sparkle with an unnatural light, and express a startling degree of ferocity. When in a cage, there is nothing more terrible. The mouth is partly open and foamy, the back arched, and the tail beating the flanks. The claws are so rigidly protruded as to cause it to walk with difficulty, and they even penetrate the floor, leaving their imprint there. When anyone presents himself before it, it flies towards him at a bound as high as the cage will permit, as if to attack the face, for this always appears to be the region of the body for which the rabid creature has a special predilection when at liberty."

The disease terminates with paralysis in the usual way.

Course of the Disease.—Two to six days.

VII. RABIES IN THE CAMEL AND DEER.

Symptoms.—These animals show both furious and paralytic symptoms. Camels are particularly dangerous, as they are naturally inclined to be vicious, and use their teeth as weapons of offence, inflicting a very severe contused wound.

Deer, usually so inoffensive, become aggressive when rabid.*

VIII. RABIES IN POULTRY.

Chickens may contract rabies naturally. Symptoms of restlessness and nervous excitability are shown. They also become aggressive, and will advance to attack man or dog, and fight with other birds. They also perform peculiar movements, evidently suffering from hallucinations. Paralysis always supervenes, followed by death.

* Outbreaks of rabies in deer have been recorded in England in 1872, 1880, 1886, and 1887.—Editor.

IX. RABIES IN WILD CARNIVORA.

The symptoms are the same as in the dog, the different species using their natural weapons of offence.

X. RABIES IN THE FOX.

This animal shows similar symptoms to those occurring in the dog. A case is on record of rabies in a tame fox. The owner, a nobleman, was bitten by the animal, and, although subjected to treatment by the Pasteur method, he succumbed to the disease. The nobleman's valet was also bitten by the same animal, and afterwards treated by the Pasteur method, but he escaped the disease.

In 1911 a case of rabies in man is recorded said to be due to a bite received from a fox in the hunting-field.

XI. RABIES IN MAN.

Synonym.—Hydrophobia (Greek *ὕδωρ*, water; *φόβος*, fear).

Symptoms.—The general course of the symptoms in man is more or less the same as in animals, and is marked by nervous derangement.

There is irritability at the seat of wound, anxiety, restlessness, insomnia, gloomy forebodings, etc. There is a haggard expression about the eyes and a shifty look of apprehension. There is thirst, but attempts to swallow excite spasms of the throat. Later, these spasms become intensified if an attempt is made to drink. As a result of this, the patient refuses to try, for the reason that the mere sight or noise of water or anything suggesting it excites violent paroxysms. It is this symptom which is peculiar to man, and to which the term "hydrophobia" owes its origin. The term is therefore incorrectly used as a synonym for the disease in any of the domesticated or wild animals, and apt to be misleading.

During paroxysms dyspnœa is extreme, and choking sounds are made in the effort to dislodge the tenacious mucus from the throat. It is these sounds which have been compared to the bark of a dog. There is a sense of tightness in the throat, and there may be retching. Reflex excitability is marked, as in animals, and the slightest disturbance may bring on paroxysms. Hallucinations and mania follow, necessitating a considerable amount of restraint. There are periods of calm and lucid intervals. Sexual excitement is seen in man, as in animals. Exhaustion and paralysis terminate the disease.

PROPHYLACTIC MEASURES.

At the second International Veterinary Congress, held in 1863, the committee resolved that the introduction and strict execution of a rational supervision of dogs was the surest means of preventing and ultimately extirpating rabies.

Here we have the pith of the whole matter as far as prophylaxis is concerned.

There are two methods by which the disease may be tackled—viz,. the radical method of stamping it out and the palliative method of keeping it under control, without resorting to the more drastic measures.

Of these methods it need hardly be said that the former is to be preferred where practicable. That the disease can be stamped out, even after it has become well established, is proved in the case of this country; and in other countries—*e.g.*, Australia—where it has never established itself, it has been kept out by rigid inspection, quarantine, etc.

The last case of the disease occurred in the British Isles in 1902.

The Order of the Board of Agriculture, to which the ultimate stamping out of the disease may be said to be due, is that dated March 23, 1897, and known as the Rabies Order of 1897.

For full details I would refer the reader to the Order in question, but will here simply mention its salient points:

1. Compulsory notification of a case of rabies or suspected rabies.
2. Compulsory slaughter of dogs diseased or suspected, or of dogs bitten by a diseased or suspected dog.
3. Post-mortem verification of a case of rabies.
4. Compulsory isolation of dogs which have been exposed to infection —*i.e.*, have been in contact with a rabid dog, whether known to have been bitten or not.
5. Seizure, detention, and disposal of stray dogs.
6. Disposal of the carcass of a dog affected with or suspected of rabies.
7. Regulations as to the disinfection of buildings, etc.
8. Punishment for infringement of the Order.

The reintroduction of the disease has been prevented by accessory Orders dealing with the importation of dogs—viz., the Importation of Dogs Order of 1901 and the Importation of Canine Animals Order of 1909. These Orders deal with the inspection and quarantine of imported dogs.*

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* As foreign foxes are introduced into Great Britain, it is certainly desirable that in order to guard against any possible source of infection, similar rules as regards inspection and quarantine should be enforced in the case of these animals.—EDITOR.

General Measures for controlling the Disease.—We will now discuss the general principles governing the control of the disease, for the proper carrying out of which State intervention is necessary.

For the suppression of any disease strict supervision over all animals of the species susceptible to the disease is a *sine quâ non.* Supervision can only be carried out by fixing the responsibility of ownership.

In the case of dogs this is best effected by a regulation insisting that every dog shall wear a collar with the name and address of the owner engraved thereon.

Any dog found wandering about without a collar should be seized by the police, and, if not claimed within a certain period, destroyed. If claimed, the owner should be made to pay a fine for infringement of the Order.

An additional method of fixing the responsibility of ownership is to put a tax on all dogs, and insist on their registration. This measure not only fixes responsibility, but also has the desirable and necessary effect of reducing the number of dogs.

In a country where rabies exists every effort should be made to put a stop to the uncontrolled wanderings of ownerless dogs, and to suppress this source of danger, at the same time diminishing the number of useless dogs.

Muzzling of all dogs is another important measure in the suppression of the disease. A muzzling Order should be put in force in every area in which the disease is known to exist. According to the extent of the disease, the Order should be enforced either universally or locally.

When it is realized that the disease is only conveyed from animal to animal by inoculation—*i.e.*, by a bite—the vast importance of the measure will be seen. Its efficacy has been proved, not only in this country, but also abroad—viz., in Berlin, in 1853. In London the Muzzling Order was enforced in 1889, with the result that the number of cases was reduced from 123 in that year to 3 cases in 1892. After suspension of the Order, the number of cases rapidly increased.

In spite of these convincing facts, there was always a certain amount of opposition to the Orders, on the ground of cruelty, although it is difficult to see where the cruelty comes in. The desire to oppose such a necessary suppressive measure could only result either from ignorance as to the reasons for it, or from the want of some more useful and philanthropic occupation.

For the suppression of any disease, and in particular rabies, education of the lay public in its recognition and their co-operation is always essential.

Fleming suggested that the best method of educating the public was to provide dog-owners, when they received their tax-paper, with printed, easily understood instructions as to the proper method of keeping their dogs healthy, and how to detect the early symptoms of rabies, as well as information respecting the preservative and sanitary police measures which they should comply with in order to prevent the disease. He also suggested that all this might be printed on the back of the tax-paper, which could, in addition, be made a valuable means of arriving at certain important information, such as the sex, age, breed, etc., of the licensed dogs.

The suggestion has its merits, and presents an easy method of disseminating the knowledge to dog-owners.

It now remains to be considered what alternative measures should be taken in the case of dogs diseased or suspected where there are no Government regulations dealing with the same.

Measures to be adopted in the Case of a Dog suspected of Rabies.—If an owner is aware of the fact that his dog is showing symptoms in the slightest degree suspicious of rabies, then there are only two courses open to him : (1) Either the dog should be immediately destroyed, or (2) immediately segregated in the same manner as if there were no doubt as to its being rabid. Segregation in this case involves the securing of the animal in such a way as to render any possible contact with man or beast impossible. To merely chain up the dog does not meet the case. It should not only be strongly secured by a chain and collar to some suitable object, but should also be kept in a cage or within four walls. Another point to be remembered in this connection is that an owner is not justified in getting any attendant to feed and look after such an animal without fully explaining the nature of the case and the risk incurred by contact with the patient. A period of ten days' segregation will usually be sufficient to determine the diagnosis, since the course of the disease in the dog after the onset of clinical symptoms is rarely more than five or six days. Hence, if the symptoms which led the owner to regard the dog as suspicious were actually those of rabies, the latter would be dead within the segregation period. If, on the other hand, the dog were apparently healthy at the end of ten days, the symptoms first manifested might confidently be declared as not those of rabies.

Of the above alternatives—viz., destruction or isolation—the former is to be recommended in the case of a dog which is not known to have bitten any other animal or human being, for the reason that all further risk to man or beast is avoided.

On the other hand, if evidence is forthcoming of some other animal or human being having been bitten, the suspected dog should not be destroyed, but should be segregated. The reasons for this are as follows :

1. In the event of other dogs having been bitten, it is important to have the case confirmed, in order that, should it prove to be one of rabies, prophylactic measures may be taken with regard to the dogs bitten. Reference to these latter measures will be made hereafter.

2. Should any human being have been bitten, a positive diagnosis is essential, in order that, should the case prove to be not one of rabies, the person bitten may be relieved of further anxiety, and if meanwhile the said person were undergoing antirabic treatment, the latter could be immediately suspended.

In the event of the suspected dog dying within the segregation period, either with or without the classical symptoms of rabies, provided that there be no evidence forthcoming of any other animal or human being having been bitten, the diagnosis may be allowed to rest on the clinical symptoms.

If, on the other hand, such evidence is forthcoming, material should always be forwarded at once to the nearest laboratory, or, in the case of a person bitten, to the institute to which he may have proceeded for treatment, with the history of the case, in order that the diagnosis may be verified by bacteriological methods.

Measures to be adopted in the Case of a Dog bitten by a Rabid Dog or Dog suspected of Rabies.—Immediate destruction would certainly be the safest and best measure to advise. Where there are no Government Regulations insisting on this procedure, it cannot be carried out without the consent of the owner.

The only alternative measure which can be considered safe is a period of six months' segregation. This segregation, to be effective, must be rigidly carried out. The animal must not be allowed to come in contact with man or beast, excepting its attendant, and must be exercised on a "lead" in an enclosed space, to avoid any possibility of contact.

Here, again, where there are no Regulations enforcing such segregation, it can only be left to the good sense and morality of the owner to carry it out. To render such segregation effective, proper establishments under veterinary supervision would be necessary for the purpose.

Measures to be adopted in the Case of a Dog which has been exposed to Infection.—This refers to dogs which have been in contact with diseased or suspected animals, but are not known to have yeen actually bitten. In these cases also there is no alternative but to advise a period of six months' segregation, unless the owner elects to take the more drastic step and have the animal destroyed.

Measures to be adopted in the Case of Animals other than Dogs (Horses, Cattle, Sheep, etc.) bitten by a Diseased or Suspected Animal.—In the case of such animals as these, which do not use their teeth as their natural weapons of offence, there is less likelihood of their spreading the disease before it is recognised. Moreover, their habit of life, in being kept more or less under restraint or in limited confinement, lessens the danger of propagating the disease. In their case, therefore, if they are kept under close observation for six months, it is sufficient. During this period of observation they should neither be sold nor removed from their own district.

Other precautionary measures consist in the proper disposal of the carcasses of animals dead of rabies, and adequate disinfection of buildings, etc.

Treatment of a Person bitten by a Dog suspected of Rabies.—In giving advice concerning the state of health of a dog which has bitten a human being, it is better to err on the side of caution. If there is reasonable suspicion that the dog in question may be the subject of rabies, the safest course is to advise the person bitten to undergo antirabic treatment.

The wounds inflicted by any dog, whether suspected of rabies or not, should be immediately cauterised with pure carbolic acid or some other reliable caustic.

In the case of a person bitten by a dog apparently in the best of health, and concerning which suspicions were never aroused, no further treatment is necessary, provided the dog in question remains in good health for the succeeding ten days.

If, on the other hand, the demeanour of the dog is suspicious, and the bitten person proceeds to the nearest Pasteur Institute for treatment, the dog should be securely fastened up and segregated. If it should develop unmistakable signs of rabies within the succeeding ten days, it should be destroyed, and material forwarded for verification to the institute to which the person bitten has proceeded. If, on the contrary, the dog should be in the best of health ten days after having inflicted the bite, the fact should be communicated by wire to the institute, so that treatment can be suspended. It follows, also, that in the case of a dog which has been beaten to death after biting a human being or other animal, brain material should be sent for laboratory diagnosis whenever possible. It is important to remember that the success of antirabic treatment depends on the time which elapses between the moment of infliction of the bite and the commencement of treatment, and for this reason there should be no delay.

Having considered the prophylactic measures which should be taken in controlling and suppressing the disease, it must readily be seen how utterly impossible such control must be where measures are merely permissive and left to the good sense of dog-owners in general. For the control of the disease Government intervention is absolutely essential. The measures to be adopted must depend on the nature and physical conditions of the country. We have seen that in a country like England it has been possible to stamp out the disease by enforcing drastic measures. As an example of a country in which different conditions prevail, we have India. In this latter country the disease is rampant, and no effort is made to control it. The country is a vast one, in which there are large tracts of jungle teeming with wild beasts of all sorts. These latter may act as reservoirs of the disease and add to the difficulty of controlling it. An attempt to make any regulations universal in dealing with the disease would be futile, owing to the impossibility of seeing them carried out. Further, the stamping-out method adopted in the British Isles would be quite impracticable in a country like India. On the other hand, a great deal might be done to control the disease by Government Regulations applied locally—*i.e.*, to towns, cantonments, etc., and their immediate vicinity.

Such Regulations or Orders should deal with—

1. Notification of the disease.

2. Slaughter of affected animals.

3. Rigid isolation of suspected animals or animals bitten by a diseased or suspected animal on the lines suggested in the prophylactic measures detailed above.

4. Reduction of the number of stray and ownerless dogs, and fixing responsibility of ownership by registration and taxation.

This policy should be relentlessly pursued.

5. Punishment for infringement of the Orders.

By some such measures as the above a great deal might be done in controlling the disease. Apart altogether from rabies, a reduction in the number of starving, ownerless, wandering, vermin-infested pariah dogs would in itself be a boon to humanity.

PROTECTIVE INOCULATION.

Pasteur introduced what is really a method of preventive inoculation, but it is seldom employed except in the case of persons that have already been bitten, and may therefore be presumed to be already infected. Nevertheless, the operation is protective rather than therapeutic. The method may be employed to confer protection on the absolutely healthy animal.

The material used as the vaccine is the spinal cord of a rabbit that has been inoculated with what is called the "*fixed* virus" of rabies. The virus is so called because of the constancy of the period of incubation, but it is also an intensified or exalted virus. It is obtained by the method of passage through a number of generations of rabbits. In contradistinction to this fixed virus, there is what is called the "*ordinary*" virus, which is of the strength usually present in natural cases of rabies in the dog.

The average period of incubation in rabbits subdurally inoculated with the ordinary virus is about fourteen or fifteen days. The "fixed virus," on the other hand, has an almost constant period of incubation of six or seven days.

Preparation of the Material for Vaccination.—The cord from a rabbit dead of the "fixed virus" is removed aseptically, and partially desiccated by being suspended in a jar over fragments of caustic potash at a certain temperature. A cord thus desiccated for fourteen days apparently no longer contains living virus, and cords desiccated for shorter periods may be said to be graded in their virulence.

Vaccine material is prepared from cords of different degrees of virulence—*i.e.*, from cords exposed for varying periods to desiccation. The treatment is varied slightly according to the severity and probable danger of the bites sustained. In cases of ordinary bites by rabid animals the plan is to inject on the first day of treatment vaccine prepared from two different cords—viz., one desiccated for fourteen days, and one for thirteen days. On the second day vaccine prepared from cords of twelve and eleven days' desiccation is used, on the third day cords of ten and nine days' desiccation, and so on, until finally vaccine prepared from a cord of only three days' desiccation is inoculated, the whole treatment extending over a period of fifteen days.

In the case of bites that are supposed to be especially dangerous—*i.e.*, when the wounds are extensive and deep, or inflicted about the face—a modified and more intensive form of treatment is practised. Two inoculations a day are given, in order to work up more rapidly to the use of a cord of only three days' desiccation, which latter is given at the end of the first week. After this for the following fourteen days changes are rung on three, four, and five day cords. The subjoined table shows the two methods:

Days.	Ordinary Method.	Intensified Method.	
	Number of Days Desiccation of Cord.	Number of Days Desiccation of Cord.	
		Morning.	Evening.
First	14 13	14 13	12 11
Second	12 11	10 9	8 7
Third	10 9	6 —	6 —
Fourth	8 7	5 —	— —
Fifth	6	5	—
Sixth	5	4	—
Seventh	5	3	—
Eighth	4	Changes rung on three, four, and five day cords, finishing up with a two day cord.	
Ninth	3		
Tenth	5		
Eleventh	5		
Twelfth	4		
Thirteenth ..	4		
Fourteenth ..	3		
Fifteenth	3		

Until quite recent times it was assumed that the unmodified "fixed virus" present in the fresh cord of rabbits would, if used straight away without previous attenuation, prove fatal to human beings by subcutaneous inoculation. It was further presumed that the inoculated person acquired a gradually increasing immunity from day to day from the daily inoculations, so as to be able to withstand inoculation with vaccine prepared from a cord of only three days' desiccation at the end of the first or second week, according to the method of treatment.

The explanation that inoculation on one day could confer immunity against inoculation with a stronger vaccine on the following day was always unsatisfactory.

Pasteur supposed that part of the protective effect of early cords was due to the presence of products of the rabic virus in the cord substance.

It is now admitted that desiccation has no attenuating effect on the cords.

The method of vaccination is one of inoculation with minimal doses of the fixed virus.

Cords desiccated for fourteen days either contain no living virus or else living unmodified virus in very minute proportions.

The "fixed virus" is not an intensified virus for man, but only for rabbits by subdural inoculation. For man and other animals it is a highly attenuated virus, and is without danger to man, and in most cases for the dog, when given by subcutaneous inoculation without any desiccation whatever.

This has been proved by experiments on animals and by different methods of treating human beings involving the use of fresh rabbits' cords.

The method consists in taking the cord from rabbits infected with the fixed virus, and making dilutions of varying strengths from 1 in 100 to 1 in 10,000. Treatment is commenced with 1 in 10,000 emulsions, the strength of the emulsion being gradually increased up to a 1 in 200 emulsion.

Even the 1 in 10,000 emulsion is fatal to rabbits by subdural inoculation.

The results obtained by this method appear to be practically the same as those obtained under the original Pasteurian method, and are probably better in the case of persons severely bitten about the head.

Attempts to obtain an antirabic serum have not been successful.

CONCLUDING REMARKS.

In any country in which rabies is rampant it is well to adopt certain precautions in dealing with canine patients, amongst some of which are the following:

1. Suspect every dog brought for treatment until satisfied by the history of the case and by observation that it is not the subject of rabies.

2. Never allow a dog, apparently healthy or otherwise, to lick the bare skin.

It is well to remember that a lick on an abrasion is equally dangerous to a bite, and that the saliva of a dog may be infective for several days before clinical symptoms are manifested.

3. When manipulation is necessary, endeavour as far as possible to avoid getting the hands soiled with saliva, and always make a habit of immediately washing the hands in some reliable disinfectant after manipulating a canine patient.

4. Should the appearance of a dog or cross-examination of its attendant arouse suspicions of rabies, no useful purpose can be served by unnecessary manipulation.

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NOTE BY EDITOR.

Semple's Antirabic Vaccine.—This method of anti-rabic vaccination was introduced by Lieutenant-Colonel Sir D. Semple. An 8 per cent. dilution of rabies virus is made in normal saline, and 1 per cent. carbolic acid is added ; the resulting mixture is then kept at a temperature of 37° C. for twenty-four hours. By this means the virus is killed. The mixture, diluted with an equal bulk of saline solution, gives a vaccine containing 4 per cent. virus (dead) and 0·5 per cent. carbolic acid. This conferred a high degree of immunity in monkeys, dogs, and rabbits, and the serum obtained from these animals had a well-marked rabicidal action on living virulent virus. Its efficacy was apparently as great as that obtained by the use of living virus, and it retains its power for some time (*British Medical Journal*, September 2, 1911).

I. CONTAGIOUS PSEUDO-RABIES.

Synonym.—False rabies; Pseudowut.

This affection simulates rabies in some respects. Up to the present time it has only been recognised in Hungary. The animals affected are cattle, dogs, cats, and rats. The virus is always present in the nervous system, and almost always in the blood and vascular organs, also in the urine, but does not occur in the bile or saliva. The causal agent has not been discovered. According to Zwick and Zeller, the virus does not pass through a Berkefeld filter, but Cadéac and others state that it belongs to the class of ultra-visible viruses. It does not appear to be easily destroyed by disinfectants or by heat. The brain remains virulent for two, and exceptionally for three, months when kept in glycerine. Brain substance preserved in glycerine retained its virulence for eight months in an ice-chest, and blood-serum under similar conditions for eight or nine weeks proved virulent. Feeding and inoculation experiments carried out by Zwick and Zeller on dogs showed that by the former method the results were negative, and by the latter large quantities of the virus were necessary in order to produce the disease. Cats and rats were successfully infected by feeding experiments. Sheep, goats, cats, rabbits, and rats were infected by inoculation, but only a comparatively small proportion of guinea-pigs were found to be susceptible. Fowls and pigeons resisted inoculation.

The disease does not appear to possess well-marked contagious characters, as on several occasions infected animals were caged with sound ones, but in no instance did the latter become infected.

The period of incubation, as judged by inoculation experiments, varies from thirty-six to ninety-six hours, according to the susceptibility of the animal, the virulence of the material, and the seat of inoculation; in exceptional cases it extended from five to eight days.

Some observers state that the primary mode of entrance for the virus in natural infection is the presence of wounds on the buccal or pharyngeal mucous membrane. Rats and mice are suggested as carriers of the infection.

In cattle the symptoms are as follows: A persistent irritation of the muzzle; the animal constantly rubs the part, so that bleeding patches, denuded of hair and surrounded by swollen and inflamed areas, result; more rarely other regions of the body are similarly affected. Bellowing and stamping of the feet are observed, also tympanites, and death occurs within twenty-four to thirty-six hours.

In the dog and cat, dulness, restlessness, salivation, loss of appetite, vomiting, and pharyngeal paralysis have been observed ; but fever was absent. Intense irritation of some part of the body occurred in about one-half of the cases studied ; the animals scratched these areas violently, producing in a few hours extensive abrasions of the skin, and even lesions of the deeper structures. In other cases evidences of irritation were absent, but the animals evinced pain by groaning. Amongst other symptoms noted were laboured breathing, unequal dilatation of the pupils, loss of the pupillary reflex, spasmodic muscular contractions, and in some cases weakness of the hind-limbs.

The disease is almost invariably fatal, death occurring within twenty-four to thirty-six hours.

When the affection is experimentally induced by inoculation, restlessness, marked excitement, and severe irritation set in after the period of incubation has passed. Inflammation, swelling, and irritation occur at the seat of subcutaneous inoculation, and the animals violently scratch, lick, or gnaw the part, or rub it against surrounding objects.

Death occurs in from three to thirty hours from the onset of symptoms, most commonly in from six to ten hours.

The lesions discovered post-mortem are not well marked. Hæmorrhage and necrosis are often found at the seat of inoculation.

The central nervous system and the cerebral meninges show hyperæmia and ecchymoses. The gastro-intestinal mucosa presents similar changes.

The differential diagnosis from rabies is carried out by paying attention to the following points : In pseudo-rabies there is an absence of furious delirium, also of paralysis of the lower jaw ; the saliva is not virulent, and the period of incubation is very short.

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II. INFECTIOUS BULBAR PARALYSIS.

Under this heading a peculiar disease of the nervous system is described by Continental authors, occurring as an enzoötic in cattle, dogs, and cats in Hungary. It is characterised by pharyngeal paralysis, nervous excitement, and the occurrence of intense irritation of various parts of the body, and appears to be very similar if not identical with the affection we have just described under the heading of Contagious Pseudo-Rabies. The disease was studied by Aujeszky in 1902, and up to that time was said to be unrecognised or to have been mistaken for rabies. According to Hutyra,* no case of the disease has been recognised outside Hungary, although references to it have appeared in French and German literature. This observer investigated an outbreak in which nine out of ninety cattle had died showing the characteristic features of the affection, and shortly afterwards cases occurred in an outlying farm amongst the three-year-old cattle, seven of these dying within a few days.

Szántó met with a case of the disease in a draught-ox, and Schaar recorded one in a milch cow; while Marek encountered numerous cases in a herd, and found that it was by no means uncommon amongst dogs and cats at the Clinique at Budapest, twenty-one cases being observed in these animals during 1908. Sczabó met with an enzoötic of the disease in dogs and cats, while Laufer recorded two cases, and stated that the affection was not uncommon among dogs. Kern observed cases in cattle and dogs in Croatia.

Etiology.—Nothing definite is known with reference to the causation of the disease. Balás observed an enzoötic among rats at a public abattoir, due to infectious bulbar paralysis, and at the same time and place dogs and cats showed symptoms of the disease. Hutyra records that during an outbreak in cattle the stable in which they were housed was frequented by many rats, and the latter decreased in number to a marked extent coincidently with the occurrence of the disease in the cattle.

Babik observed that the deaths of very large numbers of rats were coincident with cases of illness in cattle, dogs, and cats on a farm, and he formed the opinion that these animals mentioned were infected by rats. This view is strengthened by the fact that when experimental infection is carried out, the seat of inoculation becomes very irritable and painful, and this symptom is well marked in cases that become naturally infected. It is surmised that the natural method of infection is by the introduction of the virus either intradermically or subcutaneously, due to bites from infected rats. Moreover, the head, face, muzzle, and eyelids, are the parts that show evidences of intense irritation, and these are the regions most likely to suffer from rat-bites while the cattle are feeding or looking for food.

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* "Infectious Bulbar Paralysis: A New Disease of Animals," Hutyra, *Berlin. Tier. Wochenschr.*, February, 1910; translated in *Journal of Comparative Pathology and Therapeutics*, March, 1910.

It is believed that the disease is caused by a virus which is present in all the organs and in the blood of the affected animal, and it has been transmitted by inoculation with these materials to other animals. Hutyra carried out inoculation experiments in rabbits with portions of a brain taken from an ox that succumbed to the disease. On the fifth day one of the animals died. The seat of inoculation was hairless, congested, and showed small wounds due to the animal biting the part. In a second rabbit, that only received half the amount, the result was negative. In a third the material injected had been passed through a Berkefeld filter, and no effects were produced. Portions of the brain of a rat dead of the disease were prepared, and injected into rabbits and grey mice. In one rabbit death occurred in four days with similar local lesions to those described above; in another that died on the same day paralysis of the hind-limbs was observed, with spasmodic movements of the fore-limbs, turning of the head backwards repeatedly, and gnawing violently at the seat of inoculation in the right thigh. Clonic contractions of the muscles of the head and forelegs set in prior to death. A third rabbit and two grey mice also succumbed after inoculation, after showing similar symptoms to those mentioned above.

Symptoms.—In *Cattle* the symptoms observed were loud bellowing, spasmodic stamping with the hind-feet, in some cases loss of appetite, in others the appetite may be maintained for a time, salivation, tympanites, persistent rubbing and scraping of the muzzle, face, and around the eyes, and less frequently of some other part of the body. These regions were denuded of hair, and bleeding patches surrounded by œdematous swellings were produced. Death occurred in from twelve to thirty-six hours; in some instances the course was from one-half to one and a half days.

In the *Dog* and *Cat* dulness, restlessness, profuse salivation, loss of appetite, frequent vomiting, and evidence of pharyngeal paralysis, were the leading symptoms presented. In half the cases observed various regions of the head appeared to suffer from intense irritation, and the animals constantly scratched these parts with their claws or rubbed them against surrounding objects, so that extensive abrasions of the skin resulted, or even injury to the deeper tissues. In other instances the above symptoms were absent, and the animals showed evidences of pain by emitting groans and cries.

The disease is differentiated from rabies by its more rapid course, by the absence of an aggressive demeanour and any tendency to bite, by the fact that the virus is present in all the organs and in the blood, and can be transmitted from preparations of these materials to other animals by inoculation; also the incubation period of such artificial infection is only a few days, and the seat of inoculation becomes painful to a marked extent.

Hutyra was of opinion that further investigation was necessary in connection with the disease.

CANINE DISTEMPER

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Nomenclature.—Until the end of the eighteenth and at the commencement of the nineteenth centuries the term "distemper" was applied to various diseases of mankind and animals, and was synonymous with the terms "malady," "disease," "disorder," "complaint," "epidemic," "epizoötic," "contagious or infectious disease or disorder," "murrain," etc. But since the designation "influenza," introduced into human medicine by the Italians in 1741, commenced to be adopted by veterinarians at the beginning of the nineteenth century, the expression "distemper" gradually fell into desuetude, until at the present time it is only retained to designate that contagious disease of the dog now under consideration. Occasionally, however, strangles of the horse is termed "equine distemper."

Synonyms.—Distemper of the dog; Canine influenza; Contagious or infectious catarrh, pharyngitis, tonsillitis, laryngo-tracheitis, catarrhal fever, bronchitis, broncho-pneumonia, pneumonia or gastro-enteritis; Kennel or show typhus; Canine pest or plague; Canine strangles; Malignant catarrhal fever; Canine small-pox, dog-pox, or variola; Nervous fever; Pernicious anæmia; Quinsy; Snuffles; Snifters; Husk, etc.

French: Maladie du jeune âge; Maladie des chiens; La grippe; Gourme; Fièvre catarrhale maligne des jeunes chiens; Affection typhoïde des jeunes chiens; Pneumonie infectieuse; Pasteurellose du chien, etc.

German: Staupe; Hundestaupe; Hundeseuche; Hundekrankheit; Sucht, etc.

Italian: Cimurro.

Spanish: Reuma.

South Africa: Hondziekte.

Argentine Republic: Moquillo.

Definition.—Distemper is a prostrating contagious and inoculable disease peculiar to the dog, and probably to some of the other carnivorous animals, running a variable, prolonged course, with or without pyrexia, and characterised generally by a cutaneous eruption, catarrh of the various mucous membranes, and is often followed by serious sequelæ in connection with the nervous and cutaneous systems.

ANIMALS SUSCEPTIBLE.

Besides the numerous varieties of the domestic dog, the ferret is also affected with this disease. In this latter species it is generally contracted from distempered dogs. It is generally asserted that it also attacks the wolf, fox, jackal, hyæna, and the various other species of wild canidæ. According to some authorities, it is communicated to monkeys, but this statement requires proof before it can be generally accepted. As to the domestic cat, British authorities are unanimous in considering that canine distemper is not communicated to it, at least by natural contagion or infection; nor is feline distemper transmissible by the same means to the dog. On the contrary, Krajewski, Laosson, Lignières, and others, assert that, as the result of their inoculation experiments, the cat contracts the disease from the dog, and reciprocally. Man is absolutely immune to it, as well as to the feline malady. The horse, ox, sheep, goat, pig, and fowl, are also refractory to the disease. Some authorities recognise distemper in the rabbit and guinea-pig.

HISTORY.

It is uncertain if canine distemper was known to the ancients, but from the writings of Virgil,* Elian,† and other ancient authors, we find mention of a disease affecting the dog which they termed *cynanche* or *angina*, and which appeared to have occurred as an epizoötic, the lesions being confined chiefly to the throat in the form of abscesses. Aristotle‡ merely mentions the fact that the dog was affected with three diseases, of which *cynanche* was one; but he does not give us any details by which we can recognise it. Beyond this we cannot discover any writings by which we can ascertain if distemper was recognised or occurred until the year A.D. 1028, in which, according to Laosson, it decimated the canine race in Bohemia. Stegmann stated that it was observed in Germany in 1697. Ulloa, in 1746, described in his "Historical Travels in America" an eruptive disease of the dog, observed generally by him in 1735 in South America. According to Heusinger, it was brought from Peru into Europe soon after the discovery of the New World. It is asserted that the disease was first seen in Spain, from whence it spread into France, Germany, and other countries in Europe. It is unknown whether the epizoötic, complicated with gangrenous angina, which raged over Southern France in 1714, was this disease or not. The old French writers considered that distemper was imported from England, whereas the English sporting writers in the middle of the eighteenth century unanimously declared it was first introduced into the British Isles from France. In France, however, according to De la Conterie, in his "Vénerie Normande," a work published in Rouen in 1760, it raged there for twenty years, thus showing it was encountered in that country as far back as 1740 or thereabouts. In Germany it was very prevalent in 1748; in the Ionian Isles, Gibraltar, and England in 1759; in Italy in 1764; in Russia in 1770; and in Siberia in 1783. It was encountered in Norway and Sweden, and again in Siberia, where it seriously affected the transport, in 1815. In Greenland it was first observed in 1859, when it practically decimated the canine species. In Iceland the disease has been so disastrous as to threaten the canine race with total extinction. In Terra del Fuego it is said to be unknown. With the exception, perhaps, of this last-mentioned place, it is now known to exist all over the world where dogs are found. The disease is generally confined to certain centres, where it seems always to exist as a mild enzoötic, and after a long period it suddenly becomes a panzoötic, and then gradually an epizoötic, until it finally regains its former enzoötic character.

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* "Georgics." † "History of Animals," Book iv., chap. xl.

‡ *Ibid.*, Book viii., chap, xxii.

Before we close this section, attention should be called to one fact in the history of this malady. Until the epoch when distemper was first generally observed and recorded in Europe, very few of the epizoötic diseases of animals attracted much attention. When, however, they raged with such virulence and occasioned such great loss as to alarm Governments, the latter began to bestir themselves, and consider schemes to combat these outbreaks. Ultimately it was decided to study the diseases of animals, and the result was the foundation of the first veterinary school in Europe, in the city of Lyons, in 1761. This was followed by the formation of others in France and other countries, until now there exists in nearly every civilised State, schools for the study of the diseases of animals, their prevention, and their treatment. Hence distemper, among the other epizoötic maladies, was observed and noted.

Probably as time advances we shall, by further historical research, discover that canine distemper was known to have been in existence in this and other countries long before the periods given above. Since this epoch the disease has from time to time raged, with more or less virulence, in various civilised countries, and occasioned great mortality. It was particularly severe in England and Germany in 1805.

GENERAL REMARKS.

Distemper has been compared to various diseases in man, such as gastro-enteritis, small-pox, measles, etc., and to influenza, strangles, and other affections of horses and cattle, but in reality it has no connection with any of them. The question of the dog having several distinct maladies included under the generic title of " distemper " has often arisen, and those who have attempted to separate them into different morbid entities have ignominiously failed. Louis Barrier, in 1803, endeavoured to show by clinical description that there were two maladies—viz., small-pox and true distemper. Youatt likewise described these two diseases, but it is interesting to note that he only casually refers to an eruption occurring during distemper, and that his account of small-pox agrees in almost every particular with the eruption observed in the former affection. It is quite evident that Youatt confounded at least one form of the manifestation of the eruption with mange, for he says : " Distemper frequently follows mange, and especially if mercury has been used in the cure of the malady. When we see a puppy with mange, and that peculiar disease in which the skin becomes corrugated, and more especially if it is a spaniel and pot-bellied or rickety, we generally say that we can cure the mange, but it will not be long before the animal dies of distemper; and so it happens in three cases out of four." But in another place he observes : " In the pointer, hound, and greyhound, there sometimes appears on the whole of the chest and belly a pustular eruption, which peels off in large scales. The result is usually unfavourable. A more general eruption, however, either wearing the usual form of mange or accompanied by minute pustules, may be regarded as a favourable symptom." No doubt every experienced practitioner can recall to his mind that in the early part of his career he encountered similar cases to those mentioned by Youatt, and mistook the early generalised exudative eruption of distemper for mange, and treated it as such, the result being that he diverted the infection to other parts and thereby induced death. Blaine, successively the master, partner, and predecessor of Youatt, makes no such pretensions as his contemporary, but contents himself by pointing out " its versatility of character in different seasons. . . . One year it may be accompanied by a biliary affection, often associated with a pustular eruption; in another period by a phlegmonous tumour of some part of the body, but principally the head ; in the summer of 1805 with a peculiar spasmodic colic, which neither constipated nor relaxed the bowels, but after continuing two or three days usually terminated fatally. One year the disease will be marked by an obstinate diarrhœa ; another with an unusual tendency to epilepsy and spasm (chorea); while in a third a very fatal and malignant putrid type will prevail; and, finally, in a particular year, nearly every case will show a universal eruption over the body, chiefly under the abdomen and down the inside of the thighs, and is not unlike that of small-pox." Apart from distemper, Hertwig[*] (1853) described several other fevers, some of which appeared to be contagious, but from his description of them many appear to be some form or other of distemper. Camille Leblanc[†] (whose father, Urbain Leblanc, somewhere about 1841, described the eruptive form as a distinct disease, and gave it the name of "variola") wrote, in 1857, a good account of distemper, which he did not consider as a distinct and well-defined affection, but as one consisting of several forms, some of which were quite distinct, yet in others they were combined by one complicating the other. He did not regard it as correct to believe that the dog suffered from only one contagious malady, while man and other animals had many. But in order to fall in with common usage and to prevent confusion and error, he adopted in his description of the disease the generic title of distemper, and concluded his remarks upon the nature of it thus:

"1. That what we call distemper is composed of several affections, of which the principal and most frequent has its seat in the air-passages, and is termed *bronchial catarrh;* whilst others attack the intestinal mucous membrane, nervous system, or skin.

"2. That these affections may exist separately, but often one is complicated by the other.

"3. That they should be classified in the following order:

"(*a*) *Bronchial Catarrh,* commencing by *nasal catarrh,* terminating by capillary bronchitis or lobular pneumonia, and often complicated by conjunctivitis and keratitis.

"(*b*) *Intestinal Catarrh,* frequently complicated by aphthous stomatitis, and terminating generally by dysentery, rarely by icterus.

"(*c*) *Diseases of the Nervous System* { Congestion of the meninges.
Chorea.
Posterior paralysis.

"(*d*) *Eruptive Disease* (*pemphigus* or *rupia*)."

Trasbot[‡] (1868-1903) stoutly maintained there were two, if not three, distinct contagious diseases confounded under the name of distemper: (1) The true distemper, an inoculable eruptive disease or variola of the dog ; (2) *la grippe*, or infectious pneumonia, which commonly complicated distemper under ordinary circumstances ; and (3) a special infectious pneumonia. Mégnin* (1893-1906) considers that *gourme* (an old French term denoting a depurative malady such as measles, strangles, or distemper, supposed to have been derived from the Celtic *gormes*, the equivalent for *pus*) is generally confused with *la grippe*, the malignant catarrhal fever or distemper of the English. According to him, the first is a disease of nutrition seen in young puppies and characterised by catarrh and a vesicular eruption, and often complicated by keratitis, bronchitis, diarrhœa, dysentery, ulceration in the mouth, or disturbance of the nervous system. The eruption he subdivides into two forms : a *vesicular* and an *eczemato-impetiginous*. He considers malignant catarrhal fever, " la grippe," " pneumonie infectieuse," " typhus des chenils," or " des expositions canines," as a distinct disease, as it attacks dogs of all ages under the form of an infectious pneumonia, and also ferrets. But on comparing his descriptions of the two diseases, it is difficult to recognise the border-line between them. In 1898 Klett studied an epizoötic in the dog in Stuttgart, and concluded that it differed essentially from distemper. Cadiot and Breton describe in their work on " Médecine Canine," published in 1901, three diseases usually confounded under the denomination of distemper—viz., distemper, hæmorrhagic gastro-enteritis, and contagious broncho-pneumonia, the last being differentiated from distemper by the absence of vesicles. A few observers pretend there is also another disease—viz., *influenza*, or epizoötic catarrh ; but from their description of it one cannot conclude that it is distinct from distemper (see p. 867). Others, again, attempt to distinguish a disease commonly termed " husk," which is probably a form of distemper chiefly confining itself to the larynx and pharynx, giving rise to a husky cough and an expectoration of frothy, somewhat thick mucus ; it is highly infectious, and seems to be spread through the medium of the atmosphere. It is said to be an independent disease, but often associated with distemper. Furthermore, Glass of Pennsylvania describes an " infectious bronchial catarrh," or " false or bench-show distemper," which he considers quite separate from the true disease. In the differential diagnosis of the two maladies, the chief characters relied upon are that the majority of dogs affected by the false variety are not rendered immune by a first attack, but may have a recurrence of it the next year or so, and there are no eruptions. From his clinical description, however, there seems to be no true distinguishing characters between them.

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[*] Dr. C. H. Hertwig, "Krankheiten der Hunden," 1853, and second edition, 1880. French translation of second edition by Ad. Scheler (Brussels).

[†] Camille Leblanc, *Nouv. Dict. Prat. de Méd. Vét. de Bouley,* art., "Chiens (Maladies des)," tome iii., 1857, pp. 612-630.

[‡] Trasbot, *Rec. de Méd. Vét.,* 1868; *Arch. Vét.,* 1879, p. 161; *Bull. Soc. Cent. Vét.,* 1885, pp. 306, 412, 443; *Rec. de Méd. Vét.,* 1903, Août 30; *Dict. de Méd. Vét. de Bouley,* art., "Pneumonie Infectieuse du Chien," 1890, tome xviii., pp. 177-181.

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* Mégnin, " Le Chien : Élevage, Hygiène, Médecine," 1893-1906.

Finally, while Lignières accepts Carré's view that distemper is due to a filtrable virus which lowers the vitality of the system, he maintains that Pérez's bacillus of ozæna and the pasteurella can also enfeeble the system, and permit the invasion of various bacteria. If this theory could be proved there would be a plurality of diseases included under the generic appellation of distemper.

There is, however, one fact not yet considered in comparing canine distemper with the various human eruptive or catarrhal maladies, and that is, in nearly all of the latter diseases the same complications as seen in distemper would be observed if the human patients were submitted to similar secondary conditions. In the various human maladies alluded to above we observe as complications catarrh, keratitis, gastro-enteritis, pneumonia or bronchitis, meningitis, convulsions, paralysis, chorea, etc. They are not special to one infectious disease, but common to most, if not to all. Probably they would be much more prevalent in man if he were submitted to the same conditions as distempered dogs. As a rule, the human subject, when ill, is isolated, ordered to bed, and dieted suitably, even from the early stage to that of convalescence. Not so the dog, who is often a filth-eater and exposed to unhygienic conditions. Therefore we should not conclude, because we get such complications as just mentioned in an infectious disease of the dog, that he must necessarily be suffering from the effects of one specific etiological factor.

In concluding these general remarks, the writer suggests that, although there may be several specific morbid entities included under the generic term of distemper, there are, as yet, no reliable data to distinguish one from the other, and thinks that until bacteriological research, in conjunction with clinical study, settles beyond any dispute the question of the unity or multiplicity of affections included in this generic term, the various manifestations of it should be accepted as belonging to distemper. He is of opinion, as the result of a comparative study of the diseases of swine, dogs, and horses, that distemper is an analogous disease to swine fever and swine plague on the one hand, and to influenza, strangles, and contagious pneumonia of equines on the other.

NATURAL IMMUNITY.

There are many kennels of dogs that seem to resist the infection, at least for a more or less lengthy period. Experience shows that where this is obtained distemper has many years before made such havoc in a particular kennel or family that only the fittest have survived, and afterwards the more delicate or susceptible members of their offspring have gradually been weeded out by death, from subsequent visitations, so that only a very resistant race remained. A dog may even resist several or repeated exposures for a long period, and then ultimately yield to infection, especially when the latter is very virulent, or when the animal is placed in hypercontaminated surroundings or exposed to depressing or predisposing causes, so as to render him susceptible to such infection. Frequently, however, this supposed resistance is really acquired by the animal having recovered from an overlooked or mild or perhaps atypical attack of the disease. Dogs bred and reared in contaminated surroundings often acquire this relative immunity. The supposed natural resistance of the shepherd's dog, or mongrel, is probably due to the conditions under which he lives, rather than to any inherent immunity. Put the same kind of dog under similar conditions as a well- or in-bred dog to contract the disease, and it will be found that he does not possess the degree of immunity that is imagined by many. Keep a lot of mongrels together as one would foxhounds with distemper, and the majority become as seriously affected and as many succumb. This has been the personal experience of the writer. Original natural immunity, unless there be an occasional weeding out by the disease, is overcome after several generations, and the family or strain becomes again susceptible.

RECURRENCE.

Although recovery from distemper gives a certain amount of security against future attacks, it by no means follows that absolute immunity is conferred for the remainder of the animal's life. The writer has often observed recurrences, in which the same dog has developed the disease two or more times. This has also been noted by intelligent dog-breeders, kennelmen, and huntsmen, as well as by veterinary surgeons, the latter including Delabere P. Blaine, Youatt, Mayhew, Carless, and Friedberger and Fröhner. Hertwig, Röll, Sewell, and Law, give facts proving that dogs do sometimes contract the disease more than once. Death sometimes follows one of the subsequent attacks.

Many writers and the majority of the public traditionally consider one attack of distemper gives absolute freedom from any future attack, but this view is clearly opposed to clinical experience.

ETIOLOGY.

Nearly every one of the older authors noted that this disease was contracted by cohabitation, and many of them recognised that in the endeavour to mitigate its virulence by inoculation with the nasal discharge it was often produced in a more virulent form than when contracted naturally. Youatt, in a paper on "Distemper in the Dog," which appeared in the *Veterinarian*, 1830, says :

"Inoculation with the matter that flows from the nose, either *limpid*[*] or purulent, and in an early or advanced stage of distemper, will, with few exceptions, produce the disease, yet I have failed to communicate it even by this method. Inoculation used to be recommended as producing a milder or less fatal disease. So far as my experience goes, the contrary has been the result." This quotation clearly shows that at an early date distemper was recognized as an inoculable disease, and that the initial limpid discharge was infective, as demonstrated more recently by Carré.

The experimental mode of infection has also been recorded as successful by Renner and Karle, in 1844; Weiss, in 1852; Hertwig, in 1853; Camille Leblanc, in 1857; Röll, in 1867; Venuta,[†] in 1873; Trasbot,[‡] from 1868 to 1879; Krajewski,[§] in 1881; Bryce, in 1882; Lignières, in 1900; and H. Carré, in 1905.

Smearing the nasal discharge on the nostrils of a susceptible dog is the surest means of experimentally contaminating the animal. However, neither inoculation nor smearing the nostrils with the discharges are natural means of infection, but they go to show that the virulent agent is contained in the discharges, that the disease is transmissible, and that they are the most certain methods of introducing the contagium into the system. It is, nevertheless, uncertain how the contagious element gains an entrance by natural infection, whether by *inhalation* or by *ingestion;* probably the latter is the commonest mode. This is, however, a problem that yet remains to be solved.

According to the recent researches of H. Carré,[‖] Chief of the Laboratory of Researches on Infectious Maladies at the Alfort Veterinary School, the specific virus or the essential or contagious element of the disease is invisible, passes through the very porous meshes of the bacteriological filter, and is uncultivable on various media, the latter remaining sterile. These researches, it may be advisable to point out, were supervised by his distinguished chief, Professor Vallée.

The most active virus is that found in the limpid, serous, or watery nasal discharge seen in the early stage of the complaint; it is equally as virulent when obtained from the pericardial effusion after the death of an animal from peracute or rapid distemper. It is then so virulent that two or three drops of the liquid injected under the skin of a susceptible young puppy may produce, after two or three days' incubation, and without any lesions at the point of inoculation, marked dulness, disinclination for movement, and moaning, death taking place, after a more or less comatose condition and an extremely abrupt lowering of temperature (95° to 91° F.), within six or seven days. The sole lesion consists in a pericardial effusion, which is somewhat abundant, amber-tinted, and limpid. The blood contained in the heart, as well as the serosity, is sterile. Carré considers this acute form as rare in the natural disease, but the writer, in his paper "On the Etiology and Some of the Phases of Canine Distemper," presented to the Central Veterinary Medical Society on December 3, 1903, and which afterwards appeared in the *Veterinary Record*, December 19, 1903, stated therein: "Frequently dogs, especially young animals, after being exposed to infection, die without showing much premonitory disturbance, and on making a post-mortem examination few, if any, naked-eye lesions are discernible. Such animals seem to be affected with acute intoxication, and die before lesions have had time to form. At the commencement of an outbreak these cases are somewhat common, and liable to take one off one's guard as to the true nature of the cause of death."

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[*] The italics are the writer's.—H. G.

[†] Venuta, "Natura Contagiosa del Cimurro nei Cani," *Il Medico Veterinario*, 1875, p. 289.

[‡] Trasbot, "Maladie dite des Chiens," *Archives Vétérinaires*, 1879, p. 161.

[§] A. Krajewski, "Die Staupe, ihre Contagiosität und Uebertragbarkeit durch die Impfung," *Revue für Tierheilkunde*, 1881, Band v.; *ibid.*, 1882, Band vi.

[‖] Carré, *Compeès rendus de l'Académie des Sc.*, 1905, pp. 689 and 1489; *Revue Vét.*, 1905, p. 321; *Revue Générale de Méd. Vét.*, 1905, p. 321; *ibid.*, 1906, pp. 71 and 649; *Journ. Comp. Path. and Therap.*, vol. xix., 1906, p. 127; *Vet. Record*, 1906, vol. xviii., p. 146.

The filtered and largely diluted discharge, or, again, $\frac{1}{100}$ c.c. of pericardial serosity—products not containing any colorable or cultivable microbe—cause, after three or four days, a high elevation of temperature, rapidly reaching 105° F. or more, and lachrymation, followed by coryza, and afterwards by a somewhat hoarse cough, especially when the animal moves about. At the end of eight to ten days beautiful vesico-pustules appear on the places of their election—namely, the abdomen, thighs, etc. The lungs become affected; the nasal discharge, at first slight, serous, or sero-purulent, takes on a purulent character, and continues to flow from the nostrils, and the animal dies after a period of from one to three weeks.

The post-mortem examination shows, beyond the well-known pulmonary lesions such as hepatised or purulent patches, cardiac changes and effusion into the serous cavities. The cardiac lesions may be present without any of the other organs being visibly affected. They consist of hæmorrhagic spots, ill-defined yellowish patches, or chiefly, in the case of very young puppies, lenticular foci embedded in the thickness of the myocardium. In the centre of these lesions the muscular fibres have undergone changes, and are inundated by an abundant infiltration of leucocytes. Besides the exudate in the cardiac sac, a quantity may sometimes be found in the pleural and peritoneal cavities. Carré firmly believes that the early pneumonic form may supervene from the filtrable virus alone, without the action of cold playing any part. The *blood*, if collected at the time the temperature rises and inoculated into a susceptible dog, transmits the disease, thus showing that the blood behaves like the nasal discharge and pericardial effusion, and contains the infection at the early stage of distemper. Susceptible animals cohabiting with the experimentally infected animals contracted the disease, and when they recovered they could no longer be reinfected naturally or experimentally. In the more resisting animals, the filtrable virus produced a disease having a longer course and showing all the usual localisations. The blood, muco-purulent discharge, and pericardial, pleural, and peritoneal exudates in the later stages of the disease do not give an infective filtrable virus—at least, the disease is rarely transmitted experimentally by them. The same may be remarked of the pneumonic lesions, which are said not to contain the virulent material. Probably the saliva, tears, urine, and fæcal matter are virulent, at least during the earlier stages of the disease, but it is difficult to determine this by inoculation methods. The writer's clinical experience of canine distemper is that at all stages the disease is *naturally* infective to susceptible animals coming in contact with affected animals. He believes that the teachings of Carré—*i.e.*, as to the disease in its later stages not being contagiferous, and that only the virus soiling the skin and surroundings of the sick animal is communicable—seem not only erroneous, but would, if carried into practice, prove disastrous. These remarks also apply to his statement that the purulent secretions quickly lose their virulence.

The cutaneous eruption, noticed by nearly every observer, and considered by many, including Viborg, Jenner, and Trasbot, among others, to resemble that of variola, is due, as will be seen in the section on Bacteriology, to a micrococcus appearing singly, double, or in the form of a chain, which is found as a saprophyte in the intestine of the normal dog. This eruption not only appears in distemper, contracted naturally or through the medium of the filtrable virus of that disease, but also after an injection of the filtrable virus of foot and mouth disease, even after an animal has recovered from distemper and shown the eruption of the latter disease. The vesicular pustules do not contain the filtrable virus.

It is supposed the filtrable virus gives rise to disturbance of nutrition, whereby the defensive forces of the system are diminished or overcome, so that a good soil is thus prepared for the easy invasion of saprophytic microbes; the latter, by repeated passage through various distempered dogs, or by undergoing some changes under unknown conditions, may have acquired a certain degree of pathogenic or virulent power.

Without a doubt these microbes vary in kind as well as in virulence, depending on the dog or on the particular outbreak. Under certain conditions, such as the various occasional or secondary causes, they locate themselves in this or that organ, and thus set up a laryngitis, a bronchitis, a broncho-pneumonia, a pneumonia, a gastro-enteritis, a meningitis, a meningo-encephalitis, etc. One secondary infection may act alone or concurrently with another or several secondary infections, and, in conjunction with the filtrable virus, they form a mixed infection. This produces in fresh or susceptible dogs a virulent type of the disease, which no longer resembles the original type, and leading one to conclude that there are in the dog one or more maladies included under the heading of distemper, and due to different essential factors. Even some dogs in the convalescent stage, and tending towards recovery, if brought in contact with others suffering from a mixed infection, are liable to be recontaminated, and to develop a more virulent and more fatal attack than their original malady. Such has been the experience of the writer, and it is only on the hypothesis he has just mentioned that he can account for the course the disease occasionally follows. Further investigation on these points is necessary and desirable, and is likely to be productive of important results.

BACTERIOLOGY.

The credit of being the first to seek for a micro-organism as being the specific cause of distemper is due to Semmer, of the Dorpat Veterinary School, who in 1875 isolated a micrococcus and a short, extremely slender bacillus in the blood and lungs; the bacillus was plentiful in the spleen, liver, and kidneys, and he concluded it was the causal agent. In 1888 he further isolated bacilli and micrococci. Friedberger, in 1881, rediscovered the former microbes of Semmer. Krajewski, the same year, observed micrococci in the tissues and blood. Laosson, in 1882, confirmed Semmer's researches, and cultivated the micrococci and bacilli in broth made of dog-flesh, and with these mixed cultures he considered he was successful in transmitting the disease. Rabe, in 1883, isolated micrococci, diplococci, and streptococci from the cutaneous pustules and conjunctival and nasal discharges, but could not reproduce the disease with them. Friedberger, in 1884, confirmed Rabe's statement, but denied that the micro-organisms possessed any specificity. Mathis of Lyons, in 1887, detected in the fluids of the body, tissues, nasal and ocular discharges, and cutaneous pustules, micrococci, and cultivated them in neutral or slightly alkaline veal-broth; the micrococci were single or united in pairs, forming diplococci (figure-of-eight or dumb-bell-shaped bacilli), or united in the form of a chain (streptococci), and having a diameter of 1 to 3 μ. From the lungs he obtained identical cultures. He obtained pure cultures up to the seventh generation, which, on inoculation into susceptible dogs, generally produced a rapid elevation of temperature, pustules on various parts of the body, and catarrhal symptoms. Very young animals often died, as was pointed out by Trasbot with his experiments by using the crude virus of the pustules, but immunity to the disease was given to the survivors. Marcone and Meloni, in 1888, isolated a micrococcus from the pustules which, when cultivated and inoculated into dogs, caused symptoms similar to those seen in distemper; this microbe was rediscovered by Kitt in various pustules other than those of distemper, and it produced pustules, but nothing else. Lignières, Carré, and others, state it is the same micro-organism as that isolated by Mathis in 1887, and they generally admit it is the specific cause of the pustules. Mathis, on the other hand, considers that his micro-organism is identical to the diplococcus discovered by Semmer, and reviewed by Friedberger, Krajewski, Rabe, and Laosson. The late Sir Everett Millais,* Bart., discovered in distemper lesions two kinds of bacilli, which he termed *A* and *B* respectively, and also a micrococcus. All of them liquefied gelatine. He concluded (1) that *A* bacillus had nothing to do with distemper—at least, he was unable to ascertain what connection it had with the disease; (2) that *B* bacillus was the primary cause of the malady which was produced when the culture was smeared on the nostrils; and (3) that the micrococcus was the cause of the pulmonary complication—viz., pneumonia—of distemper. Legrain and Jacquot, in 1890, detected motile micrococci in the pustules, either singly or united in the form of diplococci or streptococci, and having the diameter of 0·6 to 0·8 μ; they grew on gelatine, agar, broth, and potato. The cultures on inoculation caused patches of erythema and the formation of pustules, but did not confer immunity. Schantyr,† in 1891, demonstrated the multiplicity of microbes in distemper, and endeavoured to show by their biological and pathogenic characters that there were in reality three affections—viz., (1) A typhoid fever of the dog; (2) true distemper; (3) abdominal typhus.

Galli-Valerio,‡ in 1895, isolated an ovoid bacillus, measuring 1·25 to 2·5 μ long, and 0·31 μ broad, from the brain, lung, and spinal cord of affected animals. It exhibited unusual pleiomorphism, and occurred either in pairs, alongside of each other, or in long threads; it stained by the method of Gram and liquefied gelatine. Rabbits and guinea-pigs proved immune. In agar cultures it formed small white points, and on potato a white transparent coating. In some of the ovoid bacilli polar staining was observed. The cultures inoculated into dogs are said to have produced distemper, especially the nervous type of the disease. In a later paper (1896) he stated that the bacillus was Gram negative, and in 1898 he further stated that the spores he observed were involution forms. He and Piana in 1898 placed the organism in the coli-typhoid group. Jensen, in 1896, isolated streptococci which he asserts are the cause of the pneumonia of distemper.

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* *Veterinary Journal*, May, 1890. *Ibid.*, May, 1891.

† Schantyr, "Untersuchungen über die Mikroörganismen der Hundestaupe," *Deutsche Zeitschrift für Thiermedicin*, 1892, Band xviii., p. 1.

‡ *Centralblatt für Bakteriologie und Parasitenkunde*, 1896, Band xix., s. 694. *Ibid.*, 1908, Ref. Band xli., s. 563.

Babes and Barzanesco, in 1895, found a very fine motile bacillus, without spores and not staining by Gram. It was isolated from the lungs, cardiac blood, and liver.

V. Babes and C. Starcovici, of Bukarest, in a paper on "Distemper" presented to the Berne Veterinary Congress in 1897, concluded thus: "(1) That we have found in two cases a bacillus which reproduces in dogs the general phenomena of the disease, and lesions localised in the respiratory organs and sometimes in the alimentary canal. (2) That this bacillus is distinguished from all those microbes hitherto described as present in distemper—viz., the diplococci of Laosson, Krajewski, etc., those of Mathis, the bacillus of Semmer, and that of Schantyr. (3) That this pathogenic microbe which we have isolated, when cultivated, determines a typical disease, only differing from natural distemper by killing the dogs with greater rapidity and with more certainty. (4) That this microbe is also pathogenic for the guinea-pig, rat, and rabbit. (5) That it is recoverable from the blood and lung, and sometimes from the other organs, of the experimental animals. (6) That, on the one hand, it resembles the bacillus of human typhoid fever, and, on the other, that of the bacillus found in human influenza. (7) That it is in certain cases associated with other microbes, of which saprogenous bacilli and a particular streptococcus are the most important. (8) And, finally, that canine distemper does not constitute a clinical unity, and cannot any longer be considered as a pathological entity from an etiological point of view. While certain cases are produced by our microbe, there are others that are caused by micro-organisms described by other authorities, or, what is very probable, by others at present unknown."

Taty and Jacquin, in 1898, found diplococci in the central nervous system, and they regarded them as causal organisms.

Jess of Charlottenburg announced in the *Centralblatt für Bakteriologie*, Band xxv., April, 1899, that he had found a very small bacillus, 1·8 to 2·3 μ in length, and 0·6 μ in breadth, in the nasal and conjunctival discharges, the blood, peritoneal effusion, and also in the organs. On being stained with carbol fuchsin, the bacilli exhibited polar staining; and those from the bouillon cultures were completely stained. The cultures at the seat of inoculation caused an extensive swelling, and the general symptoms lasted for a week; they killed guinea-pigs within thirty-six hours, and white mice were also susceptible. The agar cultures showed a pale grey coating, with serrated edges; on potato they exhibited themselves as an extensive white velvety coating, and the bacilli appeared always in rod form. Dogs inoculated showed the usual symptoms of distemper. Petropawlowski, in 1899, isolated a bacillus which he considered as specific; but Mari concluded this was only a colon bacillus. Monckton Copeman, in 1900, claims to have discovered, when working on the subject with the late Sir Everett Millais, Bart., ten years before, a small cocco-bacillus, which stains with the ordinary aniline dyes, but is decolorised by the method of Gram. It liquefies gelatine slowly at room temperature. Broth cultures of a week old, injected under the skin of the abdomen, often induce an attack of distemper, which terminates fatally in about a week from the time of inoculation. In conjunction with Millais, he induced a non-fatal attack of distemper in a large number of dogs by inoculation of the nasal mucous membrane. This micrococcus, when grown in broth media, not infrequently forms chains of considerable length. Apparently it is the same organism isolated and grown by Semmer, Laosson, Mathis, etc.

Lignières,* and shortly after him the late Dr. Phisalix† of Paris, in 1900, isolated a very long and fine bacillus in a pure state from the blood of the heart during the early stage of distemper, before secondary infections had set in. According to Phisalix, it may be obtained, when associated with other bacteria, by inoculation of the peritoneum of the guinea-pig with cultures from the cerebro-spinal fluid. This long bacillus, after its first passage through the guinea-pig, commences to change its aspect, and soon manifests itself as a very small cocco-bacillus which does not form chains. Virulent cultures of this organism, which belongs to the group *Pasteurelloses* (having as their type the fowl cholera organism), when injected into susceptible dogs, induce the various clinical manifestations of distemper—viz., nasal and conjunctival catarrh, vesicular pustules, lung complications, gastro-enteritis, opalescent and ulcerative keratitis, chorea, paralysis, meningo-encephalitis, and arthritis; and at will, according to the dose, the acute, subacute or chronic disease, the last after about three or four weeks' incubation. Lignières has also reproduced it experimentally in the rabbit, guinea-pig, and mouse, and still also after its virulence has been increased by a certain number (twenty to thirty) of passages through the guinea-pig, in the pig, sheep, ox, horse, ass, fowl, duck, and pigeon. This organism is not easily found after the first few days of infection, as its place is yielded to other microbes, and thus secondary infections arise. According to Lignières, it frequently performs an effaced rôle, and may not produce effects for a more or less lengthy period. Both this authority and Phisalix assert it is found in the blood of the heart and other organs in acute distemper, and that they have not been able to isolate it from the nasal, tracheal, or pulmonary mucus. Phisalix has constantly found that the microbe isolated from the dog and passed through this animal only rendered the bouillon turbid in a similar manner to the other pasteurella. Lignières makes an exception of this character for the *Pasteurella canis.*

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* *Recueil de Médecine Vét. Méd.*, 30 Juin, 1900, p. 331; *ibid.*, 30 Juillet, 1900; "Memoir on the Hæmorrhagic Septicæmias," p. 59.

† *Comp. Rend des Séances de la Société de Biolojie* (Séances du 16 Juillet, 1898, du 8 Juin, 1901, du 20 Janvier, 1900, et du 23 Juillet, 1898).

Phisalix has on several occasions found the pasteurella in the form of unusually long filamentary mycelia, having very long articles separated by a clear interspace, bending easily in all ways upon themselves. After a few cultures these long filaments gradually diminish, and at the end of five or six re-inseminations the short forms predominate. Not only do they possess all the cultural and staining characters of the typical pasteurella, but they have similar virulent properties. He has isolated them from the lung and from the pleural fluid. One passage through the dog returns them to their usual form. According to Phisalix, attenuation of this organism, the *Pasteurella canis,* is quickly obtained if a 6 per cent. glycerinated bouillon is used as a culture medium ; it afterwards becomes progressively attenuated with age and by subcultures. But in order to restore it to its former virulence, it is necessary to pass it afresh through the dog, and after thirty-five passages have been made in this animal an increase of such virulence is obtained that an inoculation of $\frac{1}{10}$ or $\frac{1}{5}$ c.c. of the culture thus obtained into the vein is sufficient to kill a young dog of 40 pounds weight in a few hours. According to the experiments of Phisalix, the *Pasteurella canis* forms a soluble poison in artificial cultures, which, when injected into susceptible dogs, acts chiefly on the nervous system, and produces disturbance of nutrition. As a result the natural powers of resistance of the animal are diminished, and the soil is thereby prepared for secondary infections. By inoculating this toxin, the majority of the manifestations of distemper can be produced. Phisalix, by injecting into the veins cultures of the guinea-pig pasteurella, produced cerebral meningitis in the dog, which resembled that occasionally seen in natural distemper of this animal. Lignières, Phisalix, and others, claim that the *Pasteurella canis* gives protection against distemper, but McFadyean, Vallée, and Carré say this organism only causes an accidental infection; Jensen states it plays no part either in causing distemper or typhus; and Vallée and Carré are agreed that it is a normal parasite of the intestine in the dog, and has been found also in the blood-stream by Almy, Bîmes, Sérès, Leclainche, Nocard, Vallée, and others, in various outbreaks of canine typhus. In 1908 McFadyean held that this ovoid bacterium did not appear to have any pathological importance even as a secondary invader in the course of distemper. Pérez, Lignières, and Bisanti fully admit that healthy dogs may harbour the *Pasteurella canis*.

Besides this microbe, various others have been found in the system of the dog dead of distemper by Lignières, and also by Phisalix. According to the former authority, the microbe found in the lung lesions is a saprophyte (*cocco-bacillus fœtidus ozenœ* of Pérez*) living commonly in the nostrils of the healthy dog. Probably this infection may be conveyed to man, and produce in him an attack of ozæna. Phisalix has observed a vast number of microbes, and notably a motile bacillus, in the air-passages and lungs of distempered dogs; also a streptococcus, that is coloured by Gram's method, in the lungs and pleural effusion, and which is very deadly in effect. The *Bacillus coli communis* is very often detected in the mucus of the air-passages during distemper.

Von Wunscheim† of Innsbruck, in 1905, found in the catarrhal and nervous forms of distemper a cocco bacillus of the hæmorrhagic septicæmia type, and probably of the pasteurella type, and named it the *Bacillus canicidus*. Pure cultures reproduced the various forms of the disease. It was obtained from the nasal discharge, cardiac blood, spleen, and kidneys.

Copeman isolated his organism from the brain; Lignières and also Phisalix recovered several organisms from the nervous system; Sabrazès and Muratet‡ did not obtain any growth from the cerebro-spinal fluid; Gallio-Valerio§ found his organism in the lesion of spinal myelitis; and finally M'Gowan did not obtain any growth from any part of the cerebro-spinal system in dogs and cats affected with paraplegia. This last authority considers the nervous lesions are due to toxins, a view held by many for a great number of years, and is in accordance with that maintained with reference to the nervous phenomena observed in human diphtheria.

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* "Annales de l'Institut Pasteur," 1899, tome xiii., p. 937; *ibid.*, 1901, tome xv. p. 409.

† O. von Wunscheim, *Archiv für Hygiene*, Band liii., and Brochure, 1905.

‡ "Bulletin de l'Institut Pasteur," 1907, tome v., p. 173.

§ "La meningo-mielite da Cimurro," *Il Moderno Zooiatro*, 1893., p. 224.

Carré of Alfort, in 1905, and again in 1906, carried out a series of experiments, and found that the virulent material of distemper was contained in the early nasal discharge, and especially when this was in the limpid or watery state; it was also present in the pericardial, pleural, and peritoneal serous effusion of dogs dead of the complaint within the first week after manifestation of symptoms. He also ascertained that if passed through the Berkefeld filter the filtrate contained no visible organism and was sterile. Two or three drops of the filtrate, or of the limpid or serous nasal discharge or pericardial effusion, produced symptoms of distemper and often death; while in animals not quite so sensitive all the usual forms of the malady were induced. The infected animals transmitted the disease to in-contact ones, and if they recovered they were immune to further infection. F. J. Bosc,* of Montpellier, confirms Carré, and, according to his view, "the virus which passes through the filter is formed of infinitesimal protozoa;" and by its symptomatology and the character of its lesions distemper should be placed among the bryocytic or protozoal maladies running an acute course, and in the same category as sheep-pox, foot and mouth disease, and rabies (see Etiology). McFadyean, Vallée, and the majority of microbiologists, have adopted, or are inclined to adopt, the view that distemper belongs to the filtrable viruses. Kregenow† (1909) does not accept Carré's view that the disease is due to an ultravisible virus, and that the advanced purulent nasal discharge is not infective.

McGowan‡ (1910-11) and also Leitch, independently of one another, have isolated an organism from the nasal and tracheal mucus, which is pathogenic to the dog, cat, ferret, rabbit, guinea-pig, goat, monkey, and man. It was never obtained from the blood, but was occasionally found in empyemata of the middle ear, meninges, and from the lungs in cases of pneumonia and empyema. The organism is a Gram-negative bacillus, which, on being stained with carbol thionin, shows polar staining on a constriction in the middle.

The length varies from 0·5 μ to 2·3 μ, and its breadth from 0·4 μ to 0·5 μ. It assumes an almost coccal form when grown in glycerine agar. It is feebly motile, and shows flagella; it does not coagulate milk or liquefy gelatine; it furnishes a brown growth on potatoes. It grows readily anaërobically. Indol is not formed, nor is neutral red reduced. It is said to have produced distemper when administered to the dog and cat by way of the nostrils. It resembles, or probably is identical with, the Millais-Copeman organism. It seems to belong to Lignières' group of salmonella, of which the old swine-fever organism is the type.

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* *Société de Biologie*, Mars 25, 1905; and *Revue Vét.*, 1905, p. 337.

† Kregenow, *Centrablatt für Bakteriologie*, 1909, Band i., p. 326.

‡ *Journal of Pathology and Bacteriology*, 1911, vol. xv.; *Veterinary Journal*, January, 1912.

Ferry* isolated a similar micro-organism to which he has given the name of *Bacillus bronchicanis.* It is a short, narrow bacillus usually found single, but often in pairs. In liquid media it may be found at times in long chains or even filaments. In broth culture, grown directly from the dog, the organism may be larger and more oval in form. According to Ferry, when cultures are taken early in the disease, the *B. bronchicanis* is found in the respiratory tract in every case, and if taken in the first stage is found uncontaminated. The micro-organism was isolated from ninety-seven dogs at autopsy. In sixty-eight cases it was isolated in pure culture either from the respiratory tract or from the blood. In twenty-nine positive blood-cultures it was found eighteen times, in thirteen of which it was unaccompanied by any other organism. It was isolated from the only case attempted during life, in the very earliest stage, by means of the bronchoscope. Ferry holds that "Koch's law has been fulfilled, and cases of typical distemper have been produced by artificial inoculation with pure cultures, the bacillus being again isolated and grown in pure cultures, all under conditions which have precluded infection from any other source."

Researches are still being continued, both in the Alfort Veterinary School and by the Board of Agriculture; the latter up to the present has not issued any report, although the Commission it formed has been sitting several years.

CONTAGION.

The *contagium* exists in the nasal and lachrymal discharges, blood and excretions. But, as already remarked, experimental evidence shows this is of a variable quality, which is only found in the early stage of the disease, and that inoculations made with the blood, viscera, nasal and lachrymal discharges, fæcal material, and urine, in a later stage fail to transmit the malady. Carré believes that the contagium exists only on the soiled skin and coat and in the surroundings after the early stage, and that the purulent secretions rapidly lose their virulence.

Krajewski has found that the contagium is not destroyed by desiccation, or by freezing down to −4° F.; but its action is lessened when kept for some months in a dry condition. Laosson has observed that the nasal discharge exposed to the open air loses its virulence after two or three weeks; according to Carré this occurs in the course of a few days. A temperature of 60° C. sterilises the virulent products.

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* *Journal of Infectious Diseases,* June, 1911.

Most authorities consider the contagium is *volatile* as well as *fixed*, and that it is conveyed through the medium of the atmosphere as well as by cohabitation or by dwelling in contaminated surroundings. According to the observations of St.-Yves Ménard, of A. J. Sewell, and those of the writer, *aerial infection*, if it exists at all, is very slight indeed; strict clinical observation is against it being regarded as a fact. The unpublished observations of F. O. Parsons and of W. H. Flook confirm this, these two practitioners having frequently noticed that distemper was not communicated from the affected dogs to the non-affected when a simple incomplete partition, or merely two wire-netting partitions a few feet apart, intervened between the diseased and healthy animals. Ménard found that a yard or passage separating an infected kennel from a non-infected one, even when both were attended to by the same person, was sufficient to prevent the spread of the disease from the affected to the non-affected. Sewell observed that puppies in a clean hamper suspended for hours from the ceiling of a room where several cases of distemper were present escaped the disease.

As to the disease being conveyed by places temporarily occupied by distempered dogs, the writer has found, time after time, that the very susceptible Japanese spaniels and other delicate breeds do not contract the infection after being placed therein. He has also found that by taking the precaution of washing the hands after touching distempered animals the disease is not communicated by either the attendant or the veterinary surgeon.

The show-benches are frequently incriminated as being the media of contagion, but providing these have been scrupulously washed with soap and water, no fear from this source need be entertained.

It is, however, quite possible, and very probable, that the hands of the attendant, the feeding utensils, bedding, clothing, brushes, baskets, kennels, medicine utensils, and other things contaminated with the fresh discharges of distemper, may, if not thoroughly washed or cleansed, convey the disease to the healthy animals.

According to the writer's experience, the *living animal* is chiefly the means of spreading the disease by *direct contact*, indirect contact or contaminated media only playing a very inferior part in its distribution. However, the veterinary examination at dog-shows assists in the malady being communicated from the apparently healthy (but perhaps suffering from the early and more virulent stage) to the healthy. Exhibitors or visitors touching or patting the exhibits also assist in this direction. According to the observations of nearly every investigator, the cutaneous eruption or discharge does not transmit the contagion.

The question as to when a dog is free from infection after an attack of distemper is difficult to answer, and must to a great extent be left to the discretion of the practitioner, notwithstanding the experimental data to guide him. Clinical experience shows that a dog may be, and generally is, infective as long as he has any discharge, diarrhœa, or cough remaining; and even when every characteristic symptom has disappeared, the animal sometimes remains a "bearer" or "carrier" of the contagium.

SOURCES OF CONTAGION.

The principal sources of contagion are dog-shows, large kennels where dogs are continuously being bred and exhibited, foxhound kennels, homes for lost and stray dogs, dog shops or stores, boarding sanatoria, quarantine kennels, and veterinary infirmaries.

It is a notorious fact that after a dog-show distemper is more prevalent, especially during inclement weather. Dogs from various parts of the country converge to a dog-show, and there a great number of them contract the infection and diverge to country towns, rural districts, and smaller shows, and thereby disseminate the disease. Sometimes a particular show is incriminated when in reality the disease was contracted at a previous show, only to manifest itself after a later show.

In some of the large breeding and exhibiting kennels distemper occurs as a continuous enzoötic, but showing itself in a mild form, which is not often serious to the dogs shown, yet dangerous to all dogs from other kennels that are susceptible or have not had the disease previously. Before one lot of puppies have recovered, another lot are born, and contract the infection after a more or less short period; this goes on nearly all the year round, and thus the source of contagion is maintained. Although after a time the majority of the inmates in such a kennel become relatively immune, showing but very slight symptoms, yet, nevertheless, the contagium causing this varied type is very dangerous to susceptible dogs from other kennels.

A similar state of affairs also prevails in foxhound kennels, where the disease is generally enzoötic. Although some of the puppies coming in "from walk" are blamed for having introduced the complaint, it is in reality the older or permanent inmates, or even some of the puppies that have not been off the premises, that are the culprits. The writer's experience of the attendants in hunt-kennels is that they seem to know very little about the nature of contagion; they usually separate an affected dog from the rest because he is in a bad way and off his food, but as soon as he is on his appetite again they consider he is well, and therefore put him back with his companions, ignoring the fact that he is still capable of infecting the others. In the majority, if not all, of such kennels there appears to be no rigid system of isolation and quarantine; and, as long as the surroundings are pleasing to the eye and the animals feeding well, no care is taken to prevent infection arising from animals that have recently recovered from the disease or those suffering from so mild an attack that the symptoms are overlooked or ascribed to some other affection.

In homes for lost or stray dogs the infection is never absent, because before one lot of dogs is cleared out others are daily coming in, and thus get infected, to pass it on continuously. It is impossible to prevent or to mitigate the infection in such places unless every dog be kept confined, isolated, and maintained isolated until disposed of by sale or by the lethal chamber. This system of isolation would limit the disease, but in carrying it out great expense would be entailed. The manner in which these institutions are at present conducted renders them a source of passive cruelty, as when dogs are sold therefrom they often develop the disease a few days after purchase, and spread it in the localities to which they are taken. Again, the recovered stray or lost dogs often develop the disease within a few days after their arrival at their old home. A curious procedure is adopted by some of these places—*i.e.*, they have an annexe institution away from the main one, where some of the ownerless strays are sent until they can be disposed of by sale. Many of these animals are suffering, or have just recovered, from distemper, and thus the infection comes in contact with those inmates sent there as boarders by private individuals. It seems needless to add that a great number of such boarders contract the disease, and after suffering more or less many succumb or are left maimed. So much for charitable institutions carried on partly by fees and partly by private or public contributions.

The apparently clean and smart dog shops or stores keep up and distribute the disease, which, if the dog has not already symptoms of it, declares itself within a day to a week from the time the animal arrives in his new home. The contagion is kept up by a process similar to that mentioned above.

College and private veterinary infirmaries, boarding and quarantine establishments—even those that profess to take in *no distemper cases*—sometimes maintain, manufacture, and disseminate the malady, chiefly through non-recognition of it during its early stage or some of its phases, and occasionally from carelessness.

Bitches sent away to the stud-dog, or dogs forwarded on approval, not rarely contract or disseminate distemper to various parts of the kingdom. Exported or imported dogs often convey distemper from one country to another.

Dogs suffering from distemper, either at the commencement or during convalescence, being exercised or allowed to run about in the streets of crowded cities, seaside towns, pleasure resorts, parks, pleasure gardens, and other public places, help to disseminate the malady; one case in such places is soon followed by many others. In a district like the West End of London the disease is never absent.

LATENT INFECTION.

According to the observations of the writer and others, the *contagium* may remain latent in the dog's system, although the animal does not manifest any serious disturbance of health. This continues for a time, until some predisposing cause starts it into activity; it may also cling to a dog long after an apparent recovery. This fact agrees with that observed in many of the other contagious or infective diseases affecting animals and mankind.

Latent infection explains the so-called "spontaneous" outbreaks of distemper and other contagious diseases. It is recognised by the Board of Agriculture, who, in order to stamp out the majority of the epizoötic contagious diseases of food animals, act upon this knowledge, and kill off all those affected and in contact, so as to prevent a further spread of such maladies. This system of "stamping out" did not, however, originate with the Board of Agriculture, for it was adopted, at least in the case of cattle plague, in the first half of the eighteenth century.

PREDISPOSING CAUSES.

Although the specific virus is absolutely essential for the development of distemper, there are other, if only secondary, causes to render the animal more susceptible to receive the infection, to continue its progress, and to induce many of the complications or to invite secondary infections during the prolonged course the disease usually runs. These causes act by lowering the vitality of the defensive system, and, in consequence, increase the susceptibility of the given animal.

No *breed* or age is exempt; even the mongrel and the suckling puppy may fall a prey to its ravages. But yet breed and age vary the susceptibility. Dogs of exotic origin are more liable to become infected than the indigenous or hardy breeds. Probably this is due to the fact that in the countries of their origin distemper is unknown or rarely occurs. The Terra del Fuegan dogs, in whose native country the malady is unknown, rarely survive. The same may be said of the Arctic breeds, such as the Eskimo, Icelandic, and Greenlander. Even those exotic breeds, such as the Japanese spaniel, the Samoyede, the Chow-chow, and borzoi, bred and reared in our country for generations, succumb readily to its ravages—the Japanese more than any other. Nevertheless, many of our indigenous breeds that have been too much inbred and too carefully nurtured, under adverse influences readily contract the infection, and furnish a great death-roll. Such are the bloodhound, fox-hound, greyhound, and the sporting dogs, among others too numerous to mention. Notwithstanding this, there are several strains of these various breeds more capable of resisting the inroads of the disease than others. This is not a new idea, but has been observed by sportsmen since distemper has been known, and they have often taken advantage of the fact by crossing the very susceptible strains with those that are more resistant. However, in the case of those breeds that are generally considered to be able to survive the trying ordeal of the complaint, there are many families or strains that are completely wiped out, the writer having encountered such instances in the fox terrier, Irish terrier, Airedale terrier, and other breeds.

Age is also an important factor in this direction. Puppies from the weaning period to that of dentition show but little resistance to the infection, and the majority succumb to its influences. This is especially noticeable in collies, dachshunds, and the various delicate toy breeds. This susceptibility decreases as age advances to middle life, from whence it gradually increases as senility is reached. Probably the resistance of adult dogs is due to the fact that they have survived an attack, or have been relatively immune to the disease in earlier life. No doubt the reason it is seen more frequently in dogs between the ages of three and twelve months is because at this period of life they commence to go out on the public highways and other places, and are therefore for the first time brought in contact with dogs suffering from the malady. And, on the contrary, younger dogs, down to suckling puppies, escape its ravages in consequence of being generally confined to the house, kennel, yard, or stable. However, in the case of breeding establishments, where dogs are kept at stud, strange bitches coming to the dog, or dogs coming from shows, are liable to introduce it to the parents, directly or indirectly, and thus to transmit it to the offspring even before their eyes are open. This is not rarely observed under the conditions mentioned. Again, increased susceptibility to it is observed where a number of dogs, even including mongrels, are herded together.

Seasons and the state of the weather play a very important part in its genesis. It is generally observed that 95 or more per cent. of cases arise during wet, chilly weather, at any time of the year, but more especially during the autumn and spring months. On the other hand, during dry, hot, or cold frosty weather it is comparatively rare, if not altogether absent. Nevertheless, it is also infrequent during a prolonged course of wet weather—no doubt from the fact that the animals have gradually adapted themselves to these humid conditions. Intense cold weather or hot weather, providing it be dry, seems to be inimical; but wet or moist weather, whether it be cold or hot, is favourable to this as to many other infectious diseases. It is the changeable moist weather that is so prejudicial to health, whether in man or in brute. A few German writers consider warmth greatly favours the development and transmission of the contagium. The writer has observed for many years that when the weather is about to change from fine to wet, distemper commences to increase, and continues to do so until just before it changes again from wet to fine. Cases begin to improve and to decrease in number, almost, if not quite, to nil, when fine weather becomes established. Thundery and stormy weather is unfavourable to this, as it is to many other maladies—notably those of the putrid type. Some German writers consider distemper to be more prevalent during the summer months; but this does not agree with British experience, which shows that the disease is more commonly met with during the changeable chilly wet months of the year. Probably this difference of experience is due to the conditions under which the disease is seen in Great Britain as compared with those obtained in Germany. In the latter country the young dogs usually go out for the first time in the summer months into public places, where they are most liable to contract the malady; whereas in Great Britain dogs are bred, travel from place to place, and are exhibited all the year round, but chiefly from October or November to June, and, in consequence, are more liable to contract the disease during this period.

Washing and not properly drying; exposure during bad seasons; exhaustive exercise during wet weather; swimming in wet, chilly weather; running in wet grass; housing in damp, draughty places; railway travelling; change of surroundings, or from one country to another, where the climate differs; insufficient or improper food; lack of exercise; early weaning; sexual excitement; the excitement of dog-shows; parasitism; the irrational administration of purgatives, emetics, and vermifuges; debility arising from other diseases; and overcrowding, have all been cited as favouring the inroads of the infection and the development of the disease. The effect of fatiguing exercise on wet soils or in long, wet grass, or during wet weather, is well exemplified in the case of foxhounds and sporting dogs, in which distemper becomes very prevalent after the animals are subjected to such exercise.

MORBID ANATOMY.

The lesions vary according to the type of the disease and to the course it has run before death has supervened. Hence they may be few in number and confined to certain organs, or they may be multiple and widely spread throughout the body.

Carcass.—When the disease is of some standing, the carcass is generally emaciated or shrunken; it gives off a very offensive or nauseous odour, rigor mortis is but little marked, and decomposition tends to occur rapidly.

Skin.—The skin has a dull, dirty, and often a scabby or scaly appearance, and may be either dry or moist. It frequently shows eruptions or cicatrices, also hæmorrhagic, suppurative, or ulcerative lesions, confined to one or more regions or generalised over the body. Very often it is of a dark or purplish colour, and rarely a more or less yellowish or greenish tint is observed when the disease has been complicated by jaundice. Occasionally, on the prominent parts, such as the elbow, wrist, anterior angles of the ilium, and hock, it is more or less deeply ulcerated, showing suppuration, cicatrices, or gangrenous sloughs, brought about by continuous pressure on these prominent parts when lying extended on the side. Petechial spotting is rarely observed; roseola is frequent, also whitish or blackish circular or ovular cicatrices. The outer surfaces of the mouth, shoulders, and fore-limbs are generally soiled by saliva (in the case of fits), or by saliva mixed with blood (in stomatitis, etc.), or by nasal or ocular discharges, vomit, liquid food, or medicaments. The hair and skin of the hinder parts are frequently soiled by fæcal or bloody material from the bowels, or by urine. Sometimes there may be partial or complete loss of hair on the body. Occasionally the skin is affected with sarcoptic or follicular scabies, or by black pigmentation. Cutaneous lesions, however, are frequently absent.

Eyes.—The eyeballs are more or less sunken; frequently the cornea is of a smoky or opalescent appearance, and may be ulcerated or may show attempts at repair in the form of reddish or yellowish granulations. Occasionally the whole eyeball is the seat of a yellowish or greenish-yellow panophthalmitis, or is undergoing progressive atrophic changes. The conjunctival membrane has a dirty whitish, bluish, or leaden colour, and sometimes an infiltrated, swollen, or congested appearance, and as in the case of the cornea, it is soiled with a serous, mucous, muco-purulent, purulent, or often bloody purulent discharge. The free margins and the external surface of the eyelids may have a scalded, granular, or ulcerative appearance, and are frequently covered with more or less thickened, hard, greenish crusts or bloody purulent material. The skin of the face below the eyelids has often a scalded, depilated appearance, due to the irritating action of the ocular discharges. As in the case of cutaneous lesions, eye lesions are frequently absent.

Respiratory Organs.—The nostrils may show a more or less thick yellowish, yellowish-green, or reddish glutinous discharge, which is sometimes dry and adherent to the nose, or may completely occlude the nasal openings. The nose itself may be dry or moist, fissured or ulcerated. The pituitary membrane, extending from the nasal channels to the ethmoidal and frontal sinuses, may be of a pale leaden or purplish colour, swollen or infiltrated, injected and covered with a mucous, muco-purulent, purulent, or often a hæmorrhagic exudate, or even pure blood; sometimes small ulcers or abrasions or ecchymotic spots are present. These changes may occasionally be found extending to the larynx, trachea, and bronchial tubes; these structures are at other times extremely congested or swollen, or may show catarrhal changes, and they often contain a blood-stained, frothy material, or even pure blood or casts of lymph. Occasionally there may be a submucous suppurative laryngitis. The smaller bronchi are filled with a grey, dirty greyish, or bloody, thick, adherent exudate.

Lungs.—The lungs are occasionally dotted all over with minute hæmorrhages, which in other cases seem to have enlarged or coalesced, so as to give these organs the appearance of being more or less extensively congested. At other times the inferior third or inferior two-thirds of both lungs are congested uniformly, giving to the cut surface a diffuse, dark, mulberry-red colour, which becomes lighter in appearance after exposure for a short period to the air. In young animals, especially in poodle puppies, the lungs are very often enlarged, swollen, and in colour darker than usual; they feel soft and œdematous, containing a large amount of exudate in the interlobular areolar tissue. When the lung is divided and then squeezed, this exudate flows from the cut surface in a great quantity.

In the majority of instances, however, the inferior third or the inferior two-thirds of both lungs are consolidated, firm, and hard, in consistence, but the affected regions do not stand out above the surface of the sound portion of lung; in other cases this consolidation may be confined to a very few or to many isolated patches, varying in size from a grain of wheat, a pea, a bean, or a Spanish nut, to a walnut or something larger. The hepatised points or patches, which may be of a greyish-red or chocolate colour, have a tendency to coalesce and form larger patches; these, when cut into, are firm, resistant, and airless, and of a chocolate, brownish, or greenish colour in the centre; their bronchioles, when squeezed, exude a more or less thick, blood-stained, frothy, or greyish material. In a few instances about three-fourths of both lungs are consolidated, and seem to completely fill the chest; when divided, the cut surfaces seem to contract or cup towards the centre, and the hepatised parts are not always of a uniform colour, but show brownish, blackish, and reddish tints here and there. This distribution of colour is also not uniform, as the various tints frequently merge into one another by wedge-shaped or stalactitic processes; the cut surfaces show thrombi or hæmorrhagic infarcts. In other cases, although both lungs may be extensively involved and enlarged, on section they show cupping, and from the cut surfaces, which have a greyish, yellowish-pink, or yellowish appearance, a more or less thick pinkish or creamy, purulent-looking material exudes. In a few instances the red hepatised lung may show numerous yellowish points, varying in size from that of a millet seed, or something smaller, to that of a tare or a pea, scattered all through its substance. In other instances there may be a few vomicæ present, containing a greenish necrotic or a reddish or yellowish material.

The Pleura.—Beyond the pleural cavities containing a little serous or sero-fibrinous exudate, pleural lesions are rarely observed. Occasionally, however, in some epizoötics we may find a more or less extensive effusion of a serous, sero-fibrinous, greenish purulent or hæmorrhagic nature; it may be thin or thick in consistency, and of an amber, orange-red, greenish, or dirty reddish tint or colour, possessing very little odour; but at times it may be offensive or even putrid. Usually both cavities are involved, but occasionally the effusion is confined to one side. The pulmonary pleural membrane, more especially over the region of the underlying hepatised lung, may occasionally show a varying degree of roughness of surface; at other times there is a more or less organised coating of lymph, which may, however, be reticulated or give off processes, or it may even be soft and present a floccular appearance, and be loosely attached. The costal pleural membrane is very rarely affected, but one may occasionally find it very vascular and dull in appearance. It is quite exceptional to find the two surfaces adhering by a highly organized tissue.

Although lung lesions are very commonly found in distemper, they may be absent in some cases, especially gross alterations of the pulmonary tissue.

The *Bronchial Lymphatic Glands* are often swollen or infiltrated, hæmorrhagic or purulent.

The HEART sometimes shows small hæmorrhagic spots, or large, ill-defined ecchymotic, purplish, brownish, bronze-tinted, or yellowish patches, which often extend deeply into the muscular substance; at other times the lesions are somewhat circumscribed, and also extend into the myocardium. It has, however, often a boiled appearance. The *epicardium* and the inner surface of the pericardial sac, and the sac itself, may be affected in a similar manner to that of the pleura and the pleural cavities. The *subendocardium* in the vicinity of the columnæ carnæ may be ecchymotic, the lesions extending to the muscular tissue; petechial spotting is very rarely present. The heart, like most of the other organs, does not usually show very many, if any, naked-eye lesions, beyond the pericardial sac containing a small quantity of a serous or sero-fibrinous fluid; this is generally present even in acute cases that succumb early.

ALIMENTARY CANAL.—The *buccal mucous membrane* is sometimes the seat of simple or gangrenous ulcers, often with adherent sloughs; these lesions are generally found on the parts coming in contact with the teeth, especially the incisors, canines, and carnassial teeth, which are mostly covered with the brownish or reddish slimy material. The *tongue*, like the buccal mucous membrane, may be of a purplish or a dirty white colour; it is rarely ulcerated, especially on its lateral surfaces, but in very few instances the anterior free portion may be hard, swollen, blackish, brownish, or greenish and necrotic. The gums, especially round the necks of the teeth, are frequently spongy, hæmorrhagic, often undergoing necrotic changes, and thus exposing the underlying alveolar processes, the teeth in which are not rarely loose and commencing to fall out, particularly in young dogs about to naturally erupt their teeth. In a few instances the upper border of the alveolar processes and the teeth are turned outwards from the mouth. This is only seen in cases of chorea affecting the jaws in young dogs with soft bones. According to Fröhner, out of 70,000 distemper cases, 429 showed buccal lesions. The writer considers they are present in at least 10 per cent. of dogs affected, especially in young toy pomeranians and other small breeds.

The tonsils may be enlarged and prominent, of a red or purplish colour, and, in common with their surrounding tissues, they are often covered with an adherent membranous exudation, or may be undergoing an ulcerative or gangrenous process. The fauces, soft palate, and pharynx may be injected, swollen, or infiltrated, covered with a thick tenacious mucus, or may show ulcerations. The underlying submucous and areolar tissues may be swollen or infiltrated with a serous, hæmorrhagic, or purulent material. These lesions are usually found only in the malignant or hæmorrhagic type of the disease. The parotid, the retropharyngeal, the submaxillary, and more rarely the submaxillary glands, or the areolar tissue surrounding or intervening between the lobules of these glands, may occasionally be found swollen and undergoing suppuration. Abscess formation may occur, and when mature the abscess contains a hæmorrhagic, purulent material; the overlying skin is of a purplish colour, and is undergoing necrosis.

Suppurative changes may also be observed in the upper, middle, and inferior cervical glands.

Stomach and Intestines.—Lesions are sometimes met with in these organs. In the stomach the alterations occur chiefly in the pyloric region; the gastric mucosa is swollen and congested, and is covered by a thick glairy mucus.

The small intestine shows a reddish marbled, streaky, or mottled appearance of the mucous membrane, and is covered by a pinkish creamy material.

In the large intestine the mucous membrane may show a purplish redness distributed in lines running the entire length of the bowel, and is covered by a greenish, brownish, or blood-stained material, more or less mixed with fæcal material.

At other times the intestinal mucosa shows congestion which extends to the muscular coat of the bowel, and sometimes the lumen contains blood or sanious mucus. Hæmorrhagic spots and small ulcerations scattered here and there, or extending for the entire length of the canal, may be observed. The solitary glands and Peyer's patches are swollen, and appear as if sunken in the inflamed and swollen mucosa.

The terminal portion of the bowel may be intensely inflamed and swollen, and erosion of the anus may be present.

Intussusception is not uncommonly met with. According to the writer's experience, the variety of this lesion more commonly present is as follows: A portion of the small intestine passes into the large bowel, and appears outside the anus, simulating a simple *prolapsus recti*. At other times the cæco-colic portion of the large intestine becomes inverted, the prolapsed portion showing two orifices, one being the lumen of the cæcum, the other the ileo-cæcal opening; or a simple prolapse of the rectum may be present. These lesions are generally observed several days prior to the fatal termination. In cases of distemper that have gradually wasted away from inanition, the stomach and intestines are found in a contracted state.

The Mesenteric Glands are often enlarged, swollen, succulent, hæmorrhagic, or even purulent; abscesses may occasionally be observed.

The Peritoneum may show dulness or roughness of its surface; it is rarely congested. A moderate amount of amber or orange-red tinted serous fluid is generally found in the peritoneal cavity, and occasionally a quantity of brownish or greyish-brown liquid is present.

The PANCREAS is very often congested and swollen.

The LIVER may be swollen, congested, friable, and succulent, and when cut into the vessels contain an abundance of black tarry blood; at other times this organ has a palish or anæmic appearance, as if it had been partially boiled. It may contain abscesses. The *gall-bladder* generally contains its usual amount of bile, which is often found to be inspissated. The bile transuding through the coats of the gall-bladder stains the in-contact portion of the viscera its own colour. This is no doubt a post-mortem change. Jenner records that in cases where jaundice complicates distemper he has not observed "anything obstructing the biliary ducts." This agrees with the writer's experience, although he admits that he has not found hepatic complications common in the disease. Most authors imagine that the jaundice is due to catarrh of the biliary ducts.

The KIDNEYS are often congested, and their capsules occasionally show increased vascularity. It is well to note here that on making post-mortem examinations of aged dogs dead of distemper, chronic or degenerative changes of the valves of the heart and lesions of the liver and kidneys may be encountered, which have no connection with distemper.

In some cases the abdominal viscera may show no gross lesions.

The INGUINAL GLANDS are rarely the seat of suppuration. The *sheath* and *penis* are frequently covered with a yellowish, thick, purulent material.

The MUSCULAR TISSUES are generally wasted, of a pinkish-grey colour, soft, greasy, or soapy to the touch, and without lustre; but they often show a streaky or fibrillar or intersected appearance. Sometimes, however, in the case of an early death, the muscles are purplish. The fat is lustreless, dirty, soft, and greasy.

The BRAIN AND SPINAL CORD often appear normal even when nervous symptoms were present before death. In many cases, however, the external vessels are found dilated with black blood; the subarachnoid spaces, as well as the lateral ventricles, and rarely the central nervous canal, contain a serous fluid. In the case of the ventricles there is sometimes a considerable quantity of fluid present. At other times the meninges, brain, and spinal marrow have a dull appearance, the two latter being soft, and the convolutions of the brain often losing their well-defined contour.

In addition to the lesions of various organs mentioned, a variety of histological changes, as well as microbes, may be found, but further research in this direction is required before any definite pronouncement as to their nature can be made.

In many acute cases nothing is discernible after death beyond, perhaps, a little serous fluid in the pericardial sac. Occasionally, when the lungs are affected with an acute or croupous type of pneumonia, the case may be complicated by a submucous suppurative laryngitis, or with suppurative foci in other parts, notably the liver, submaxillary, retropharyngeal, cervical, or other glands in various parts of the system.

The Blood is often diminished in quantity, and paler than usual; but when septicæmia complicates distemper it may be black, tarry, and not inclined to coagulate. When this latter condition is present, there are often more or less extensive extravasations in various organs.

In many cases after death the swollen and vascular conditions of various lesions which were present during life disappear.

PERIOD OF INCUBATION.

This varies according to the method of infection, the quality, age, and quantity of the virus received, the age and susceptibility of the animal, the season or state of the weather, and the surroundings.

When the active virus is injected into the vein, muscle, or one of the visceral cavities, symptoms may, as shown by Carré, be induced in two, three, or four days. If the crude nasal discharge be smeared on the nostrils of a susceptible dog, the disease appears in from four to fourteen days. The crude inoculation into the skin produces the malady in four to seven days. When merely kept in contact with diseased dogs, the in-contact animals develop symptoms within one to three weeks.

Krajewski, Mégnin, Laosson, and Babes and Starcovici, compute the period of incubation at 4 to 7 days; Venuta, 4 to 6 days; Trasbot, 5 to 8 days; St.-Yves Ménard, 8 to 12 days; Sewell, 4 to 14 days; Nocard and Leclainche, 12 to 15 days; and Hobday, 3 to 4 days to 3 weeks. Jenner states that evidences of illness appear during the second week after exposure to infection.

Fear, excitement, fatigue, railway travelling, wet or changeable weather, washing, weaning, dentition, the injudicious use of aperient or worm medicines, operations, anæsthetics, loss of blood, sexual excitement, parturition, and other debilitating causes, may so lower the system that the disease manifests itself in a shorter period than when the animal is in a robust and otherwise good state of health.

SYMPTOMS.

This disease is frequently insidious in its invasion and multiform in its manifestation. It occasionally runs a rapidly acute course, but often this is deceptive, as what is taken to be the naturally acute course is in reality only a later phase of the malady, the initial or milder symptoms being not rarely overlooked or misunderstood. Thus the disease may be ushered in with a slight but temporary elevation of temperature, some unaccountable dulness, a capricious appetite, a staring coat, desire for warmth, rigors, a cutaneous vesicular eruption, a watery eye, and perhaps a slight diarrhœa, vomiting, or convulsions. These may pass away for a week or two, or even longer, until some sudden outburst of fresh phenomena is manifested.

As the symptoms vary so, not only in one particular outbreak, but also during different seasons or epochs, it is advisable for convenience of description to describe them under the following headings :

1. *The cutaneous or eruptive.*
2. *The ocular and nasal.*
3. *The respiratory*, including the laryngeal, tracheal, bronchial, pulmonary, and pleural.
4. *The alimentary*, including the oral, pharyngeal, gastric, and intestinal.
5. *The nervous.*
6. *The suppurative or phlegmonous.*
7. *The infantile.*
8. *The malignant.*
9. *The hepatic.*
10. *The urino-genital.*
11. *The muscular.*
12. *The febrile.*

It must, however, be understood that two or more of these groups of symptoms are generally present in the same animal during the course of the malady, also that it is rare for any two cases to show exactly the same manifestations.

1. Cutaneous or Eruptive Symptoms.—This manifestation of distemper has, until quite recently, been generally overlooked, or, if observed, its significance was not usually understood. It was, however, pointed out by Louis Barrier in 1793, who, nevertheless, mistook it for variola of the dog ; Eric Viborg in 1795, and Edward Jenner, both of whom considered distemper to be a variolous disease ; Urbain Leblanc in 1841, who described it as canine variola ; Delabere P. Blaine in 1817 ; William

Youatt in 1845, who did not connect it with distemper, but described it as a separate disease under the denomination of "dog-pox"; Mayhew in 1851; Hertwig in 1853; Camille Leblanc in 1857; Trasbot during 1868-1903; Mégnin in 1896, under the name of "gourme," which he considers quite distinct from the malignant catarrhal fever of the dog or the English distemper, and "la grippe," or infectious pneumonia of kennels, or malignant typhus of dog-shows. Nocard and Leclainche, Friedberger and Fröhner, Lignières, Phisalix, Cadéac, Law, Carré, and the writer, among others too numerous to mention, also recognised the importance of this symptom. Woodroffe Hill and Sewell casually refer to it.

Sometimes it is the sole external manifestation of the disease, and at certain periods it predominates over the other manifestations, leading one almost to conclude it was a special affection, and not connected with distemper. In the majority of cases of distemper the eruption occurs to a varying degree or extent. When it predominates over all the other symptoms, it is generally observed in young dogs during hot weather, or when the animals have not been exposed to chills or to inclement weather. However, the writer has observed it in adult and even aged dogs, following a high internal temperature, the latter declining as soon as the eruption has appeared. When the patient is kept under good hygienic conditions, the eruption may not be succeeded by any other symptom or complication. It is not known if this eruption may arise from any other infection than that of distemper, or occur as a disease, *per se*; but it may be induced experimentally by inoculating a dog with the filtrable virus of foot and mouth disease, even though the animal may previously have had distemper; also by inoculating with the bacillus of Mathis, and with the sero-purulent material from a vesico-pustule of a distempered dog. Trasbot looks upon it as the essential lesion of distemper, all the other manifestations being secondary or foreign to it.

The advent of the eruption is usually preceded by a slight dulness, perhaps a little loss of appetite, or occasionally a slight attack of convulsions, and probably always with an ephemeral elevation of temperature, which in uncomplicated cases recedes as soon as the eruption appears. It is, however, frequently overlooked or not observed in consequence of the practitioner not, as a rule, being called in until the disease is far advanced, when the eruption has by that time generally vanished. Still, on searching carefully for evidences of the eruption, one may often find traces, such as dark or dotted areolæ, shreds of blackish or brownish scabs, brownish or blackish pigment spots, white cicatrices, etc., all indicating that it has been present in the case.

The *skin lesion* is a vesicular pustule containing a quantity of amber-tinted serous fluid, which gives the vesicle a bladdery or pock appearance. It varies in size from a pin's head to the diameter of a split pea or bean, or to that of the small finger-nail. In shape it is sometimes circular, but generally the outline is ovular. The surface is smooth, convex, and usually glistening, and the summit is always flattened, but never conical or umbilicated. It is often surrounded by an areola of a pinkish or purplish colour, the remains of the primary circumscribed spot of subepidermic congestion. The eruption usually starts in the form of minute red or purple spots, resembling those due to flea-bites; but each spot is of a uniform pinkish or purplish colour, without a darker centre spot. At the end of twenty-four hours each spot appears as a small nodule, slightly raised above the surrounding epidermis; it increases in height, and gradually fades into a clear amber-tinted vesicle. Its maximum size is attained on the third or fourth day, when it ruptures and the contents escape. The latter, in combination with the thin superposed layer of epidermis, dry up and form a thin scab, which generally becomes displaced, a small moist, slightly granular surface being left, which gradually fades away by the fifth or sixth day. On pigmented skins a clear or even blackish circular spot or cicatrix often remains for some time after all other traces of the eruption have disappeared. Occasionally small abraded surfaces remain, and for some time discharge a greasy thin material, which usually decomposes and gives off a very nauseous odour. The eruption is rarely pustular in character; it usually involves the deeper layers of the epidermis. The surface of the dermis being vascular in the dog, confines the lesion to the parts mentioned. Occasionally, however, the eruption becomes pustular, involving the dermis, and leaving permanent, indelible cicatrices, which have a glistening surface and remain bald for life. This condition has been now and again observed by the writer. In other instances minute vesicles occur, which exude a yellowish serous material. This dries up, and gives the skin an appearance as if a dry adherent material containing sulphur, or the yolk of an egg, had been applied to it.

The eruption varies as regards the number of the vesico-pustules present. In some instances from one to three or more are observed, and occasionally they are spread all over the body, even invading the auditory cavities and the nasal, buccal, bronchial, and anal mucous membranes.

The usual seats of the eruption, however, are the almost hairless skin of the abdomen, inside of the thighs, and sometimes inside the arms, and not uncommonly on the scrotum, perineal region, the inside of the flap of the ear, and in the female on the labia of the vulva. Renewed crops of the eruption may be observed in some cases, and while one set is disappearing, another is appearing, this process continuing until convalescence is reached. In the majority of instances the eruption develops in from twenty-four hours to three or seven days, and then completely disappears. But there are cases not rarely met with in which cutaneous symptoms are observed for the first time during the stage of convalescence.

When the vesico-pustules are few in number, they are generally discrete; but when very numerous, they may coalesce and form a large oozing surface, which gives off a thick exudate that mats the hairs together. At other times extensive ulcerating surfaces are left on various parts of the body, which become encrusted with scabs and emit an offensive odour.

Pruritus is generally absent in cases where the eruption is confined to the usual seats of its election ; but when the eruption becomes generalised, intense pruritus is observed, the animal constantly scratching the affected parts to such an extent that the case may be mistaken for one of eczema or sarcoptic mange.

Exposure to chill, inclement weather, or any other adverse influences during the time that the eruption is likely to appear, may cause it to be suppressed or to become aborted.

Under such conditions it is not long before some complication, such as bronchitis, broncho-pneumonia, gastro-enteritis, or nervous symptoms, appears. Again, if the generalised eruption be mistaken for scabies or some other skin diseases, and treated as such, pneumonia, gastritis, or some other complication generally supervenes, which ends in a serious illness or even death. Furthermore, if it be mistaken—as it often is—for a skin disease due to worms, and treated by areca-nut or some such drastic vermifuge, gastro-enteritis or dysentery may follow, and become protracted, or it is not uncommon for death to occur within a few hours.

When the eruption appears early in the complaint, and is unaccompanied by any serious complication, it is usually a favourable sign, and provided the animal be treated rationally, recovery generally results. If, on the contrary, it appears late in the attack, when the animal has become prostrate from a serious complication, it is rather a grave prognostic—at least, one should, as a rule, regard it as such.

In very malignant outbreaks, sometimes the whole of the skin has an abraded appearance, and discharges a profuse serous material, which is often so abundant that it wets the bed or ground on which the animal lies, and gives one the idea that the animal is sweating freely. The secretion has a very nauseous odour, and is usually the forerunner of death.

At times, towards the crisis of distemper, myriads of small bloody abscesses, varying in size from a hemp-seed to a fowl's egg, and resembling somewhat follicular mange, may appear. They frequently coalesce, and when they have ruptured or been surgically treated, they give the skin a lacerated appearance. They occur chiefly under the jaws and on the limbs, but often the whole body becomes involved.

There is another form of skin disease that seems peculiar to distemper. The skin becomes dry, hard, thick, and scaly, and after a time pigmented (black or purple pigmentation) ; the hair falls off ; there is no pruritus ; it does not appear contagious ; is liable to recur ; all the dogs in a litter may have it at the same time ; the sebaceous follicles do not contain the demodex ; and the disease is curable if treatment be persisted with.

Urticaria may arise during the course of distemper, as also may eczema and other types of skin disease. Follicular mange seems to be piloted into existence by distemper, and sarcoptic mange, or ringworm, is often favoured by it. The writer considers that follicular mange does not arise as a disease *per se*, but only follows on some other malady, more especially distemper. The external and middle ear may become the seat of catarrh or of a purulent process, which gives rise to the usual symptoms of such conditions.

2. OCULAR AND NASAL SYMPTOMS.—A catarrhal discharge from the eyes and nostrils is generally characteristic of distemper, but it may be absent, even during the entire course of the malady. Very frequently the nostrils alone are affected ; at other times only the eyes. The discharge is generally preceded by a serous or watery flow from the nostrils or eyes, or from both. It is then ordinarily followed by the appearance of a clear, thick mucus, which afterwards becomes tinged with a yellowish purulent material, and in very severe cases, or in advanced stages, with streaks, strings, or clots of blood. The conjunctival as well as the Schneiderian membrane is at first very red, congested, and afterwards often swollen. Frequently, however, these mucosæ are neither congested nor swollen, but have a dirty white appearance, sometimes tinged with purplish venules. In such instances the discharge is always serous for a short period, and then vanishes. In the majority of the smaller dogs the nasal or conjunctival discharge does not often become muco-purulent, purulent, or hæmorrhagic ; in them it is usually serous. In a few uncomplicated cases the nasal discharge is composed chiefly of a creamy laudable pus, which is, as a rule, a favourable sign. During certain enzoötics of the disease the conjunctival membrane is so swollen that it becomes

everted, and hides the greater portion of the eyeball and eyelids from view. It is then of a deep pinkish hue, and secretes a profuse amount of creamy pus ("pink-eye form"). In a large number of instances the discharge is of a sero-fibrinous nature; it becomes dry, and adheres tenaciously to the internal angles of the eyes, where it appears as a greenish resinous and hard flake or speck. At other times it is composed chiefly of a thick sticky mucus and pus, and when profuse it dries up, becomes hard, and glues the eyelids together, especially at night-time. It plugs up the nostrils, and is with difficulty removed therefrom, leaving after a time an excoriation of the underlying membranes, which become granular and often bleed on the slightest touch or friction. Sometimes only one eye is affected, but usually both are involved. In a large number of cases the catarrhal condition of the eyes and nostrils does not appear before the seventh or eighth day. Sometimes this is delayed for two or three weeks, occasionally later; but in a few instances, especially in very acute cases, it may be present on the third or fourth day following the initial symptoms.

When the eyelids become affected, the sight is obscured, or photophobia is generally present; the animal frequently rubs the eyes with his paws or against some obstacle. When the nostrils are the seat of the trouble, the animal shows difficulty in breathing, and in order to remove the obstacle, sneezes, snorts, or snuffles, and occasionally rubs his nostrils with his feet or along the ground. When the posterior nares contain a large amount of mucus or muco-purulent material while the nostrils are free, the animal, in an attempt to dislodge the obstruction, takes repeated deep nasal inspirations and expirations, and in doing so makes a harsh or coarse naso-guttural sound. Frequently the tears are of an irritating character, so that the skin of the naso-maxillary region below the eyelids becomes excoriated, and the hairs fall out, leaving a pinkish granular, moist, or ulcerating surface. Sometimes the discharges from the eyes disappear, to reappear at some future date. The nose often becomes dry, cracked, and ulcerated.

The catarrhal process may not only attack the conjunctival membrane, but also extends to the cornea, and even the deeper structures of the eye. As a rule, when it extends to the cornea, it is in the form of a minute vesicular eruption, which ends in ulceration of one, two, or more spots, usually in the centre or a little above the centre of this structure. In some instances one ulcer appears above the other, or they may be grouped together in a small patch. This eruption is generally preceded by photophobia, twitching or blinking of the eyelids (which are usually closed, or partially so), profuse lachrymation, and accompanied with pain. After a time, this eruption breaks down, and leaves a small ulcer or ulcers, which subsequently becomes opaque. The surrounding corneal tissue becomes greyish or bluish-white in appearance, and eventually minute blood-vessels spread out in a fascicular or fanlike network, or may fringe the circumference of the sclero-corneal junction, and may ultimately extend to the ulcers, which then usually become reddish and granular. At times these ulcers do not become opaque, and do not give rise to any inflammatory reaction ; they are disinclined to heal, and leave the surrounding corneal membrane for a variable period normal in appearance.

The ulcers may attack one or both eyes ; they vary in depth as well as in circumference, and may be no larger than a pin's head, or may attain the circumference of a split pea. In some cases their depth may be very shallow, only extending to the superficial layer of the cornea ; in others they extend deeply, involving two or more layers, even to perforating the whole thickness, or ultimately the membrane of Descemet. In this last case the result is that some of the aqueous fluid contents of the anterior chamber escapes, and the free edge of the iris protrudes into the breach thus formed. The latter process prevents any further escape of fluid, and forms a pea- or grape-like prominence or projection on the front of the eye (*staphyloma*). Or the ulcer, although deep-seated, may not extend through the whole thickness of the corneal membrane ; it stops short at the membrane of Descemet. The latter, by losing its anterior support, is pressed through the ulcer in consequence of the intra-ocular pressure of the fluids, and forms, as in the perforative ulcer, a pea- or grape-like projection, constituting a *keratocele*, which varies in size according to the circumference of the ulcer. As in the deep perforating ulcer, the free edge of the iris may be pressed forward, and become included in the corneal gap. At other times the cornea does not undergo any ulcerative process, but becomes the seat of a more or less diffuse bluish or opaque *parenchymatous keratitis*, involving one or both eyes, without the presence of any conjunctivitis. Again, this form of keratitis may produce a deformity of the cornea, which in some instances appears flattened in one direction and prominent in another ; while on other occasions it is simply conical, or rarely faceted. This, as well as the ordinary bluish or opaque parenchymatous keratitis, is often accompanied with *iritis* or *iridocyclitis*. Uebele* has endeavoured to show that this form of keratitis is due to the action of toxins on the circulation. Occasionally one eye in cases of distemper may become the seat of a very acute primary destructive *panophthalmitis*, which usually appears and runs its course within twenty-four to thirty-six hours; at other times this is secondary to an infective perforating ulcer.

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* Uebele, "Keratitis Parenchymatosa beim Hunde," thesis for doctorate. Giessen, 1900.

As to the causation of the ulcerative process of the cornea in distemper, some have attributed it to the action of accumulated and decomposing pus, or to irritation or injury due to friction with the paws; also to malassimilation. But all these alleged causes are frequently present without any ocular lesions resulting, and very often corneal ulceration occurs in the absence of such factors. These theories are, like the "sugar" theory, untenable, the writer having seen kennels of dogs, such as pugs, toy spaniels, etc., affected by an enzoötic of ulcerative keratitis, and these animals were never given sugar in their lives. The "prominent eye" theory is also untenable, as those breeds with well-covered eyes very frequently become affected.

This ulceration of the cornea, with or without catarrhal conjunctivitis, may frequently be the only symptom present in distemper, and often appears in this form as an enzoötic affecting the majority, if not all, of the inmates of a kennel, especially in the case of pugs, Pekingese and Japanese spaniels.

The uncomplicated superficial ulcerative process, provided no heroic or caustic treatment be adopted, usually runs its course and ends in absolute recovery, without a trace of cicatrix remaining. The deeper ulcerative process always leaves a cicatricial opacity, which varies according to the circumference and depth of the ulcer and the treatment adopted. Frequently it has a narrow section of the iris adhering to the posterior limiting layer of the cornea behind the ulcer (anterior synechia). In *staphyloma* the iris is always included in the lesion. In *keratocele* the iris may or may not be adherent to the membrane of Descemet. *Black pigmentation* often results from ulceration of the cornea, as it may from any other superficial corneal disease. Cataract, amaurosis, optic neuritis, nystagmus, etc., may remain as sequelæ.

Not rarely, after all the active symptoms of distemper have passed away and convalescence has become established, and recovery taken place, *a chronic purulent conjunctival discharge remains*. This has been regarded by some as similar to the gonorrhœal or blenorrhagic conjunctivitis of man, but such a view is erroneous, as all attempts to transmit gonorrhœa from man to dog have failed, and dogs themselves do not suffer from any form of gonorrhœa. Balanitis, which is very often present in distemper, has been, and is, often mistaken for it.

In the examination of dogs at shows especial attention should be paid to the examination of the conjunctival sacs by everting the conjunctival membrane, which in health is pinkish and glistening, and in distemper is often of a deep pinkish velvety hue, and the conjunctival sacs contain flakes or strings of yellowish muco-pus; the nostrils also should receive attention; in health they are pinkish and moist, and do not contain any droplets of serous fluid. In distemper, however, the nasal channels are often dry, and contain a little adherent dark greenish or brownish hard, or sometimes soft, sticky, fine, or stringy mucus. The exhibitor often wipes away the discharge adhering to the outside of the eyelids and nostrils just before presenting the dog for veterinary examination.

3. Respiratory Symptoms.—In the majority of instances the respiratory tract, and especially the lungs, become involved in an obscure manner, chiefly in the form of broncho-pneumonia. This complication accounts for the greater part of the mortality, and is often succeeded by disturbance of the nervous system. Indeed, disturbance of the nervous system in the form of meningeal symptoms or convulsions frequently masks the pneumonic process. Hence the presence of the pulmonary affection is often overlooked, as neither disturbance of respiration nor cough may be observed, and lesions of the lungs may only be discovered for the first time at the autopsy.

One of the earliest noticeable or pronounced symptoms of distemper is a *cough*, which varies in character according to the part or extent of the respiratory tract involved in its production. When it proceeds from laryngeal, tracheal, and bronchial catarrh, it is at first harsh, hoarse, or husky and dry, but later on it becomes moist, and at the end of each fit or paroxysm of coughing, muco-catarrhal or muco-purulent, or rarely bloody sputum is expectorated into the mouth and then swallowed, while on rare occasions it is ejected from the mouth on to the ground. On inspection of the larynx through the mouth, this organ is found to be heightened in colour or injected, often swollen, and covered here and there with flakes of yellowish muco-purulent material. On pressure being applied to the outside of the larynx, or even to the trachea, cough is usually and easily induced; swelling is observed on palpation of the external laryngeal region. In short-haired dogs this swelling, when extensive, is very noticeable by simple inspection.

When the trachea and larger bronchial tubes are involved in the catarrhal process, besides the hoarse or husky paroxysmal cough, there may be an increased frequency of the respiratory and abdominal movements; but, as a rule, dyspnœa is not a well-marked feature. When the hand is placed on the chest, rhonchial fremitus may be felt over a large area. The percussion sound is exaggerated from over-distension of the lungs; the latter are not much altered or deficient in resonance. On auscultation, the respiratory sounds are loud when the tubes are free; but later on, when they become plugged by secretion, the sounds are often feeble, or may be totally suppressed. The rhonchi are dry in the early stages, but moist râles are more or less heard over a large portion of the lung when once secretion sets in. When the bronchial tubes become filled with a secretion that is not viscid, sounds of a rattling character and various rhonchi may be heard over various areas at the same time, according to the seat and stage of the bronchitis. This localisation of the complication of distemper is not, as a rule, very serious, but it may be accompanied by malaise or dulness, with a slight diminution of appetite, a slight elevation of temperature, perhaps a little diarrhœa, and occasionally vomition during the paroxysm of coughing. Milder cases show very little general disturbance; there may be a slight frequency of the pulse, or a rise in temperature, and no interference with the appetite.

In young dogs, exposed to inclement weather, wet, or fatigue, *capillary bronchitis*, involving the bronchioles, frequently succeeds the laryngeal, tracheal, and bronchial catarrh; but usually it occurs independently. It is a very serious complication, and is the cause of a high mortality. The dyspnœa is well marked, the respirations are rapid, or there is paroxysmal orthopnœa. The cough is continuous, often violent and most distressing; expectoration is difficult. In very severe cases the temperature is high and the pulse frequent. When the case takes an unfavourable turn, the visible mucous membranes become purplish, the temperature falls, the pulse is small and rapid, the extremities are cold, delirium or coma supervenes, the respirations are short and catchy, and the animal dies from asphyxia, or from the presence of fibrinous clots in the heart and large bloodvessels.

On auscultation, submucous and subcrepitant râles accompanying inspiration and expiration are heard with facility, especially towards a large part of the inferior two-thirds of the lungs.

Occasionally in young delicate puppies, such as poodles, when about three months of age, the lungs become the seat of *œdema*, which is evidenced by extreme dyspnœa, often amounting to orthopnœa, and a troublesome retching variety of cough, with a somewhat difficult and abundant frothy serous expectoration. On percussion, a deadened sound or note is found on both sides of the chest. On auscultation, the respiratory murmur is weakened or absent, and fine bubbling crepitations are heard. Extreme weakness, rapid emaciation and restlessness, are usually present, and are generally followed by death.

In the majority of complicated cases of distemper, *broncho-pneumonia* is present, but it is impossible to determine the period at which it starts, as often the animal dies after showing convulsions, meningeal symptoms, or coma, without manifesting any symptoms which would lead the practitioner to examine the chest. In a large number of instances there is a frequent, short, hacking cough, accelerated respirations, and a slight elevation of temperature—symptoms which are sometimes ushered in by shivering, convulsions, or vomition. Usually, when marked acceleration of the respirations or dyspnœa is present, a high temperature is observed. At times the breathing becomes more rapid, or the dyspnœa much more marked, than at others, and occasionally the latter is paroxysmal in character. When the lung lesion follows a pre-existing bronchitis, there is an increased elevation of temperature immediately preceding it, but there is seldom a sudden rise such as occurs in the croupous type of pneumonia; nor does the fever run a definite course, as in the latter affection. The daily variation of remissions or exacerbations of temperature is often considerable or irregular; sometimes the fever is higher in the morning than at night. The cough, being paroxysmal in capillary bronchitis, gradually becomes less so when broncho-pneumonia supervenes. The pulse is greatly increased in frequency, and as time goes on it becomes soft, small, and feeble. Vomition is often observed during coughing.

As the disease progresses, the breathing becomes rapid and superficial; the dyspnœa is so marked that the abdominal movements are of a "pumping" character, the cheeks puff out at each expiration, and the strength fails; the visible mucous membranes become of a purplish colour; there is extreme restlessness or apathy, or a semicomatose condition may occur, which is from time to time interrupted by an ineffectual or muffled cough. With rapid failure of strength and extreme emaciation, the cough may almost cease; cyanosis is present, and the animal dies from exhaustion, or merges into a more or less comatose condition, death sometimes occurring during a fit of coughing or of convulsions.

In those cases which do not show marked features of capillary bronchitis, the symptoms are usually less severe, the elevation of the temperature less marked, and the onset more gradual; the pneumonic process supervenes later, and the course of the disease becomes more protracted. A slight and pronounced irregularity of the temperature, and an increasing difficulty of breathing, with emaciation and loss of strength, are very noticeable in young animals, but in the older ones the general disturbance is less and the disease is more latent, with some elevation of temperature, a slight cough, dyspnœa, and muscular weakness. Death may take place from the tenth to the twenty-first day, but in very young animals it may occur earlier, from disturbance of the respiratory function, which disturbance is more marked than in the croupous type of pneumonia. This interference with the respiratory function is due to diminution of the respiratory area, in consequence of the consolidation and collapse of the lung tissue, and also to the impediment to the entrance of air caused by swelling of the bronchial mucous membrane and accumulation of secretions in the smaller bronchial tubes ; over-distension is produced, and failure of the right heart occurs, as in the croupous type of pneumonia. However, in many instances death cannot be attributed to this cause, as on post-mortem examination the pulmonary area affected is very slight. It is quite possible in such cases that the toxins acting on the nervous system bring about the fatal termination.

When the case does not terminate in death, the temperature gradually descends for several days, and occasionally weeks are occupied in its defervescence. Frequent interruptions, with exacerbations and remissions of temperature, cough, and dyspnœa, are observed, and the appetite slowly returns ; but recovery is protracted in consequence of the bronchial symptoms. Many of such cases seem to suddenly improve, but may be soon followed by convulsions, coma, delirium, chorea, or paralysis.

The physical signs are similar to those of bronchitis. Rhonchi and moist râles are heard over both sides of the chest. There is not much alteration in the percussion resonance throughout the course of this complication. The chest is flattened, and falls in. The involvement of the lung is indicated by the increase of temperature and dyspnœa rather than by evidences of physical changes. There is great difficulty in detecting the consolidation of limited areas in the early stage, as these are surrounded by normal and emphysematous lung, so that resonance is not much impaired. In the larger coalesced areas, which may occupy quite one-half or more of the inferior part of both lungs, the impaired percussion resonance is easily detected. On auscultation, the moist bronchitic râles take on rather a metallic sound ; they become finer, but are not so fine as the crepitations present in the croupous type, and they are more superficial. The presence of superficial, rather fine, moist râles over small areas during inspiration, and not removed by cough, is often the only physical sign present, and in consequence it affords valuable information. When larger areas are consolidated, there may be tubular breathing and some disturbance of resonance on percussion.

Occasionally the lungs take on the *croupous or lobar type of pneumonia*, which is more often seen in adults than in young dogs. It is sudden in its invasion, and often quiet in its course. There may be some dulness, loss of appetite, shivering, and a high temperature.

The cough is short, hacking, and frequent, but rarely paroxysmal ;

the expression is anxious and heavy, but not pinched, as in pleurisy; the breathing becomes shallow and hurried; there is great thirst, extreme prostration, quick, full pulse, and, when pleurisy is present, moaning on palpation or on percussion. Towards the crisis there is expectoration; the pulse, although full and strong, becomes irregular, small, and compressible. The temperature obtains its maximum, and when the crisis is reached it falls rapidly. But if an unfavourable course is being pursued, the animal becomes restless; there is great dulness, followed by coma, delirium, or convulsions. Sometimes, after very grave symptoms have been present, such as total loss of appetite, a pumping action of the abdominal muscles, and a puffing out of the cheeks during expiration, the animal suddenly improves, but after a short time symptoms of cerebral intoxication or infection, such as convulsions, meningitis, or coma, usually supervene. Death then follows after a brief period.

Frequently, however, the respirations are not much disturbed; the animal occasionally pants, there is no cough, and death takes place from failure of the right heart. The temperature at the commencement of this complication rapidly rises, and goes on increasing for a few days; then it is maintained at a certain height until the crisis is reached, and after this it somewhat suddenly falls, and then gradually returns to the normal in a short time.

The respiratory sounds are usually weak, harsh, and not distant, although occasionally during the earliest stages they are harsher and louder than normal; but as the disease advances they become bronchial in character, superficial, high-pitched, and metallic, and at times blowing. During the congestive stage there are fine crepitations, consisting of a number of fine, dry, crackling sounds, following one another in rapid succession, and resembling those sounds produced by rubbing the hair between the fingers close to the ear. They are heard during inspiration, are intensified by coughing, and persist until consolidation is complete. They reappear as soon as resolution sets in, when the bronchial breathing (blowing or tubular breathing) loses its metallic sound. Percussion during the early stage is not pronounced, but as the consolidation process progresses, the sounds become dull over the hepatised portions, and tympanitic over the area of normal lung tissue.

When *pleurisy* is associated with pneumonic distemper, there is a moaning or groaning on the part of the animal when the chest is percussed or manipulated, and this is often observed during inspiratory efforts. This symptom may disappear as soon as effusion takes place; the dyspnœa then becomes increased, and the animal rapidly wastes away and sinks from exhaustion.

In the short-nosed dogs it is generally difficult or impossible to auscultate the respiratory tract, in consequence of the proper sounds being masked by the guttural or naso-guttural sounds.

Gangrene of the lung is rarely observed in the course of the pneumonia of distemper. Its presence is denoted by the characteristic odour of the breath, which is intensified when the animal coughs.

In many outbreaks the majority of the dogs die during the early diffuse congestive stage of pneumonia, which may be accompanied by hæmorrhage from the lungs and from the nostrils, death usually taking place in such cases within twenty-four to forty-eight hours. In others a septic or purulent pleuro-pneumonia occurs, associated with eruptions or even bloody abscesses scattered all over the body.

4. Alimentary Symptoms.—Lesions generally occur in one or more parts of the alimentary canal during the course of distemper. In the majority of instances the intestinal canal becomes the seat of catarrh, which is evidenced by a more or less profuse and persistent diarrhœa, sometimes associated with tenesmus and the passage of bloody fæcal material. Usually the diarrhœa is preceded by a passive constipation. The latter is, however, due partly to the loss of appetite, with the result that little or no food is ingested: hence there is not sufficient fæcal material formed to act as a stimulus to the colon or rectum to empty their contents; and partly to the depression of the nervous system, in consequence of the action of the toxins and of the high temperature thereon. Very often this intestinal catarrh becomes aggravated by improper food, or by the irrational employment of drastic purgatives, or from exposure to chills during the course of the affection. Under such influences the inflammatory process extends to the submucous coat, and produces a swelling of the mucous membrane, which is then easily abraded. Small ulcerations and hæmorrhages are thus induced, and are evidenced by the frequent passage of fluid sanguineous stools containing mucus, and associated with tenesmus. In such cases the animal shows rapid emaciation, and arches his back; the abdomen is retracted, the thirst becomes excessive, but the appetite is suppressed. He shivers very much, and evinces a great desire for warmth. Should the profuse diarrhœa (which no medicine seems to check) spontaneously cease in an abrupt manner, and the appetite, hitherto in abeyance, become ravenous, it is generally a bad omen, and is frequently followed by convulsions, coma, chorea, meningitis, etc. Another very grave sign is when rapid emaciation occurs and the fæces change from a light colour to a leaden or greenish tint, become very fœtid, and gradually assume a mucous and hæmorrhagic character. On the other hand, although the diarrhœa may be profuse, it is not to

be regarded as a serious symptom, provided it yields to treatment and is not accompanied by marked emaciation.

A very serious complication of the catarrhal enteritis of distemper is prolapse of the rectum or colon, rendered still more grave by the presence of an invagination of the small intestine, and accompanied by protrusion of the invaginated portion from the anus. If proper surgical measures be adopted immediately, this condition is not necessarily a fatal one. Even without attention the patient may live for several days, or even a week or so. This has occurred several times as an enzoötic in large kennels of Great Dane and other large breeds of puppies when between two and three months old. Here we may remark that in some cases of distemper the only symptoms observed—at least, for several weeks—may be persistent diarrhœa, an ephemeral slight elevation of temperature, and a brownish or slimy condition of the teeth ; then nervous symptoms may develop, such as chorea or paralysis.

In old dogs a persistent or chronic diarrhœa or dysentery may be the only symptoms present in abortive cases of distemper. Occasionally the stomach may be implicated in the catarrhal process, either *per se* or in conjunction with the intestine. The symptoms usually observed are shivering, marked thirst, persistent vomiting of a thick, glairy, viscid fluid, which after a time may become altered to a thin serous character, and is often tinged with bile or with blood. All food or medicine forced on the animal is quickly rejected. A fatal termination may be expected when coldness of the surface of the body, rapid emaciation, a subnormal temperature, sunken eyes, and the vomition of a putrid brownish or chocolate-coloured material, are prominent symptoms.

The *oral* symptoms arising during the course of distemper are by no means uncommon. They have been noted by Blaine, Youatt, Funke, Duttenhofer, Mayhew, Hering, Anacker, Friedberger, Hertwig, Krajewski, Konhäuser, Semmer, Arnous, Harlimann, Nocard and Leclainche, Cadéac, Law, and the writer. These oral lesions are well known to dog-fanciers, who term them "mouth distemper." The phenomena may be observed in large outbreaks of the disease, and also in isolated cases, especially during certain years. They are more frequently met with in toy dogs and young puppies than in the larger breeds and adults, and are evidenced by a brownish or rusty slime on the teeth, a hæmorrhagic condition of the gums around the necks of the teeth, and buccal ulcerations of various degrees, extending even to gangrene, especially opposite the canine, carnassial, molar, and incisor teeth. In young dogs just about to shed the temporary teeth, the latter are very liable to be evulsed by the gangrenous process. The tongue may be involved in rare instances by a similar ulcerative process, especially at its anterior or free portion. A flow of dirty sanious or putrid saliva from the angles of the mouth is often associated with the above conditions. Frequently an herpetic eruption appears on the outside of the lips, especially at the angles of the mouth. Occasionally the gangrenous ulcerative process of the buccal membrane extends to the overlying muscular and cutaneous structures, especially at the angles of the mouth, leaving large gaps or holes in the cheeks, and thus exposing the teeth.

The *tonsils* occasionally become prominent and swollen, and are of a bright red or strawberry colour. They may vary in size from a small French bean up to a Spanish nut or a walnut, and may attain such dimensions as to almost occlude the lumen of the fauces. This symptom is one that is often overlooked by the practitioner.

The soft palate, fauces, and pharynx are frequently injected, swollen, and often nodulated, in consequence of the swelling of their follicles. A thick ropy mucus or muco-purulent material, sometimes tinged with blood, is secreted by their mucous membranes. The symptoms induced are cough, of a husky or croupous character, sometimes accompanied by the expectoration on to the ground of an abundant dirty mucus;* dysphagia; difficult breathing; swelling of the pharyngeal region, which may extend to the parotid region. Pharyngeal spasm may be observed in some cases, accompanied by a suffocating cough and respiratory distress. If food or medicine be forced on the animal when suffering from the above symptoms, spasm of the glottis may be induced, followed by sudden death, or the fluids may enter the bronchi and set up septic broncho-pneumonia or pulmonary gangrene.

The anal glands occasionally become hot, swollen, and painful. Suppuration may occur in them, or they may secrete an abundance of purulent material, and thus induce an obstructive constipation. The anal and rectal mucous membranes may show marked redness and swelling, followed by the formation of ulcers and a tendency to bleed.

5. NERVOUS SYMPTOMS.—Nervous symptoms may usher in distemper, or may arise during its course or appear long after an apparent recovery. They are generally very grave in importance, and the mortality arising from the lesions producing these symptoms is higher than that of the other complications or manifestations of the disease; besides, if they do not lead to death, they often leave the animal a permanent or hopeless cripple. The older authorities, such as Youatt and Dick, considered that infection of the central nervous system occurred from the nasal channels

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* This material is said by many to be derived from the stomach, but this view is an erroneous one.

via the cribriform plates of the ethmoid bone. Blaine was doubtful of this mode of transmission, and was more inclined to believe in the generally accepted metastatic theory. Nervous symptoms are sometimes associated with ear lesions, and it is quite possible for a secondary infection to take place by way of the internal ear.

When the nervous symptoms arise at the onset of distemper, they appear in the form of epileptiform convulsions, which vary in degree from a few slight twitchings of the eyelids, ears, and mouth, up to convulsive movements of the whole body. They may be intermittent, and succeed one another with a more or less long interval, or may be continuous by one fit of convulsions arising before another fit is completed, and so on, until the attack lasts from half an hour to an hour or longer, and terminates fatally, unless relieved by treatment. When they arise towards the third or fourth week during the course of distemper, or when they succeed a sudden improvement in the symptoms of a grave case of broncho-pneumonia, or a profuse enteritic diarrhœa, or a prolonged total abeyance of appetite, they must be regarded as very serious symptoms. If, however, treatment affords relief, a recurrence may not take place—at least, until a later stage of the malady. When they arise in such a delicate breed as the Japanese spaniel, they are chiefly confined to a few twitching movements of the jaws and eyelids at the initial stage, but later on they become general, and then nearly always prove fatal. Convulsions, however, in the early or initial stage of distemper are rare, and when met with they are usually attributed to causes such as intestinal parasites, the presence of œstrum, excitement, or indigestion, etc. As a rule, these nervous phenomena are not regarded as evidences of an approaching attack of distemper, especially as symptoms suggestive of the disease, such as catarrh of the eyes and nostrils, may not appear until from one to three weeks afterwards. In some instances the nervous symptoms are rapidly followed by temporary paralysis; in others the paralysis may not appear until the third or fourth week. Should convulsions not appear before the period mentioned, they must be regarded as a very serious symptom, and may be succeeded by delirium, general paralysis, usually of a progressive type, choreic movements, and, finally, death in a condition of coma.

In some of the mild cases of distemper running a long course—from six weeks to two months—and only showing a slight eruption recurring from time to time, an occasional trivial diarrhœa and a slight serous or muco-purulent discharge from the angles of the eyelids, convulsions may occur suddenly, and the nervous symptoms may then apparently cease, the animal appearing in a normal condition. But without any warning a recurrence takes place, the nervous attacks appear with greater frequency and increased severity, and meningitis, paralysis, coma or chorea supervenes. This form of the disease is commonly met with in Chows, Samoyedes, and other Arctic breeds.

Occasionally young dogs, after apparent recovery from a mild form of distemper, if exposed to a cold wind, wet weather, fatiguing exercise, etc., within a period of one to three weeks afterwards, may suddenly develop recurring attacks of convulsions, followed by paralysis and death. On post-mortem examination of such cases, we find a quantity of serous fluid in the lateral ventricles of the brain or in the pericardial sac.

Chorea may appear quite suddenly during the course of distemper, but usually it is not observed before the third or fourth week. In some instances it may not appear until one to three months after an apparent recovery from distemper, especially in the case of toy pomeranians. The writer encountered one case in a dachshund in which chorea developed eight months after an attack of distemper. This condition may appear in the absence of any previous attack of convulsions, and it may be accompanied by meningitis, delirium, paralysis, or coma; or the choreic symptoms may pass off, and paralysis of a progressive type may succeed them.

In the writer's experience, chorea always originates from distemper, being either associated with the latter or following closely upon it. Although it frequently arises during a mild type of distemper, especially during certain years, it is often found associated with the severe form.

The choreic twitchings are said to arise from the toxic action of micro-organisms on the cells of the inferior cornua, the latter ultimately undergoing a process of sclerosis. Either a single muscle, one or more groups, or the muscles of the entire body, may be affected by the disease. The muscles of the head, sometimes those of the fore-limbs, and occasionally of the hind-limbs, are those most commonly affected.

The abdominal panniculus or the diaphragm may alone be affected. Chorea is not necessarily fatal, but when associated with other nervous complications it is usually so. The choreic movements rarely disappear altogether, but may become lessened in activity as the system recovers its tone. The writer has seen them completely disappear in one case after six years; in another case they were succeeded by a progressive paralysis, but ultimately absolute recovery resulted. When the muscles of the jaws become severely affected in young dogs, the constant intermittent pressure of the upper jaw on to the lower may gradually divert the teeth and yielding alveolar processes in an outward direction, so that the internal surfaces of the upper teeth rest on the internal surfaces of the teeth of the lower jaw. Chorea affecting the jaws may also be succeeded by a progressive paralysis of the jaws, and ultimately by death from general paralysis and coma. Chorea may occur in young puppies even when they are suckling their dam ; it is then not of serious importance, as a great number of such cases ultimately make a permanent recovery.

Paraplegia, or even *paralysis of all four limbs*, may appear alone, without previous convulsions, either early in the complaint or later in its course—generally about the third or fourth week. Usually, however, it is associated with other nervous phenomena, and is then very serious. When it appears alone, recovery is the rule, provided rational treatment be adopted. The writer has observed a case of paralysis of all four limbs in a Japanese spaniel that recovered after being in this condition for several months, during which time the poor creature could only shuffle about on its breast. It ultimately recovered, and maintained itself in perfect health for ten years (1901-1911), when brain symptoms appeared, and continued several months before the animal succumbed to a natural death.

Other nervous phenomena, such as hemiplegia, various forms of monoplegia, amaurosis, a difficulty of avoiding obstacles, loss of smell or hearing, loss of voice, whistling, roaring, rotatory movements, going in a circle, muscular spasm, hysteria, different types of paresis, unequal degree of contraction of the pupils, nystagmus, idiocy, depraved appetite, etc., are encountered during the later or convalescent stages of, or after an apparent recovery from, distemper.

Occasionally *meningeal symptoms*, such as an irresistible desire to cry or whine, or a continual hoarse barking, extreme restlessness, peevishness, hyperæsthesia, a hot head, and a congested state of the optic disc, are observed to succeed a slight diarrhœa, with little or no serous or catarrhal discharge from the eyes, a mild cutaneous eruption, a little vomiting, or a slight elevation of temperature. Such cases generally terminate fatally, and on post-mortem examination one may find a bronchopneumonia, the presence of which was not suspected during life. On the contrary, one rarely finds any lesions in the meninges or brain substance to account for the symptoms. As a rule, however, meningeal symptoms, which are occasionally rabiform in character, are associated with other nervous phenomena. In very rare cases the writer has observed a *meningo-encephalitis*, especially when involving the vicinity of the bulbar region, that led to *self-mutilation*. At first the animal shows an irresistible desire to lick a part, such as the paw, a small circular patch in the ilio-abdominal region, or a larger surface on the croup, or even the tail. This continues for some time, the animal eventually gnawing the patch until skin, and then muscle, and even bone, is consumed. In the case of the ilio-abdominal region the process has been carried so far as to perforate the abdomen and to allow the escape of the viscera. When the croup (which is a favourite part) is the region attacked, the hair and skin are removed over a large surface, which is left raw, oozing, and even bleeding. Death is the usual result, preceded by general paralysis and coma. When, however, the croup has been the region attacked, the writer has known of some cases of recovery. Mayhew is the only British author who refers to these lesions, and he is, according to the writer's experience, quite correct in attributing them to distemper. Several French and other Continental veterinary surgeons have described similar cases, which have, no doubt, been generally attributed to rabies or other causes. Hunting has also observed such cases. Cadiot met with a case in which the animal bit off one of its hind-limbs at the stifle joint. It should be mentioned here that Phisalix, Lignières, and others, have induced meningo-encephalitis in healthy dogs by injecting into their veins virulent doses of the pasteurella, or the virus of distemper. Therefore, in face of these experiments, it is important to bear this in mind when carrying out the diagnosis - inoculation of rabies, as it is quite possible for rabiform symptoms to be induced by material that is not of rabific origin, as has been shown by Macé, and by Harvey, Carter and Acton.*

Hydrocephalus sometimes complicates distemper. It is evidenced by an unsteadiness of gait, a blunting of the intellect, defective eyesight, and often by a vacant staring at obstacles which come in the animal's way, or by the animal getting into corners from which it seems unable to come out. It may follow on suppression or a sudden checking of the eruption in consequence of the animal being exercised in the open during wet or chilly weather. The lateral ventricles contain a quantity of fluid, in some instances amounting to several ounces.

Underlying nearly all these nervous phenomena are lung lesions. The latter may be masked by the former, or the pulmonary symptoms may disappear when the nervous phenomena arise. But the lung lesions usually remain in existence until the animal either dies or recovers. Recovery, however, is seldom observed. Therefore in every case in which nervous symptoms present themselves it is advisable to auscultate the chest carefully, in order to determine the presence or absence of lung lesions.

6. SUPPURATIVE OR PHLEGMONOUS SYMPTOMS.—During the course of distemper at certain periods one observes that some dogs, especially pugs,

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* "Pyocyaneus Infection in Dogs, and its Similarity to Rabies" (*vide Veterinary Record*, vol. xxiv., pp. 57-59, July 22, 1911).

bulldogs, and fox terriers, are attacked occasionally by phlegmonous and suppurative swellings in various parts of the body, but chiefly in the parotid and submaxillary regions. Probably this was the type of distemper referred to by Aristotle, Elian, and Virgil in their writings; also by Taplin in 1796, and other authors already alluded to. These swellings may also occur at the side of the jaw, in the parotid region, on the forehead, on the lips, above the eye, in the middle of the neck, in front of the shoulder, around the navel, and rarely in the inguinal region. These phenomena are either rapid or slow in progress. Sometimes they appear, mature, and rupture within three days; at other times they take a week or more to appear and disperse. The size attained may equal that of an orange or a cocoanut, and they are painful on pressure. After a time the skin over the centre becomes doughy, then purplish, and finally gangrenous, when they rupture, and give issue to a more or less thick straw or cream coloured purulent, or even hæmorrhagic, material. Occasionally the pus is thin, serous, and contains clots of fibrin. Healing takes place with difficulty, even when the abscesses rupture spontaneously, and often a fistula remains in communication with an extensive abscess cavity, especially in the region of the throat. Before attaining maturity they induce total loss of appetite; the animal is disinclined to move, and when the head is affected, it is held out in a straight line with the neck, which prevents the animal from easily turning his body or raising his head, and often he is unable to lie down properly or comfortably. The condition is sometimes associated with a suppurative process of the laryngeal or pharyngeal regions, or even in the internal organs. It may occur in an enzoötic form, all the dogs in a kennel being affected with this type.

7. Infantile Symptoms.—Although very young puppies suckling their dam may be affected with the usual catarrho-eruptive form of distemper (even before their eyes have properly opened), in consequence of the dam herself being affected with the disease, they may at the weaning period, or soon after, suffer from a form that is not usually recognised as distemper, but the symptoms are attributed to diarrhœa, indigestion, " worms," or a chill. In consequence of such errors in diagnosis, thousands of young puppies are destroyed every year by the drugging which they receive for worms. This form of the malady is manifested by a catarrhal enteritis, diarrhœa and vomiting, and the passage of a few worms with the vomit or with the fæcal material. Emaciation becomes rapidly profound; there is tympanites, a pot-bellied appearance, a dull, open, or staring coat; the animal feels cold, is clammy, moans, groans, grunts, or cries; is restless, disinclined to feed, and has a little serous fluid, sometimes tinged with a clot of yellowish muco-purulent

material, issuing from the eyelids, which soils the parts below these organs. A similar fluid is discharged from the nostrils. Sometimes there are eruptions, or the remains of such, on the almost hairless under-surface of the abdomen or the skin inside the thighs and arms, or even under the ears. There may be a slight elevation of temperature, but this is generally absent when the case is brought to the notice of the veterinary surgeon. At other times the puppies are found crying or moaning, and beyond showing a few eruptions, or perhaps a slight serous discharge from the eyes, there are no other characteristic symptoms. Usually such animals die in a comatose condition, and beyond a few isolated patches of broncho-pneumonia in the lungs, or a serous exudate in the pericardial, pleural, or peritoneal cavity, no other lesions are discernible. Some cases, beyond moaning, grunting, and crying, being dull or dazed, and having a tense, hard abdomen, may show no other symptoms. Even after death it may be difficult to connect such instances with distemper, unless some cases of the disease were known to have recently occurred in the same kennel or establishment. Puppies of ten or twelve weeks of age are very liable to have as a complication intestinal invagination, simple or complicated.

8. MALIGNANT SYMPTOMS.—During certain years one observes that in some kennels and in various localities distemper often assumes a very virulent and fatal type. It runs a very rapid course, and is accompanied by extreme weakness, rapid emaciation, absolute loss of appetite, and profuse diarrhœa, with frequent passing of fæces, composed chiefly of a brownish mucus, blood, and bile. One also observes a profuse ocular and nasal discharge of a bloody purulent material, occasionally of pure blood; the skin of the lips, cheeks, and ears becomes excoriated, and undergoes ulceration; the gums bleed, the inside of the cheeks become ulcerated, the tongue often undergoes necrotic changes, and the body gives off a putrid odour. Besides these symptoms, there may be rapid breathing; extreme dyspnœa, often amounting to orthopnœa; a troublesome short cough; pulmonary hæmorrhage; a swollen purplish or gangrenous condition of the fauces and pharynx; profuse expectoration; marked difficulty in swallowing; great thirst; delirium; a staggering gait; swollen anus; and ultimately a comatose condition, in which the animal succumbs. Death usually supervenes within three or four days from the onset, but in other cases it is delayed for two or three weeks.

9. HEPATIC SYMPTOMS.—The majority of authors and a large number of practitioners recognise jaundice as being of common occurrence in connection with some outbreaks of distemper—in fact, by many it is regarded as a special type of the disease. The writer has had very little

experience with this form of distemper, and it seems to be uncommon in London.

Blaine recognised the occurrence of jaundice in connection with distemper, and stated* that he had seen the disease accompanied with a marked biliary affection in every dog attacked in one season. He drew attention to the association of the pustular eruption with jaundice, and remarked that such cases, termed by sportsmen "the yellow disease," were of a very fatal character. The same author referred to the opinion of Youatt, who observed that jaundice was far more likely to occur when little catarrhal exudate from the nostrils was present. Blaine, however, had seen jaundice in cases accompanied by a profuse nasal discharge, but generally in instances where the distemper had assumed a virulent and putrid type.

Youatt, in his work on "The Dog," remarked: "In the pointer and hound, and particularly when there is little discharge from the eyes and nose, an intense yellowness often suddenly appears all over the dog. He falls away more in twenty-four hours than would be thought possible; his bowels are obstinately constipated; he will neither eat nor move about, and in two or three days he is dead. . . . Not one in twenty recover."

The clinical history of jaundice in connection with distemper, as observed by practitioners having experience of it, is as follows: The complication is more commonly met with in young hounds, and in some seasons entire litters may be decimated. As a rule, the evidences of jaundice appear suddenly, and the affection gradually spreads from dog to dog. Obstinate vomiting, constipation, extreme weakness, and entire loss of appetite, are the prominent symptoms observed, death being preceded by a comatose condition. A pustular eruption and an ocular and nasal discharge may be observed in some cases, but in the majority of instances these symptoms are absent. Gastro-enteritis may accompany the affection, but is not the cause of the jaundice. Invagination of the intestine is frequently met with as a complication.

The writer is of opinion that the etiology and pathology of this type of distemper require to be investigated. At present it does not seem possible to differentiate it from the malarial form of jaundice occurring in the dog, as the symptoms and post-mortem lesions are similar in both (see Jaundice, Vol. II.). Indeed, it is permissible to doubt the connection between this enzoötic form of jaundice and distemper until some definite knowledge is forthcoming on the subject. No doubt some practitioners meet with cases of distemper that during the course of the disease develop evidences of jaundice, while hepatic lesions are found on post-mortem. In such instances the jaundice is usually regarded as a complication of the disease, but it must be remembered that a dog may be suffering from an unsuspected affection of the liver prior to the attack of distemper. Formerly the malarial jaundice observed in Africa and elsewhere was considered by the colonists as a type of distemper, until its true nature was pointed out (*vide* Canine Piroplasmosis).

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* "Canine Pathology," 1841.

10. URINO-GENITAL SYMPTOMS.—Urino-genital symptoms are very rare, with the exception that the genesic instinct both in the male and female may be excited at the earlier stage of the malady. During the later stages the internal lining of the prepuce and the external lining of the penis may take on the catarrhal process, and secrete a large amount of yellow or straw-coloured muco-purulent or catarrho-purulent material, which bathes their mucous surfaces, and flows even in drops from the opening of the prepuce, soiling the surrounding parts and producing in them excoriation and even ulceration. Occasionally the external surface of the penis is covered with an herpetic eruption, which breaks down and forms small ulcerations. These bleed at times, giving rise to a profuse hæmorrhage. Such a condition is particularly seen when priapism seizes the animal. This discharge during distemper, or from other causes, is mistaken for that of gonorrhœa or true specific urethritis; but the latter does not exist in the dog. This preputial discharge, like that from the eyes and nostrils, occurring during the course of distemper is inoculable. A similar discharge may flow from the vulva of the female.

The bladder often becomes distended during distemper, particularly when the animal is suffering from great prostration, paralysis, or other nervous complications. It often gives rise to great distress and to whining, due to the discomfort it causes.

The *kidneys* may become affected by a catarrhal, parenchymatous, or interstitial nephritis; then symptoms attributable to such conditions may be observed.

11. MUSCULAR SYMPTOMS AND EMACIATION.—Occasionally rheumatic symptoms appear as the earliest manifestation of distemper, even a week or ten days before the advent of catarrhal symptoms. They are very misleading, and their true nature can only be ascertained as the case progresses, and some pronounced manifestation of the malady appears. The muscular weakness arising during the course of distemper is partly due to fatty degeneration of the muscle fibres, induced by the high febrile or toxic reaction on them, and partly to the diminished nervous power. It may be so extreme that the animal cannot stand, but assumes the lateral recum-

bent position, and is not necessarily a fatal sign, even when accompanied with a semicomatose condition, as the writer has seen cases accompanied by such symptoms that terminated in absolute recovery. The muscular paralysis or clonic convulsive movements nearly always result from some disturbance of the central nervous system. Muscular cramps, especially of the thighs, are occasionally seen, as well as spasm of the diaphragm. Gradual emaciation occurring during the earlier stage of this complaint, especially if accompanied with elevation of temperature, profuse diarrhœa, and very little appetite, is not in itself a bad omen. On the contrary, should the emaciation be rapid in its progress and accompanied with profuse watery diarrhœa which nothing will check, an absolute loss of appetite and a semicomatose condition, associated with an offensive odour from the body generally, it is mostly a fatal prognostic. At times there is no loss of condition, even when there are such serious complications as meningitis and myelitis, with delirium, convulsions, or a semicomatose state. Delicate dogs during the convalescent stage often faint from cardiac muscular weakness.

12. FEBRILE SYMPTOMS.—Most authorities have considered distemper as a febrile disease, but in practice one may observe numerous cases that never show any elevation of temperature during the whole course of the malady, even when it terminates in death. Probably, however, if one could observe every case from the initial stage, one would find that there was a temporary but ephemeral high temperature in the majority of such instances. Still, a great number of other instances show a short continuous febrile stage, which usually disappears as soon as the skin eruption is manifested, or when the malady concentrates itself upon some particular organ or region, and sets up some reactive disturbance. As a rule, the temperature is elevated at the commencement and even during the course of a complication or secondary infection of an important organ, such as the lungs or central nervous system. It is usually atypical, but if it should be continuously progressive, it generally ends abruptly, and does not decline gradually. A continuous high temperature during the earlier stage may be followed by absolute recovery. But if arising late in the course of the disease, or after an apparent recovery, it is often an indication of some complicating infection, particularly of the lungs or nervous system, and is, in consequence, generally a grave sign, as these cases mostly end in death or an incomplete recovery. Many puppies in infected surroundings, but otherwise placed under favourable conditions, may only show an erratic elevation of temperature, and perhaps an occasional period of dulness, but no other symptoms.

When the infection is rapidly acute, and a profound impression is thereby made upon the nervous system, a subnormal temperature may be encountered. But this, although generally a grave sign, may not always be the forerunner of death, as some at least of such cases end in absolute recovery. It is, however, always a fatal prognostic sign when it appears late in the malady, after a course characterised by high temperature and marked emaciation, or when accompanied by jaundice or some septic condition.

COURSE.

Usually distemper takes five or six weeks to run its course, but not rarely, with complications or secondary infections, this may be extended to two, three, or more months. Occasionally an animal seems to suffer not more than "a mere catarrh," and is well within a week or two. Death may take place at any period during the course of the disease.

PROGNOSIS.

This is always difficult and uncertain, even in apparently slight or grave cases of distemper, and one therefore cannot be too guarded in giving a forecast of the termination of any particular case, however so hopeful or adverse it may appear to be. Youatt, one of the most classical masters of this subject, said more than eighty years ago: "There are some seasons when we can scarcely save a dog; there are others when we must wilfully destroy him in order to lose him. There are some breeds in which, generation after generation, five out of six die of distemper, while there are others in which not one out of a dozen dies." These conditions should be taken into account before forming an opinion as to the prospects of a recovery.

The prognosis is always very grave in the case of the Japanese spaniel, of which 90 per cent. or more die; grave in the young dog before he has erupted his permanent teeth; doubtful in such breeds as the Chow-chow, borzoi, Samoyede, and miniature varieties, and in those of any breed after ten years of age; more hopeful in those over twelve months old, especially in the case of the hardier breeds or mongrels.

The pneumonic or nervous manifestations are always very grave, or almost hopeless, especially in the case of the former, when there may be extensive hepatisation, pulmonary œdema, hæmorrhagic muco-purulent discharge from the eyes or nostrils, laboured and buccal breathing, suppressed or muffled cough with no expectoration, rapid and maintained emaciation, total loss of appetite, persistent refusal to swallow nourishment, jaundice and a pustular eruption or suppurative skin lesions appearing late in the course of the disease, an offensive odour given off from the skin, and a sudden improvement in the appetite after it has been in total abeyance for some time. It must be particularly pointed out that, should these conditions seem to improve, they are then generally followed by chorea, convulsions, delirium, and coma or paralysis ; so that if the animal does not die immediately or soon after, it has, as a rule, to be destroyed. The nervous manifestations, such as convulsions, chorea, delirium, coma, etc., generally appear late in the malady, and, with the exception of uncomplicated chorea, are nearly always fatal. If, on the other hand, the convulsions appear early and the seizures are only few in number, or there is an early generalised eruption without pneumonic complications, or a diarrhœa, even profuse or continuous, without much emaciation, and if, in addition, the appetite be good, there is a fair prospect of the animal recovering.

A continuous high temperature appearing in the later stages of a hitherto favourable case, in which the temperature in the early stages after the invasion period has been normal, is rather a serious sign, usually indicating that a secondary infection has taken place, and probably that lesions are in process of development, either in the lungs or nervous system. However, grave symptoms may be present, and yet the temperature has been continuously normal. A sudden spontaneous cessation of a hitherto profuse and continuous diarrhœa, a sudden improvement in the condition of an animal suffering from severe pneumonic lesions, or a sudden improvement in the appetite after it has been in total abeyance in very serious cases, portend a so-called translation or metastasis of the disease, usually to the brain, and a termination in death from convulsions, delirium, or coma.

A storm following dry, hot weather, or mild, wet weather succeeding dry, cold, or frosty weather, frequently alters what was up to then a favourable course of an uncomplicated case into a serious or protracted one.

In a dog not quite recovered from distemper, a relapse or a repeated infection derived from another animal suffering from a more virulent type of the disease, is very grave, and in the majority of instances ends in death. One should not lose sight of the fact that the so-called sequels of distemper, such as chorea, paralysis, meningo-encephalitis, fits, and other nervous disturbances, pneumonia, and skin disease, may arise after an apparent recovery from distemper. The writer has seen numerous instances of such sequels arising one, two, three, or more months afterwards, and, as already remarked, in one case chorea appeared eight months after convalescence.

MORTALITY.

This varies according to the season, the age and breed, the type of the disease, and the locality of its occurrence. It is highest during the changeable chilly weather of spring and autumn, especially after dog-shows, or in cases of infection contracted from dogs coming from dogs' homes or dog-shops; in places where a great number of dogs are congregated together; in animals under six months and over eight years of age; in those of exotic origin; and in places, districts, or countries where the disease has never been known before, or where it reappears after a long interval of freedom from the malady.

In Iceland and Greenland nearly all the dogs were destroyed by it. Quite 90 per cent. of the Japanese spaniels bred in England, 50 per cent. of the Arctic breeds and the Chow-chow, succumb; whilst in the Pekingese spaniels the mortality does not go beyond 25 per cent.

Of the indigenous breeds, the bloodhound, Newfoundland, and collie under six months of age, out of every hundred attacked fifty die; the fox-hound, one out of three; the toy pomeranian and the toy spaniels, one out of four; the fox terrier, Irish terrier, and Airedale terrier, one out of six; and the sporting spaniels, retriever, setters, and pointer, one out of five, succumb.

In certain kennels during particular years most, if not all, of the young dogs of whatever breed die; in others scarcely one is lost. An isolated dog living in the open country or a mongrel kept by itself, in nineteen cases out of twenty survives it.

In adult or middle-aged dogs the death-rate is not very high. In certain years when the malignant type prevails, the mortality is very heavy; whereas when the milder types predominate it is very low. It is highest in the pneumonic and nervous types, and where there are frequent relapses and repeated infections, but is lowest in the uncomplicated catarrhal and eruptive forms, the abdominal type standing midway between these two extremes. On an average, however, it is quite 25 per cent. Youatt computes it at "fully one-third of the canine race."

DIFFERENTIAL DIAGNOSIS.

This is carried out with facility in a well-advanced case exhibiting the characteristic symptoms of the disease, but is very difficult, or may be impossible, at the early stage, or even during the course of atypical cases in which we may not observe any of the usual phenomena by which the malady is generally recognised.

When several animals in one place are ill, it should make one suspicious of distemper. A sick animal purchased a few days before at a dog-shop, a dogs' home, or even from a breeder, or coming from a show, should make one more suspicious, and in nine cases out of ten time will prove one's surmise to be correct.

A dog, especially a young one, manifesting an unaccountable dulness, capricious appetite, a disinclination to play or run about as usual, having a tendency to sleep at every opportunity, showing an elevation of temperature, slight shivering, and a vesico-pustular eruption on the almost hairless skin under the abdomen, should afford one no difficulty in determining the nature of the illness. A young animal suffering from a persistent diarrhœa, with gradual emaciation, or a series of epileptiform seizures, accompanied by a rise of temperature, should be sufficient to put the practitioner on his guard until some more pronounced symptoms appear, such as the vesicular eruption or a little thickened yellowish or yellowish-green mucus in the inner angles of the eyelids, and enable him to declare the presence of distemper. Very often an apparent recovery occurs in such cases after a few days' treatment, but the symptoms reappear with more marked energy after a short period, and the practitioner may be taken off his guard by such cases.

At another time a dog may be presented suffering from an ulcerative condition of the corneal membrane. It is easy to arrive at its true nature if there are several animals showing similar lesions or perhaps characteristic symptoms of distemper; but in an isolated case we can only conclude that it is one of distemper by the injection or congestion of the conjunctiva, which is swollen and secreting an abundance of yellowish or straw-coloured pus, by the elevation of temperature, the condition of the animal in general, and perhaps by an eruption inside the ears, under the abdomen, inside the thighs, on the labia of the vulva, or on the scrotum. In other cases, especially in the toy pomeranian or other small dogs, there is a diarrhœa which is persistent, but the animal may be lively, and take its food as usual. The true nature of the case may be determined by the fact that there is a brownish, rusty slime covering the back teeth, chiefly in the vicinity of the precarnassial and carnassial molars, and that there are a few scattered eruptions on the skin in the vicinity of the abdomen.

In any case of illness accompanied by a very injected conjunctival membrane, a slight secretion having a greenish resinous appearance in the corner of the eyelids, a fit of coughing succeeded by expectoration, some dulness or lassitude, and an elevation of the internal temperature, the practitioner may safely come to the conclusion that the case is one of distemper.

If, on the other hand, the cough be frequent, especially during movement, and if of some duration, and one observes an occasional vomiting after or during the act of expectoration, gradual emaciation, an evening rise of temperature of from 1° to 2° F., a bright facial expression, and a fair appetite, although a physical examination of the chest reveals the presence of pulmonary lesions, or of pleuritic effusion, then one may suspect the case to be one of pulmonary tuberculosis. Of course, the diagnosis should be confirmed either by a bacteriological examination of the sputum or, preferably, by the employment of the tuberculin test (see p. 182). Some forms of distemper are characterised by the presence of laryngitis, bronchitis, or pneumonia, accompanied by cough, a thick muco-purulent discharge from the eyes or nostrils, emaciation, and depression. In others convulsions, chorea, or ulceration of the cornea, are associated with the general symptoms of illness, or with diarrhœa.

The writer feels obliged to confess that he is yet unable to distinguish clinically those so-called sporadic cases of gastro-enteritis, tonsillitis, bronchitis, pneumonia, enteritis, etc., from similar affections accompanying distemper. No doubt the sporadic cases (which in his experience are rare) are not, with the exception of pneumonia, infectious, and only one out of several dogs in the same establishment is attacked. In the case of distemper, however, several dogs in a large kennel are affected, and such outbreaks are very common. Sporadic cases of bronchitis or bronchopneumonia generally occur in aged dogs, but they also may contract the infectious form if in contact with dogs suffering from distemper.

It should not be concluded that because a dog has no elevation of temperature, and an absence of cough, catarrhal discharge, and eruption, he can be regarded as free from distemper. From the point of view of prophylaxis, it is far wiser to isolate any dog showing even suspicious symptoms until a definite diagnosis can be arrived at. Early recognition of the disease is of importance in connection not only with preventive measures, but also with treatment.

Youatt's teaching as to the multiform aspects of distemper should not be overlooked. He says (1830): "In one season almost every dog with distemper has violent fits; in another, in the majority of cases there will be considerable chest affection, running on to pneumonia; a few months afterwards a great proportion of the distempered dogs will be worn down by diarrhœa, which no medicine will arrest; and presently it will be scarcely distinguished from mild catarrh." In tropical and in some European countries distemper may be mistaken for canine piroplasmosis and trypanosomiasis, in which catarrhal, ocular, and nervous symptoms may be present.

PROPHYLAXIS.

This is divided into (1) *prophylaxis proper*, or measures directed to the prevention of contamination by the virus, and of the extension of the disease to other dogs; and (2) *immunisation*, or rendering the animal refractory to the disease, so that the virus does not gain a foothold and generate itself in the system.

1. Prophylaxis Proper.—Much time, money, a great deal of disappointment, and an untold amount of suffering, misery, and loss of animal life, could be prevented if dog-owners were to cast aside their prejudices and erroneous ideas, and to obtain instead an intelligent knowledge of the nature of contagium and methods of prevention. The current idea seems to be that if an animal appears well, or is only suffering from a so-called "slight ailment" or chill, or is just "over distemper," no danger is to be anticipated, but the dog is to be regarded as almost guaranteed free from infection. Hence it is considered safe to introduce a newly-purchased dog, or one just recovered from distemper, to the healthy inmates in the kennel. Some dog-fanciers behave in a manner peculiar to fatalists, manifesting stolid indifference, and considering that if a dog is to have distemper he will get it in spite of whatever they may do to prevent it. Others, again, are too confident, and opine that, because they have never had distemper in their kennels, they are proof against its invasion. There are yet a few who innocently believe what is told them, or even guaranteed verbally or in writing by a friend, or a dealer, or a fancier, that the animal they are purchasing is "over distemper," or is suffering from a mere chill, a cold, "worms," a slight skin eruption, or "that's nothing," when in reality the dog is already, or will soon be, suffering from the malady.

In a kennel of healthy dogs care should be taken that no strange animal coming from outside—especially from a home for lost and stray dogs, a dog shop or stores, a dog-show, a sanatorium or boarding establishment for healthy dogs (where no distemper is *admitted*), or a veterinary infirmary (in which no distemper cases are taken)—is introduced or brought in contact with its inmates until it has been *rigidly isolated and confined for at least three weeks*; and if during this period it has not manifested the least sign of any illness, it may be washed in a bath of water, to which is added kresophen, and may then be considered free from infection. Even a bitch going to the stud-dog should be regarded with suspicion unless it has *all* the appearances of good health, and a guarantee is obtained from her owner that distemper does not exist, nor has existed for the past three months, in his kennels. The converse should also be insisted upon, because a healthy bitch may contract the infection at the stud-dog establishment, and take it home to her own kennels and contaminate her companions.

In a kennel of fox-, otter-, or stag-hounds, each couple of pups returned from " walk " by farmers and others should be strictly inspected and isolated in a separate kennel, or separated by a partition ; also, they should be exercised by themselves for at least *three weeks.* (The partition or compartment should be of board, brick, or sheet-iron, without any holes, lattice-work, or crevices for the neighbours to sniff at or put their tongues through to lick their friend's nostrils.) If at the time of their incoming they are not absolutely healthy, they should be sent back to the walker; or if they manifest any sign of illness during their period of quarantine, this should be maintained, or the animals sent right off the premises, and their kennel thoroughly washed and disinfected before any fresh dogs are admitted thereto. Before the pups come in from " walk," strict attention should be given to the permanent adult inmates and to the young pups that have never been off the premises, as often these are suffering, or have just recovered, from distemper, and transmit it to the returned couples, who too frequently obtain undeserved blame for its introduction. The same attention should also be given to the kennels and utensils, which should have been thoroughly scraped, scrubbed, and then disinfected before the quarantined couples are set free to commingle with the old stock. There is no law, so far as the writer is aware, that compels an owner to keep his dog suffering from distemper off the public highway or any other public place, and hence there is a great danger of a pack of hounds or even any dog becoming contaminated in this manner. Dogs at liberty should be rigidly excluded from the surroundings of kennels containing valuable dogs, as they often introduce the infection.

In the case of the dog-showing public much remains to be done with regard to the suppression of distemper. The Kennel Club should compel an exhibitor to give a guarantee in writing that his or her kennel is free, or has been so for at least three months previous to the dogs being benched, and it is only on this guarantee that the dogs should be permitted to enter for any show held under their rules. Should an exhibitor present a dog or dogs showing symptoms leading the official veterinary surgeon to suspect or to declare that the animal is suffering from distemper, the owner should offer the Kennel Club or their veterinary surgeon (who should not be the owner's professional adviser) every facility to visit and inspect the kennel and its inmates. If distemper is found to be present in any of the dogs, or in case the exhibitor refuses to allow the Kennel Club or their veterinary experts an opportunity to inspect the kennel or dogs, the Kennel Club should suspend the exhibitor for an indefinite period or for all time. In the case of a difference of opinion arising between the exhibitor's veterinary representative and that of the Kennel Club, the different representatives should nominate a veterinary umpire (who is not, and never has been, an adviser to the exhibitor under trial), and his decision should be held binding between the parties. An attempt should be made to obtain powers in the form of an Act of Parliament * to deal with the question of a dog-owner being compensated for loss arising from the negligence of other dog-owners in sending diseased dogs to shows and other places to disseminate disease. In the case of an exhibitor's dog or dogs contracting distemper from a dog or dogs the property of another exhibitor who knowingly or carelessly ignores the existence of the disease in his animals, or breaks the guarantee, the aggrieved exhibitor would then have power to sue in a court of law for loss or expense sustained in consequence of the other exhibitor's carelessness or breach of agreement.

The Kennel Club should give their veterinary surgeons more powers than the latter seem to possess at present in the inspection and exclusion of dogs at shows. No dog should be permitted to enter a show when found to present the least trace or suspicion of distemper, or of any other disease. Not only do the present rules require alteration and enforcing, but also the members of the Kennel Club should not complain when their veterinary inspectors endeavour to carry out these rules. It is a well-known fact that some veterinary surgeons refuse to officiate at dog-shows in consequence of the difficulty in enforcing rules, and also because the measures adopted are not sufficiently stringent to be successful in preventing the spread of infection.

It is advisable that a veterinary surgeon officiating at a dog-show in conjunction with one or more colleagues should not examine dogs the property of his clients, but when such are presented, he should depute their examination to one of his colleagues.

A healthy dog sent for sale or on approval to a dog shop or stores, or other place, should, in case of its return, be isolated for three weeks, as often disease is introduced into a kennel by such a transaction, the dog contracting infection at the dog-shop or other place.

In the case of a veterinary infirmary, every dog should be *strictly isolated*, by reason of the fact that an apparently healthy dog may have the infection in its system, and is capable of spreading the disease to other animals before evidences of the affection have declared themselves, or before the animal has been removed to the distemper ward. Every animal having a dry nose, a watery discharge from the nostrils or eyelids, manifesting a cough, an elevation of temperature, a pustular eruption, a mere chill, diarrhœa, vomiting, convulsions, loss of appetite, or dulness, should be treated as a suspect, and therefore isolated in a special ward until the nature of the malady can be determined. No dog or cat should be allowed to roam about an infirmary, as it is liable to convey the disease from the sick to the healthy, even when it is perfectly healthy itself. Rats and mice should be cleared out of an infirmary or a kennel where sick and healthy animals are confined, as they may be the means of conveying the disease from the sick to the healthy.

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* The writer is informed that the present state of the law is quite sufficient to obtain legal redress in connection with this matter.

A properly equipped veterinary infirmary, where cleanliness is observed and care maintained, is not so likely to be the means of spreading the disease as a distempered dog being allowed in the streets to come in contact with all and sundry dogs, and to communicate to the susceptible ones its malady. This is commonly observed in London and other large towns, and also at seaside resorts, where dogs coming from various parts congregate. The writer has had many years' experience in this direction, and from it can confidently assert that when distemper spreads to healthy animals in a veterinary infirmary, it is partly due to non-detection of the disease, partly to improper isolation, and partly to carelessness. The experience of St.-Yves Ménard, Parsons, Flook, and others, confirms this. Where care is used, distemper cases may be isolated and confined under the same roof as non-infectious cases, without distemper spreading to the latter. It, however, frequently spreads in those establishments which profess not to take in dogs suffering from the disease. The infection does not appear to be carried by the atmosphere ; it is by the living animal, the hands of the attendant, the thermometer, feeding utensils, medicine-spoons or bottles, clothing, grooming utensils, and excreta, that it is chiefly conveyed to the healthy. The writer has never known it to be conveyed by a veterinary surgeon who washes his hands and thermometer before touching another case. The mere presence of a distempered dog in a room for a short time does not, according to his observations, leave the infection behind it, as he has frequently seen healthy dogs, even Japanese spaniels, go into the same room, immediately or soon after it has been vacated by the sick animal, without contracting the disease.

The prevention of distemper at a home for lost and stray dogs seems a hopeless task, especially as such places are generally conducted on sentimental rather than on hygienic lines. Provided expense is no object, its prevention, or at least a reduction in its spread, is realisable. Every dog should have a separate compartment in which it could neither see its neighbour nor communicate infection to it ; every dog should be exercised by itself. Dogs kept like this would be far happier, quieter, and less liable to injury and to distemper, than when a lot of nondescripts, many of them suffering from various diseases, including distemper, are congregated together to snarl and snap at one another, transmit disease, and to continuously make an unbearable noise. There should be no difficulty in carrying on a home for lost and stray dogs on hygienic and therefore on humane principles, especially as such places receive ample support from the public, and are supplied with every convenience.

In the event of a case of distemper occurring in a large kennel, immediate isolation should be carried out, and it is of importance to do this on the first appearance of suspicious symptoms. The following instructions should be given to the attendant: (1) Keep separate feeding utensils for the sick and the healthy animals. (2) Wash the hands thoroughly before handling a healthy dog. (3) Wear a smock when attending sick animals, and remove it when going near healthy animals. (4) Always feed and attend the healthy before the sick. (5) Take the temperature of all the unaffected dogs twice daily, and if any show a rise of temperature above 102·8° F., or the presence of symptoms such as discharge of a watery material from the nostrils or eyes, shivering, loss of appetite, cough, or diarrhœa, carry out immediate isolation. (6) Every kennel or compartment in which an affected dog has been kept should be thoroughly scrubbed and disinfected, or, when convenient, limewashed.

Disinfection.—The contagium is readily destroyed. This fact has been observed by Nocard, Ménard, the writer, and others. Thorough washing of a kennel seems sufficient to destroy all infection. Jenner recommended lime-washing with freshly slaked lime. Blaine and his followers were staunch advocates of the use of chlorinated lime or chlorinated soda for the disinfection of kennels. Sewell speaks well of sulphurous anhydride, but for this to be efficacious it is necessary to close up every crevice, hole, or opening, including those in the windows, door, fireplace, and ventilators, by gluing or gumming paper over them. The gas may be generated by burning sulphur on an oil or gas stove, a coke-fire, a red-hot shovel, or by igniting a specially made sulphur candle. This method is, however, impracticable in the majority of kennels, and probably not very efficacious under the best of conditions.

Kresophen is a very good and reliable disinfectant, and as it has rather a pleasant odour, is soapy, and mixes well with water, in the proportion of 1 part in 20 of water, may be conveniently used for washing down the walls, the floors, baskets, whips, chains, collars, clothing, brushes, etc. It is also advisable for the attendant to wash his hands and the thermometer in a similar solution of this fluid after attending a distempered dog.

In France a cresol preparation is largely used for general disinfection purposes. It was introduced by Paul Adam,* Professor of Chemistry at the Alfort Veterinary School, at the instigation of Dr. Roux, for the French Committee of Epizoötics. The formula is as follows:

Cresol	1 part.
Soda lye, 30 per cent. (d = 1332) ..	1 ,,

Mix.

This readily dissolves without heat, and is very cheap. It easily mixes with water in any proportion, remains clear, and leaves no more deposit than boiled water. It may be diluted with 100 to 400 parts of water, according to the strength of the disinfectant required. It does not injure varnished work.

A kennel, room, benching, etc., after being well washed and disinfected, may be used immediately it is dry for healthy puppies, without any fear being entertained of the animals contracting distemper therefrom. This is also Sewell's experience.

2. IMMUNISATION.—This is subdivided into (1) *active immunisation, vaccination*, or *protective inoculation*, when one desires to render the animal refractory to the infection; and (2) *passive immunisation*, when infection is imminent, or has already gained an entrance to the system. This is often termed *curative serotherapy*, or *antitoxic treatment*.

(1) *Active Immunisation, or Vaccination.*—The first method introduced was that of *inoculating with the nasal discharge* of a distempered dog, the object being to produce a milder or less fatal disease. Blaine and Youatt condemn this practice in consequence of it having produced the disease in a virulent form in the vaccinated animals, even when the material was taken from the mildest types; and in some instances it caused fatal results. In spite of this, Krajewski, Woodroffe Hill, Hayne, Bryce,† and others, have since recommended it. Following this, Viborg,‡ Saccho, Jenner,§ Hamont, Nauche, Trasbot,‖ Spinola, Sutcliffe Hurndall,¶ and others, believing in the variolous nature of distemper, practised and advocated the use of *cow-pox virus* or *vaccine*. But Professor William

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* *Recueil de Méd. Vét.*, tome lxxxii., pp. 485-493, Août 15, 1905.

† Bryce, *Veterinarian*, 1882.

‡ Viborg, *Veterinar. Skrifter* 3, Copenhagen, 1795.

§ Jenner, Medico-Chirurgical Transactions, March 21, 1809.

‖ Trasbot, *Arch. Vét.*, 1879, p. 161; *Bull. Soc. Cent. Vét.*, 1885, pp. 306, 412, 443; *Rec. de Méd. Vét.*, Août 30, 1903.

¶ Sutcliffe-Hurndall, Proceedings of the National Veterinary Association, London, 1902.

Sewell,* "in 1799, and when distemper was very virulent, inoculated several puppies with vaccine matter procured from the small-pox hospitals. His place of inoculation was the inside of the thighs. He must confess that he never found it to be a preventive of distemper; several of the dogs afterwards sickened and died of that disease. . . .

"He does not deny that many dogs, after being vaccinated, never exhibited symptoms of distemper; but it does not follow that all dogs should have distemper, any more than that all colts should have strangles."

Youatt † vaccinated sixty dogs with cow-pox virus in one year. He says: "In the greater part of them I could not, or at least did not, produce the characteristic pustule; but where I did, or very nearly so, some of them resisted the power of contagion. But others—and more than half—before the expiration of the second month had become distempered; consequently, and without the slightest hesitation, I drew the conclusion that this boasted preventive had no power at all."

Blaine ‡ says: "Vaccination so performed is a proof of the gullibility of the public and readiness to believe what is wished, particularly when it comes from a popular source, and where it would be *unfashionable* to disbelieve. . . . It neither exempts dogs from distemper, nor does it appear at all to mitigate its severity." Dupuis,§ of the Brussels Veterinary School, denied that it had any value whatever (*vide* Canine Variola, p. 594). Sewell ‖ says: "It is perfectly useless." Hobday ¶ considers "it is mere humbug to use it."

Trasbot, after numerous experiments by vaccinating with the vesicular pustule of distemper, which he considered was the essential lesion and contained the essential virus, claimed to have always communicated the disease, and after recovery immunity was conferred on the animal. However, towards the later years of his life he abandoned this procedure, as complications were frequent and deaths often occurred.

Active Immunisation with Pure Cultures.—Mathis (1887), Millais (1889), Gallio-Valerio (1895), and many others too numerous to mention, endeavoured to obtain pure cultures of bacilli, which they respectively considered to be the essential organisms in the production of the disease, and with these cultures they attempted to induce active immunity. But, like the previous experimenters with the crude virus or with the cow-pox virus, they failed to satisfy the demands of dog-owners and scientific observers.

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* William Sewell, "On Vaccination of the Dog," abstract of the Proceedings of the Veterinary Medical Association, January 23, 1838, pp. 62-63.

† Youatt, "On Vaccination of the Dog," abstract of the Proceedings of the Veterinary Medical Association, 1837-38, pp. 60, 61.

‡ Blaine, "Canine Pathology," 1841.

§ "Recherches Experimentales sur la Vaccine et la Maladie des Jeunes Chiens," *Annales de Méd. Vét.*, 1887, p. 305.

‖ Sewell's edition of "Mayhew on the Dog."

¶ Proceedings of the National Veterinary Association, London, 1902.

*Phisalix's Monovalent Vaccine.**—Phisalix, Chauveau's assistant, in 1901 isolated a very small bacillus, having all the specific characters of the pasteurella or fowl-cholera group, from dogs destroyed during the early stage of distemper. This bacillus, when inoculated into susceptible dogs, produced all the characteristic manifestations of the disease. Cultures were easily obtained on a medium composed of a 6 per cent. glycerinated bouillon. By subcultures or by age they became progressively attenuated, and vaccines of various degrees of potency were produced. In practice he advised two vaccines, one weak and the other stronger. Of the weak or *first vaccine* 2 c.c. were injected under the skin inside the thigh of a puppy of six or eight weeks of age. Ten or twelve days later the *stronger* or *second vaccine* was injected under the skin inside the opposite thigh. In cases where dogs were subject to hypercontamination he recommended a *third* or *strong vaccine*, which was almost a virulent culture, and was to be used two or three months after the second inoculation.

Lignières' Polyvalent Vaccine.†—This vaccine is prepared from the same organism as that of Phisalix, the *Pasteurella canis*, but various strains are used. This mixture of organisms is grown on agar-agar in series for more than a year by inoculating culture media every two days, so that they are renewed more than five hundred times, as Lignières believes there is by this method less risk of the microbes returning to their former virulence. For their preparation the cultures are made in flat-bottom flasks, containing a layer of peptonised bouillon 1 to 2 c.c. thick. For the *first vaccine* the temperature of 42° to 43° C. is maintained for five days, and only two days for the *second vaccine*. One c.c. of either of these vaccines in proper order is used in the same manner as those of Phisalix.

Copeman's Vaccine.‡—This is a different organism from that of Phisalix and of Lignières. It is a cocco-bacillus, which forms streptococci when grown on certain media, liquefies gelatine, and resembles Mathis's bacillus, isolated in 1887. A vaccine is prepared by heating a broth culture of the bacillus at 60° C. for half an hour, and subsequently adding a small quantity of carbolic acid as a preservative. It is standardised according to the method devised by Haffkine and Wright in the preparation of antiplague and antityphoid vaccines for man. The dose is 2 c.c. for a dog of the size of a fox-terrier puppy.

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* "Recherches sur la Maladie des Chiens, Vaccination du Chien contre l'Infection Expérimentale par Bacille Spécifique," *Comp. Rend. des Séances de la Société de Biologie*, Séance du 20 Juin, 1901.

† "Sur la Vaccination contre la 'Maladie des Chiens,'" *Rec. de Méd. Vét.*, 30 Juillet, 1903, p. 341 ; "La Vaccination de la 'Maladie des Chiens,'" *ibid.*, 30 Août, 1903, pp. 371, 384.

‡ Proceedings of the Royal Society, vol. lxvii. ; *Veterinary Record*, 1901, vol. xiv., p. 313.

(2) *Passive Immunity or Serotherapy — Lignières' Antidistemper Serum.*[*]—Lignières, in 1902, introduced a *polyvalent preventive* and *curative serum* by inoculating horses with small doses (5 to 20 c.c. at the most) of his polyvalent vaccine mentioned above, and repeating them at intervals of a few days. The injections are made under the skin, but as soon as the animal acquires a certain degree of tolerance they may be made into the vein. As a curative agent the serum obtained should be used early in the course of distemper, even when the lungs are involved. It is without action in the case of secondary infections. The intravenous dose is from 5 to 10 c.c., the subcutaneous dose being from 15 to 30 c.c.

Piorkowski's Antidistemper Serum.[†]—This is prepared in a similar manner to that of Lignières, but the writer is unable to state what microbes are used or how they are cultivated. It is employed both as a preventive and as a curative agent. The potency of this polyvalent antitoxic serum is ascertained on a guinea-pig of a given weight, and only a serum of definite strength is used. The present serum contains 20 immunity units in 1 c.c. As 1 unit is that quantity of serum which protects ten guinea-pigs against an infection with ten times the minimum lethal dose of the virulent culture, it follows that $\frac{1}{2000}$ c.c. is capable of protecting one guinea-pig from an infection with a tenfold lethal dose. It is asserted that in practice Piorkowski's serum has been used with the greatest success, and it is claimed that 90 per cent. of cases of distemper treated with it recover rapidly, and that it proves efficacious with the catarrhal and nervous forms as well as with the intestinal ones. It is advised to use it as early as possible, and care should be taken to avoid all forms of contamination. The serum is injected subcutaneously, in the neck for choice. The selected spot should first be thoroughly cleansed by scrubbing with soap and water, and then swabbed with alcohol. But the writer has found this to be unnecessary. The injection is then made with a sterilised serum syringe. The seat of injection should then be smeared with collodion. This is also, in the writer's opinion, unnecessary. The *preventive dose* is 5 to 10 c.c., according to the size of the dog. Immunity lasts about five months, when a second injection should be made to carry the dog over one year. For a *curative dose*, 10, 15, 20, or even 50 c.c., should be used, according to the severity of the case and size of the patient. It is quite harmless, and does not produce any fever or injurious complications. The serum is carbolised and aseptic, and retains its activity for at least one year, if kept cool, but free from frost, and in a dark place. Mr. William Frederick Garside, F.R.C.V.S., to whom the writer is greatly indebted for much information on this serum, speaks very highly of its efficacy.

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[*] Lignières, J. et M., "Vaccination contre les Pasteurelloses," *Comp. Rend. de l'Acad. des Sciences,* 1902; Lignières, J., et Spitz, "Sérum Polyvalent contre les Pasteurelloses," *ibid.*

[†] A. Leitch, *The Kennel,* March, 1912.

There are various German and American antidistemper sera on the market, but their composition is unknown.

Ferry has introduced a polyvalent vaccine prepared from cultures of several strains of his *Bacillus bronchicanis*, which are sterilised before use; also a polyvalent but polymicrobial vaccine prepared from cultures of several strains of *B. bronchicanis*, *Staphylococcus albus*, *S. Aureus*, and *Streptococcus pyogenes canis*. Their practical utility remains to be demonstrated.

Polyvalent Antistreptococcic Sera.—Several polyvalent antistreptococcic sera are on the market for use in the streptococcic complications of distemper. They are prepared from various strains of streptococci, which are cultivated on artificial media and then injected into the horse. As they are quite harmless, they may be tried in this disease.

The writer is unaware whether the practice adopted in the case of cattle-plague, in which the blood of a recovered animal is injected under the skin of one side of the neck and the virulent blood under that of the other side, has ever been carried out on susceptible dogs with the object of inducing immunity against natural infection in distemper.

Although great claims have been made in favour of these various vaccines and sera, there is at the present time none on the market that have given the profession those results which the inventors claim for them, nor given the dog-owning public that confidence in their utility which was expected. F. Richter* of Dessau has given several of the recent productions an extensive and careful trial, including that of Lignières, Phisalix, Copeman, Gaus, and Piorkowski, and pronounced against their utility. Sewell and Hobday declared that those of Copeman and of Phisalix did not give immunity. The writer, a few years ago, after giving both Lignières' and Piorkowski's sera a trial in several outbreaks of distemper, came to the conclusion that they were useless. Moore's toxin has not given everybody satisfaction, although it is well advertised among dog-owners. McFadyean concluded that a method for bringing about artificial immunity is yet to be discovered. Carré's investigations appear to support these adverse opinions. But is it not possible to vaccinate against or to overcome complicating secondary infections which bring about the majority of serious complications, and account for nearly all the deaths from this disease ? Small-pox and rabies are two diseases that can be vaccinated against, and yet their etiology has not been settled. Both Jenner and Pasteur had practical minds, and discovered preventive means for diseases which have not been thoroughly elucidated.

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* F. Richter, *Die Hundestaupe*, Dessau, 1908; and *Revue Générale de Méd. Vét.*, 1909, tome i., p. 438; *Journal of Comparative Pathology and Therapeutics*, 1904, vol. xviii., pp. 240 and 274; *ibid.*, 1908, vol. xxi., p. 238; *Vet. Record.*, 1904-05, vol. xvii., pp. 266 and 380; *Veterinary News*, 1904.

Should any readers, however, be desirous of obtaining further information on this subject, the writer would refer them to his paper on "The Immunisation of Dogs against Distemper," in the *Veterinary Record*, December, 1904 ; also his article "On the Etiology and Some of the Phases of Canine Distemper," in the same journal, December 19, 1903 ; to the Proceedings of the National Veterinary Association, London, 1902 ; to Hoare's "Veterinary Therapeutics," 1906 ; and to *The Foxhound*, July and October, 1911, and January, 1912.

TREATMENT.

This may be conveniently subdivided into (1) *Hygienic treatment ;* (2) *dietetic treatment ;* and (3) *medicinal treatment.*

1. Hygienic Treatment.—Especial attention should be given to the suitable housing of a distempered patient. The animal should be placed in a well-lighted *room,* shed, or kennel, which can be well ventilated without allowing any draughts to play on the sick animal, particularly during damp or wet, boisterous weather. Draughts of cold air are very injurious, and frequently set up pneumonic or nervous complications when these are not already in existence. The *floor* should be well strewn with sawdust, small chips, peat, chaff, etc. These, being good non-conductors of heat, prevent coldness of the feet and body should the animal get out of its bed and lie on the ground. They also absorb the moisture from the urine, fæces, or vomit, and can be easily removed, and, being very fine or short, do not impede movement like straw or hay littered on the floor.

The *bedding* may be composed of fine shavings contained in egg or orange boxes, or large soft sacks filled with straw or hay may be used. When the boxes get soiled they should be broken up and made firewood of, and replaced by fresh ones. Any soiled material should be removed as soon as possible, or at least twice a day.

The *temperature of the room* should, if possible, be maintained between 55° and 60° F. Distempered dogs do much better if they are not subjected to an alternation of temperature in an artificially heated place. It seems that a calm state of the atmosphere of the room is of more importance than its temperature.

During inclement, chilly, or cold weather, the sick animal should be enveloped in a *woollen coat,* which may be made out of either house-flannel, old rugs, left-off clothing or blanket, or Gamgee tissue, and when it gets soiled or torn it should be burnt and a fresh one applied.

The clothing should be made on the waistcoat principle, so that the lower portion of the neck, chest, and a part of the abdomen, as well as the whole length of the other part of the surface of the body, is covered. There should be two small openings made through which to pass the animal's two fore-limbs, so as to prevent the coat slipping backwards and getting removed. The free edges passing over the back should lap over one another for 4 or 5 inches, and these should be fixed together by inserting a few safety-pins along the whole length, not on top of the back, but along the side. To prevent pressure sores and discomfort to the animal, the clothing should be removed at least twice in twenty-four hours.

On no account should a dog suffering from distemper be exercised, or even permitted to run about the house, as fatigue and excitement arising therefrom often induce complications, and nearly always depression. The human physician orders his patient suffering from one of the infectious maladies to bed ; long experience has taught him that this is the best and safest course, as it renders his patient less prone to develop complications, and nearly always assists towards a recovery. The opposite has generally been adopted in veterinary practice, and, in consequence, the mortality has been much heavier than in human practice. Too early exercise before the convalescent stage is completed accounts for a great number of deaths, and also numerous cases of convulsions and chorea.

However, the writer would like to point out that in the case of puppies of the heavier or lymphatic breeds, if they are not allowed a certain amount of free movement, they are liable to develop rickets and become hopeless, unsightly cripples, and consequently worthless from a commercial point of view.

Therefore, of the two evils it is better to run the risk of the puppies dying from distemper by allowing exercise than to have them developing rickets by denying them exercise, which should be taken voluntarily rather than forcibly. In order to carry this out without unduly submitting the puppies to unfavourable conditions towards recovery, they should be placed in a large closed barn or shed, the floor of which should be well covered with a thick layer of pine sawdust, chaff, peat, or short straw, and allowed their liberty to wander about in it at their will, and kept there until they are quite well.

In the case of a *veterinary infirmary* each dog should be isolated in a separate pen or partition, so as to prevent a mild case becoming re-infected or hypercontaminated by another case of greater virulence coming from another kennel or locality. Some of the complications—especially certain pneumonic types—seem also to be transmitted to milder and perhaps hitherto uncomplicated cases. If, however, it is not convenient to isolate each dog in a separate pen or compartment, it would be advisable to clear out one lot of cases as soon as they are well enough before a fresh lot is admitted into the same place, in order to avoid hypercontaminating the first lot by the new-comers.

The floor may be sprinkled or sprayed with cresol (acid. cresylic.) or vapophen, both of which will drive away fleas, give off a somewhat pleasant odour, and, when inhaled, act as a mild, stimulating antiseptic to the respiratory mucous membrane.

The toilet of the sick animal should be attended to. The coat should be gently brushed once at least in twenty-four hours. The eyes and nostrils should have the discharges removed from them frequently by means of warm water and a piece of lint, and then be smeared with boric ointment. The outside of the mouth and around the anus should be sponged and then dried. The mouth and teeth should be sponged out or brushed with a solution of hydrogen peroxide two or three times a day. The ears should not be overlooked.

2. Dietetic Treatment.—The dietary, along with the hygienic care of the patient, plays a more important part than the medicinal treatment, and neglect of the two former cannot be counterbalanced by the last, however so selected or rigidly applied. During the earlier stages, especially when there is an elevation of temperature and before the disease reaches its crisis, the system should not be encumbered by a lot of highly nitrogenous food, that is likely to overtax the digestive system, which is often already the seat of the disorder. The animal has very little, if any, appetite. This is an indication that it is prompted by Nature not to take into its system what is likely to prove injurious to its well-being. Nearly everybody, however, considers loss of appetite a sign to be fought against vigorously by forcing more food upon the patient's unwilling stomach than it would demand if in health, when, in reality, it should be a signal to offer the animal what it will take voluntarily, and to avoid the forcible administration of nourishment.

As milk contains all those elements necessary to sustain life and to build up the system—at least, for a certain time—is not an irritant to catarrhal mucous membranes, and is generally easily digested and readily assimilated, experience has shown that it is the best staple diet for the distempered dog, which usually partakes of it voluntarily after a little coaxing. If, on the other hand, the appetite be in total abeyance, small quantities of milk should be repeatedly poured gently into the buccal pouch ; or the raw white of an egg might be tried. Sometimes when the milk is slightly thickened with ground rice or arrowroot it is swallowed more readily without inducing cough. Should the stomach reject it by vomiting, it should be given in smaller quantities still, and if again rejected, it should then be mixed with an equal quantity of aerated soda-water. Still, if there be only a little appetite left, it is advisable not to force any nourishment upon the animal too readily, as even a small quantity taken willingly will be far more beneficial than any amount of forced feeding. The forcible administration of food is often injurious, or at least causes nausea, and destroys what little appetite the animal has left. If the appetite be good, the milk may be boiled with bread or rice, and the animal allowed as much as it will take. As a rule, however, the milk is readily lapped up, but the bread or rice left behind. Sanatogen may be given for a change.

If the animal has been ill some time, and the appetite is still in abeyance when the temperature has dropped to normal, it may be considered advisable to give as a change a well-made concentrated beef-tea, chicken-jelly, or mutton-broth. One, however, should not overlook the fact that meat extracts, in the presence of a moderate but persistent diarrhœa, are very liable to increase the frequency and quantity of the dejecta, thereby further irritating the intestinal mucous membrane, and, in consequence, further weakening the animal's system. If the appetite should suddenly improve in a critical case of distemper, and the animal crave for food after the appetite has been in a state of total abeyance for some time, it is advisable to be cautious, and not to allow a large quantity at once, but to give it gradually, so as to avoid any sudden relapse to a grave state, or, perhaps, to induce convulsions. But although it appears very probable a sudden change from fluid to solid nourishment may set up convulsions or bring about a relapse, it is quite possible these latter conditions are preceded by an intense desire for solid aliment. This condition is too frequently due to a sudden cessation of a cough symptomatic of an underlying broncho-pneumonia, or of a profuse diarrhœa, and is a signal that there is a transference of morbid energy from the lung or intestine to the brain. This process simulates that of a counter-irritant artificially applied, only that in the case of the natural metastasis the organ irritated is a vital one (the brain), whereas in that of the artificial counter-irritant the organ to which it is applied is on the surface (the skin).

When it is deemed necessary to allow a return to a more solid diet, bread soaked in mutton-broth or beef-tea should be offered in small and repeated quantities for a day or two, and after this we may order scraped, minced, or finely chopped raw beef, the quantity being gradually increased according to the tolerance of the appetite or stomach. This may be supplemented by raw eggs beaten up in milk and thickened with biscuit, toast, or arrowroot. Should there be cardiac failure or fainting, small quantities of brandy should be added to the food.

But there are a great number of instances where dogs never go off their appetite for solid food, and provided there is no hyperpyrexia nor loss of condition, nor serious symptoms present, raw or cooked meat, boiled rabbit, biscuit, etc., may be given with benefit.

3. Medicinal Treatment.—When distemper is recognised in its initial stage (which is not often the case), and put under good hygienic and dietetic conditions, as outlined above, the majority of such cases would recover without much, if any, medicinal treatment whatever. Unfortunately, the practitioner is not, in nineteen out of twenty cases, called upon to intervene until the disease is far advanced, or perhaps has become complicated with serious lesions of vital organs, or, what is not rarely the case, when the animal is *in extremis*. The dog-owning public then think that the veterinary surgeon should be able to remedy by some mysterious power what their carelessness or ignorance has brought about. It would appear, from what one hears or reads in papers devoted to dogs, that this section of the community is as gullible and as self-satisfied in its discriminatory powers as it was in the early part of the nineteenth century, when Blaine, Youatt, and others, complained of sportsmen, hangers around mews, and such-like, who boasted of cures or the special virtues of this or that remedy, but, notwithstanding which quite fully one-third of the canine population succumbed to distemper. Even at the present time we have those who believe in the virtue of a "bent halfpenny," "a piece of tarred string" tied round the neck, a daub of tar on the nose, Christian scientific treatment, and such-like humbug. Our papers abound with mendacious advertisements claiming mysterious powers for some concoction or another. It seems the more ignorant such advertisers are of physiology, pathology, and clinical knowledge, the more confident they become in pushing their wares before the notice of the gullible members of the community. Much torture is

unwittingly inflicted on dogs by the administration of patent "cure-alls" and so-called specifics for distemper. These, in reality, are often responsible for the delay in sending for early professional assistance, and the practitioner frequently finds that his patient has been dosed *ad nauseam* with a combination of irritating agents which aggravates the existing diseased condition, and has the effect of altering what might have been a favourable case under rational treatment into a hopeless one. Many a case of distemper has terminated fatally in consequence of the owner taking the advice of some "doggy" friend, and treating the animal for "worms" by administering one of the many irritant concoctions advertised for the cure of what often is an imaginary affection in the dog. Unfortunately, dog-owners do not yet appreciate the importance of a correct diagnosis, clinical skill, and early professional attendance, but prefer to be led by cleverly-worded advertisements and the information derived from the veterinary inquiry column of a sporting paper.

During the early stage of distemper, when there may be an elevation of temperature, but no complications, it may be advisable to administer aspirin in 2½ to 30 grains, according to the size of the dog, two or three times a day, until the temperature has become normal, when this drug may be replaced by ½ grain to 5 grains of sulphate, acid hydrochlorate, or salicylate of quinine. If there should be no diarrhœa, the above may be supplemented with a few small doses (from ¼ grain to 2 grains) of calomel, given during the intervals until the bowels are acted on; this agent also acts as an intestinal disinfectant, and thus prevents some of the complications so often arising from the intestine. In uncomplicated cases no further treatment is called for.

Should there be *diarrhœa* of a profuse and persistent character, accompanied with an elevation of temperature, there is no scientific nor practical reason why it should not be checked by the repeated use of bismuth subnitrate, given in large and repeated doses. The old bogey that if the diarrhœa be suddenly checked by drugs it will produce complications such as convulsions or meningitis is pure imagination on the part of those who are fond of trotting it out in order to substantiate their opinion. On the contrary, if the diarrhœa suddenly cease *spontaneously* after it has been profuse and persistent, it is a grave sign. As it is not a critical diarrhœa, it should be checked, or at least moderated, so as to prevent rapid emaciation and exhaustion of the animal. If, however, it cannot be checked with such agents as repeated large doses of bismuth, it usually ends in death. Moderate diarrhœa, unaccompanied with much wasting, is not of serious import, but, nevertheless, it should receive attention, so as to prevent further complications, such as enteritis, ulceration of the bowel, straining, dysentery, or even invagination or prolapsus recti. When the diarrhœa is got under control, salicylate of bismuth, β-naphthol, and powdered wood charcoal, may be given in a keratin-coated capsule, morning and evening, as a substitute for the bismuth subnitrate. Should there be tenesmus, it must be overcome by the internal administration of chloretone, chloralose, or morphine; the last may also be hypodermically introduced, if considered advisable. The whites of raw eggs beaten up in a little water are sometimes very useful, and act as a demulcent as well as a food. Frequently the appetite is so good that remedies will be readily taken with the food, and this method is far preferable to forcing medicines on the animal.

In some cases there is a repeated vomiting of glairy albuminous-looking fluid or gastric mucus, which is generally an indication that the stomach is the seat of catarrh. This may be controlled or overcome by careful dieting and by the administration of bismuth carbonate. If it does not respond to this treatment, chloralose, chloretone, or opium, may be tried, with the repeated administration of small doses of Brand's essence of beef (jelly), small pieces of pure ice, and aërated soda-water. Counter-irritation, leeches, or the hot-water bottle over the region of the stomach, is sometimes advisable. A hypodermic injection of apomorphine sometimes gives speedy relief when other agents have failed.

Dysenteric diarrhœa, the dejecta containing blood and mucus, indicating an intense congestion, and even an abrasion or ulceration of the mucous membrane of the posterior or so-called large bowel, which is generally accompanied with a more or less marked straining, may be held in check by the use of large doses of the liq. morph. et chloroformi (B.P.), bismuth subnitrate, morphine, or chloralose. Sometimes it is advisable to insert carefully into the rectum a soluble suppository composed of cocaine, morphine, and acetate of lead.

In some cases, after all the acute symptoms have disappeared, and convalescence seems in a fair way to be established, there remains a chronic diarrhœa, which often persists for weeks and months after an otherwise complete recovery. This condition frequently proves obstinate to ordinary treatment. The writer has observed such a condition to be controlled, and finally overcome, with a mixture composed of infusion of cloves, logwood, and dilute sulphuric acid. Tannigen, tannalbin, or coto-bark, may be given for a similar purpose.

The principles for treating diarrhœa and also vomiting in distemper are to allay irritation of the mucous membrane, check peristalsis, abstain from exercise or excitement, avoid laxative or irritating food, such as meat or some of its extracts, and to induce alimentary antisepsis by rendering the secretions unsuitable for the development of deleterious bacteria.

Should the action of the posterior bowel remain in abeyance in consequence of the animal partaking of only a little fluid nourishment or rejecting it by vomition, the condition need not cause alarm, although the owner may regard it as serious. An enema of warm water, however, may be given every third day, so as to remove the small amount of fæces or, rather, dirty mucus from the bowel.

In case the *mucous membrane of the mouth*, including that of the cheeks, gums, tongue, etc., becomes *the seat of ulceration*, with its often resulting gangrenous sloughs, which may extend to the whole thickness of the tissues forming the angles of the cheeks, and also to the underlying bony tissue of the gums, prompt attention is necessary in order to mitigate its evils, or even to prevent the occurrence of the lesions. With this object in view, the mouth during the course of distemper should be daily inspected, and the teeth, which are generally covered with a greyish rusty or dirty-brownish scum, should be brushed or washed two or three times a day with solutions of peroxide of hydrogen, a solution of quinine acidulated with hydrochloric acid, Sanitas, or permanganate of potassium. Young dogs shedding their temporary teeth should have all the loosened ones removed, in order to prevent ulceration of the gums, gangrenous sloughs, or necrosis of the alveolar processes, which, in the presence of a coexisting debilitated condition of the system, often brings about death from septicæmia. Should the animal recover from the advanced gangrenous state of the mouth, the necrotic underlying bony alveoli should be left alone, as by a natural process the necrotic plate of bone soon exfoliates, and the remaining breach becomes rapidly filled up by granulation. Youatt, more than eighty years ago, advised for this gangrenous condition and the putrid type of distemper the administration of yeast, which to-day is prescribed in the form of antigourmine, nuclein, nucleinic acid, and similar preparations bearing various names.

The nostrils generally become plugged with a quantity of mucopurulent, purulent, or bloody purulent material, which often becomes dry and adheres to that region. In order to enable the animal to breathe more freely, this secretion should be removed several times a day with a swab of cotton-wool and warm water containing Sanitas or hydrogen peroxide. The swab should be burnt after use, and the nostrils smeared with vaseline or boric ointment.

When the laryngeal or the laryngo-tracheal mucosa becomes the seat of catarrh, it is usually accompanied with a more or less loud and harsh cough, which, although it is not a dangerous sign, but rather the opposite, is very irritating to the animal and annoying to the owner. To cut this short, the animal should be kept warm, and have warm, stimulating food or soup. The outside of the throat should be painted with tincture of iodine two or three times a day, or, what is more effectual and prompter in action, a few drops of oil of turpentine should be injected under the skin in that region by means of a hypodermic or serum syringe. After a few hours the cough ceases altogether, and an abscess gradually forms, which in a few days becomes matured, and if not opened, bursts of itself, leaving an abscess cavity which takes some time to heal. At the same time an expectorant mixture should be continued, notwithstanding the disappearance of the cough, as the latter may reappear after the effects of this rapid method of counter-irritation have passed off. The best mixture for this purpose is one composed of the syrups of codeine, apomorphine, ipecacuanha, and squills. In old dogs with cardiac valvular disturbance, tincture of nux vomica and digitalis may be conveniently added to this mixture. When the *catarrh* has extended to the *bronchial tubes*, a mixture composed of carbolic acid, spirits of chloroform, syrup of squills, ipecacuanha wine, and syrup of tolu, should be given instead of the one containing codeine.

The *broncho-pneumonia* which so frequently complicates distemper, and is the cause of the majority of fatal terminations, requires to be treated with promptness—that is, if it can be recognised, which, unfortunately, is not always the case, as, in the absence of a cough, convulsions or meningeal symptoms frequently mask its presence. Expectorants, with ammonia and alcohol, should be administered, and if a high temperature is present, aspirin or the ammoniated tincture of quinine may be given alternately with the expectorants. The chest should be well enveloped with a layer of Thermogene wool, and the sides painted twice daily with tincture of iodine; or the subcutaneous turpentine counter-irritation already mentioned may be employed, the site selected being the front of the chest.

On no account should the *short hacking cough*, which is so distressful and so frequent, occurring in fits of more or less duration, be stopped or checked, as it is purposeful in function to relieve the bronchioles of the source of irritation. It may be assisted in this direction by inhalations of steam containing compound tincture of benzoin, vapophen, or cresol (cresylic acid), oil of turpentine or terebene, or equal parts of cresol and oil of eucalyptus. In very severe cases in valuable animals the inhalation of oxygen should be tried. A hypodermic injection of apomorphine sometimes gives speedy relief.

In the *very acute congestive* or *hyperæmic stage of pneumonia,* which accounts for a very large mortality in acute virulent outbreaks of distemper, treatment must be prompt in effect and early adopted in order to obtain any good results. Powerful stimulants, whether internal or external, often bring about a satisfactory termination. Nux vomica, digitalis, ether, and ammonia, should be given internally, and tincture of iodine, oil of mustard, or strong turpentine liniment, applied to the sides. The normal saline solution should be introduced under the skin by gravitation from a douche arrangement. Inhalations of oxygen are also useful.

In those cases in which the *pneumonia* is of the *croupous type*, often becoming well advanced before it is recognised, in consequence of the frequent absence of cough, and very little, if any, disturbance of the respiratory rhythm, the acetate and iodide of ammonia may be given, and the sides of the chest painted with tincture of iodine until tenderness is produced, when it should be discontinued.

The *nervous complications* arising at the initial stage or during the course of the disease, but generally towards or even during the convalescent stage or succeeding it, are not commonly treated with success. Indeed, the majority of such cases succumb, are left incurable, or become permanently maimed.

As to the *convulsions*, which very rarely arise at the commencement of the malady, they may be checked or overcome by morphine, chloralose, chloretone, or chloral, or by the inhalation of chloroform. Their reappearance should be prevented by the bi-daily administration of bromide and iodide of potassium, dissolved in water, and then given in the food. These convulsions, however, may reappear when the disease reaches the critical stage, and are often the forerunner of paralysis. They are not so fatal as those that make their first appearance at the later stages of the malady.

Should the convulsions first make their appearance at the critical stage of distemper, or after a sudden cessation of cough (dependent upon extensive pneumonic lesions), or following a sudden spontaneous disappearance of diarrhœa, they are an almost certain indication that the nervous system has become infected. They are usually succeeded by cerebro-spinal meningo-myelitis, chorea, paralysis, etc., which are generally fatal in results or rarely curable.

Although bromides are generally recommended to check or control these so-called, but erroneously termed, "epileptic fits," they are in actual practice rarely of any benefit, but often seem to aggravate the condition. However, as medicinal agents of any kind seldom prove of value, the bromides may be given in conjunction with the iodides, this combination occasionally giving successful results. Chloralose, codeine, apomorphine, or morphine, may also be given, alone or in conjunction with the mixed iodide and bromide. They may be mixed with the food if there is an appetite and the symptoms are not urgent; otherwise it would be preferable to give the bromide and iodide and the chloralose orally, and the apomorphine, morphine, or codeine subcutaneously. If the convulsions are got under control, the mixed bromide and iodide should be continued in the food, but the other sedatives should be omitted.

When there is meningitis or cerebro-spinal meningo-myelitis, associated or not with chorea, paralysis, self-mutilation, or great excitement, the case becomes a hopeless one in ninety-nine instances out of one hundred, and unless the owner forbids it, the animal should be at once destroyed. If, however, the owner desires that some treatment should be adopted, counter-irritation, in the form of a biniodide, tartar emetic, or cantharides blister, or a subcutaneous injection of the oil of turpentine, directly over the occipital crest, may be carried out; or, where this is objected to, the ice-bag may be applied after the skin has been wetted or damped with cold water. Sulphonal, veronal, or hypnone, as well as the mixed iodide and bromide, may be given internally.

Uncomplicated choreic movements, which may occur in any group or involve the whole of the systemic muscles, including those of the eyes, ears, jaws, neck, shoulders, abdomen, quarters, or limbs, are likely to somewhat abate if the animal recovers, and the system regains to a certain extent its former tone. In a strict sense it is an incurable disorder, but occasionally the spasms disappear entirely, even after the symptoms have persisted for years. *On no account should nerve stimulants or tonics, such as strychnine or nux vomica, arsenic, phosphorus, etc., be administered, or electricity be applied, during the acute or early stage of chorea, as such agents would only stimulate the already overstimulated nerve centres, and probably increase their degeneration.* These agents are only indicated in this disease when all the active phenomena of distemper have disappeared and convalescence is established. The writer doubts then whether they are of any use, as from his experience better results are obtained by leaving the case to the *vis medicatrix naturæ.* The iodides, aspirin, and sodii salicylas, appear useful during the acute stage. Youatt speaks of nitrate of silver as being the sheet-anchor of the practitioner, if used in the early stage of chorea.

As to the *paresis, paraplegia,* or the various forms of *monoplegia,* the remarks made above upon chorea are here applicable. Such cases often recover without being tormented by any special treatment.

The catarrhal or purulent condition of the conjunctivæ (*conjunctivitis*) is often moderated by the repeated use of chinosol, bichloride, or biniodide of mercury lotions (1 in 4,000), and then afterwards smearing the eyelids with boric ointment or vaseline. Should pain be evinced and accompanied by ulceration of the cornea, a few drops of a solution of stovaine or cocaine should be dropped into the conjunctival sac several times a day ; or the eyes may be simply smeared with cocaine ointment as often as is deemed necessary. If the ulceration should threaten to perforate the anterior chamber, it would be better to puncture the floor of the ulcer with a cataract needle, and allow a certain amount of the aqueous fluid to escape, so as to prevent a natural but wide outlet, which would involve the iris and destroy the sight. When this has been done, or in case there is irido-cyclitis or vascular keratitis present, a few drops of atropine solution should be instilled into the conjunctival sac. If, on the contrary, the ulcer be near the sclero-corneal junction, a solution of eserine salicylate should be used instead of the atropine. There need be no apprehension of increased intra-ocular pressure from the use of atropine, as this agent only increases this pressure when it is already present from glaucoma. On the contrary, eserine reduces intra-ocular pressure only when the latter is abnormal, as in glaucoma.

No violent remedies, such as the application of caustics or cauterising the ulcers or their subsequent granulation (proceeding to absolute resolution), by the use of nitrate of silver or by the thermo-cautery, are required, as, by leaving these serious-looking lesions to Nature and a little simple treatment, the majority will recover with little or no blemish remaining. The resulting blemish, if sluggish, might be assisted by the gentle use of yellow oxide of mercury ointment.

The *exanthematous eruption* should not be checked, but rather encouraged by warmth and warm stimulants. It should be left alone unless it causes itchiness or produces moist ulcerous surfaces. In the former case it should be carefully smeared with sulphurated vaseline, taking care not to create a chill. In the latter a mixture of French chalk or fuller's earth and boric acid may be dusted on the moist or ulcerated parts.

The *deep suppurative changes or small subcutaneous abscesses*, generally containing bloody pus, should be opened and the cavities washed out with a 1 in 1,000 biniodide or bichloride of mercury solution, or hydrogen peroxide, and then dusted with boric acid and fuller's earth. This should be repeated daily.

The *vesico-pustular eruption* on the under surface *of the flap and internal lining of the middle ear*, causing what is generally termed " canker," is moderated by dusting the affected surface with equal parts of kaolin and boric acid. A few dustings with this application bring about complete recovery in a very short time.

The *sheath and the penis of the male* and the *vagina* of the bitch, and also the *anal glands* of both sexes, are frequently the seat of *a catarrhal or purulent discharge*, which often flows in such abundance that it soils the parts with which it comes in contact. This may be moderated or removed by daily injections of chinosol, biniodide or bichloride of mercury solutions (1 in 4,000).

Distemper, being a specific disease, will run its course in spite of therapeutic interference; but by careful treatment it may be placed under favourable conditions, so that it runs this course unaccompanied by secondary infections or complications, which are, in the majority of instances, the cause of its frequently virulent character and resulting high mortality. Heroic treatment will not overcome any of the morbid conditions, and generally the less medicinal treatment adopted the better it is for all concerned. During its course, and especially in the convalescent stage, exercise, exposure to chills, and improper diet, should be specially avoided.

MEDICO-LEGAL ASPECT.—Frequently dogs are purchased with a guarantee that they are over distemper, and should such develop distemper after the sale, the veterinary surgeon may be called on to answer the following question: *Has this dog had distemper prior to the sale?* In the case of a young dog under six or seven months old not showing any lesions, but being in a good state of health, and having a good coat, this may be answered in the negative. If, on the contrary, the animal has chorea, which always arises from distemper, or has an erosion of the enamel on the teeth, which occurs from distemper during the period of the eruption of the second dentition, *the query may be answered in the affirmative.* If there should be any ulceration of the cornea, old cicatrices, nebulæ, or leucomæ, *one may doubtfully give a positive opinion,* as in the majority of instances these conditions of the eye arise during the course of distemper. In the absence of these points there is no evidence to assist the practitioner, unless he actually saw the animal suffering from the disease, whether or not a given dog has had or has not had distemper. On the contrary, dogs may sometimes have the disease a second, third, or a fourth time, even during the same year, but never under six or seven months of age, without showing some traces of it. Often at the time of sale the animal shows symptoms of distemper, or is incubating it in his system, so that it develops in a few days to a week or so after he has been in the purchaser's possession.

DISTEMPER IN THE CAT

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Feline catarrhal fever; Feline contagious catarrh; Typhus fever; Influenza; Contagious gastro-enteritis; Contagious snuffles; Parotitis felis (Darwin).

French.—Maladie des chats; Maladie de jeune chat; Pasteurellose féline.
German.—Staupe der katzen; Katzeseuche.
Italian.—Cimurro.
Argentine Republic.—Moquillo.

HISTORY.

According to Fleming,* nineteen widespread outbreaks of this malady have been recorded, occurring from A.D. 1414 to 1832. In 1779 it was observed over a wide tract of Europe, and notably in Denmark, France, Germany, and Italy, where it affected nearly all the cats. During the years 1782, 1783, and 1784, Barrier† encountered it on the farms in the neighbourhood of Chartres, killing nearly all the animals seized. In 1796 it was most disastrous, when the cats in England and Holland were generally afflicted by the disease, and in the following year, when it had spread over Europe, it broke out in America. In 1803, according to Delabere P. Blaine,‡ it again appeared in England and over large parts of Europe. Hurtrel d'Arboval states it was seen in the Brie in 1820. During 1835 it was observed in France, in several places in the Brie, chiefly in the canton of Claye. Since 1890 to 1912 the writer has every year encountered it in various parts of London, and it is frequently seen at the present time in many large cities and various parts of the country, either in an isolated or an epizoötic form. In several large catteries for lost and starving cats it is always enzoötic. It would appear that the reason it is so prevalent at the present day is due to the fact that a greater interest is taken in the improvement of the breeds of these animals, the parents of which are artificially selected, and they, together with their offspring, are unnaturally nurtured in surroundings unfitted for them, so that the system has become weaker and less resistant to the disease. Another reason is that cat-shows and cats' homes have increased, and act as a further means of keeping up the disease and spreading it all over the country.

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* Fleming, "Animal Plagues."

† Landrin, "Le Chat," Paris, 1894.

‡ Blaine, "Diseases of Dogs," 1817.

NATURE.

This is a contagious and an inoculable malady, probably due to an ultravisible or filtrable virus. According to Krajewski, Laosson, Lignières, Jess, and others, the virus of canine distemper can be transmitted to cats and reciprocally, thus proving the identity of the two maladies. Blaine informs us in a work "On the Diseases of the Dog," written in 1817, that "little similarity as there is between the cat and the dog, yet they partake of this disease in common between them, and each is capable of giving or receiving it from the other." But this eminent master of canine pathology, in a later work, published in 1841,* does not speak with such confidence on this subject, for he says: "The cat certainly is occasionally subject to a virulent coryza; but I am not aware that the affection bears the other specific characters of the complaint [distemper], such as its obstinate and fatal diarrhœa and liability to be translated to the nervous system, and its contagious nature." Be that as it may, the writer has never observed natural transmission of distemper from the dog to the cat, and *vice versa*, and this is the experience of Sewell, Hobday, and many other veterinary surgeons in this country.

Not much is known of the microbiology of this malady beyond the fact that Lignières isolated a pasteurella or organism of the fowl-cholera type, was able to cultivate it outside the body, and to induce with cultures thus prepared all the symptoms and lesions found in the natural disease. But grave objections have been taken to this theory. Gaertner, in 1909, studied the disease, and isolated an organism of Lignières' pasteurella group. Jess also isolated a microbe which he cultivated and found to be pathogenic to the cat. M'Gowan studied in 1910 a disease of dogs and cats clinically resembling in many points that of distemper in these species respectively. He isolated and studied a cocco-bacillus which resembled Copeman's organism in the majority of aspects, and was transmitted and recovered from either animal reciprocally.† Nevertheless, an Austrian worker demonstrated an ultravisible or filtrable virus derived from a pharyngeal disease, which was probably only a type of feline distemper.

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* Blaine, "Canine Pathology," fourth and last original edition, 1841.

† *Journal of Pathology and Bacteriology*, 1911, vol. xv.; *Vet. Journal*, January, 1912, p. 7.

PROPAGATION.

The principal sources of the infection are cat-shows, catteries (especially those belonging to people who exhibit), veterinary infirmaries, homes for lost and stray cats, and institutions that take in these animals as boarders while their owners are out of town. The cat-dealer's shop is not free from blame; many newly-purchased kittens develop distemper a few days after purchase, contracted, no doubt, at the dealer's shop. Many cases have been traced to the cattery where the queen (female) has been sent to stud. Hampers, cages, etc., coming from infected catteries are so many media of contagion.

PREDISPOSING CAUSES.

Although the disease may be seen at all times of the year, it is most prevalent during spring and autumn, especially if the weather is boisterous, changeable, and wet. It is, however, more commonly observed from the middle of February to the middle of April, when the March winds prevail and the cats are "rutting,"* when they congregate. It is, however, not rare from August to October, and this is chiefly due to the fact that in large towns residents (when leaving home for the seaside, country, or abroad) send their cats to be taken care of during their absence from home, to some institution or place, where they (the cats) become infected by some animal suffering from the disease or from a cage in which some diseased creature has been confined.

The Effect of Dampness.—Moisture of the atmosphere favours the development of distemper. Wet following a long spell of very dry weather, continuous dampness, or rain, all predispose an animal to take on the disease. Where catteries or homes for lost and strays are continuously being washed out and not properly dried (especially in damp weather) before the cats are installed into the rooms, the malady is very prevalent.

Overcrowding.—Where too many cats are crowded into a given space, especially if the building is badly lighted and not very well ventilated, this is favourable for the contamination of the inmates.

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* Practitioners should be careful in castrating cats during this period, as in consequence of the "rutting," owners take their cats to the veterinary surgeon to be castrated, and the animals frequently have the infection in the system, which may develop a few days after the operation, and the illness or death is erroneously attributed to the operation. The same remarks apply when a cat has been just purchased at a cat-shop.

RECURRENCE.

Many cats are resistant at one time to the infection, others develop the disease in a mild form, and yet others are attacked severely; but this does not always prevent them from having it again at some future period. Indeed, the writer's experience is that a cat once affected is frequently more susceptible to future attacks, just like the human subject is to influenza, and he has known instances in which an animal has succumbed to the disease after having recovered from two, three, or more previous seizures. However, many cats have a natural immunity to the complaint; others may acquire this immunity and resist future infections. It is much more virulent in localities where it has not occurred previously, or is only encountered after very long intervals.

PERIOD OF INCUBATION.

This varies according to the degree of potency of the virus, the susceptibility of the animal, and the occasional causes, such as the weather, feeding, and housing. The average period varies from one to three weeks, although the disease may develop in four or five days after exposure to the infection.

SYMPTOMS.

During some seasons the disease takes on one form, and at other times it differs entirely from previous manifestations, leading one to think that there are several different maladies classified under one generic term. One year it may appear as a contagious catarrh, in another as an infectious sore throat, at other times as a bronchitis or a broncho-pneumonia, and at still other periods as a contagious gastritis or gastro-enteritis. Frequently all these forms may coexist in one outbreak, and often a single animal may show the whole of these manifestations.

For the convenience of description of the symptoms of this multiform malady, the writer believes it advisable to divide it into five principal forms, as follows:

1. *The catarrhal,* attacking chiefly the eyes and nostrils.
2. *The pharyngeal or tonsillar,* affecting the region of the throat.
3. *The pulmonary or thoracic.*
4. *The abdominal or gastro-enteric.*
5. *The cachectic or wasting.*

1. The Catarrhal Form.—This is the type of distemper which is generally seen in the cat, and is the least fatal of any.

Symptoms.—The first noticeable symptoms are a watery discharge from one and sometimes from both eyes, the lids of which may be partially or completely closed, so as to hide the front of the eye, also a frequent licking of the upper lip and nose, as if they were parched and burning, is observed. After a day or two the inner lining of the eyelids may become very much reddened and swollen, giving rise to a yellowish-white or greenish-white thick discharge, which adheres to the lids and seals them together. In certain enzoötic outbreaks the conjunctiva becomes so swollen that the mucosa is everted, and hides the eyelids, and the greater part, if not the entire, eyeball (pink-eye form). There may also be shivering fits, a dull open coat, and a great desire for warmth, this being so intense that in some cases the animal gets under the grate when a fire is in it. There is frequent sneezing, followed by a snuffling kind of breathing; the nostrils discharge a thick, ropy, whitish or greenish matter, which clings to their openings, and very often closes them up. When the pharynx or larynx becomes involved in the catarrhal process, there are frequent fits of coughing.

The appetite is diminished or absent, but thirst is, as a rule, well marked. There may also be seen at times vomiting, diarrhœa, or constipation. Emaciation is generally gradual and slight, but may sometimes be rapid and extreme, varying according to the mildness or severity of the symptoms. The breathing is not altered to any extent in the majority of cases, but in a few instances it becomes frequent. The temperature is at the earliest stage elevated a few degrees, but soon falls; it may oscillate, and is often found to be normal. The body and limbs feel cold to the touch, and the former sometimes gives off an offensive odour. The tongue, lips, cheeks, hard and soft palates, and gums (especially around the necks of the teeth), become deprived of epithelium, either in minute points or large surfaces, and are sometimes more or less deeply ulcerated. Occasionally the eyes become the seat of ulceration, which, in rare instances, terminates in perforation of the cornea; at other times they become affected with a severe inflammation, which extends to the whole eyeball, and destroys this organ. There is at times extreme dulness or drowsiness, and the animal seeks dark corners or gets under the furniture. Many cats, however, from sheer nervousness, especially in a strange place, avoid the fire, and seek obscure or lofty positions.

Recovery generally takes place within a fortnight or three weeks, but death may supervene within twenty-four to forty-eight hours from the commencement of the attack.

2. THE PHARYNGEAL, TONSILLAR, OR THROAT FORM.—This is the most deadly manifestation of feline distemper.

Symptoms.—The first symptom to attract attention is the drivelling of clear ropy saliva from the corners of the mouth. The animal sits upon all four of its limbs in a crouching position; there is a frequent gulping movement, and a sound is emitted from the throat as if there was an attempt to swallow the thick ropy saliva which clings about the mouth and pharynx; deglutition seems difficult or impossible; food is refused, but thirst is constant, although the animal seems incapable of swallowing; there is extreme weakness, great depression, and the cat appears indifferent to its surroundings.

On examination of the pharyngeal region, it is found swollen and painful, the glands are enlarged, and there appears to be a gurgling noise at each inspiration and expiration. On inspection of the mouth and back of the throat, the tongue and pharynx are found to be covered with a thick, ropy, bubbling saliva; the mucous membrane is swollen and deeply congested, and the soft palate is of a pinkish or even dark reddish arborescent appearance, due to the congested state of the small vessels. The pillars of the fauces are also swollen and congested, and the tonsils re very much enlarged and heightened in colour, and stand well out and above their crypts. Sometimes ulcers appear on the hard and soft palates. After a day or so the depression increases; there is a discharge from the eyes and nostrils, which appears at first as a clear, viscid material, and afterwards becomes yellowish or dirty green in colour, and, should the animal live long enough, ultimately bloody, in consequence of the irritating effect produced on the mucous membranes and surrounding skin of the eyes and nose. There may also be a catarrhal or purulent fœtid discharge from one or both ears; but this is quite exceptional, and is mostly seen in cases having a fatal termination.

If the prostration be very great, and if there be rapid loss of weight and condition, and the discharge from the mouth, nostrils, and eyes becomes very fœtid, with a total loss of appetite and no abatement of the other symptoms, a fatal termination is to be expected. Late in the complaint the pharyngeal mucus may become of a dirty colour and sanious in character; purple spots appear on the tongue, gums, and lips, and there is a moan or cry emitted at each respiratory effort; convulsive or clonic movements of the muscles of the temporo-maxillary region, of the shoulders, and of the thighs, set in, and death takes place from toxæmia.

The temperature is at first elevated, but when a fatal termination is to be anticipated, it falls several degrees below the normal.

3. The Pulmonary or Thoracic Form.—This form, although so common in the dog, is in the cat comparatively rare, and may appear from the outset as a distinct localisation, or follow or intervene during the course of one of the other forms.

Symptoms.—It may or may not be ushered in by shivering fits; the coat becomes dull and open; there is sneezing or coughing, or both may be present; tears run from the eyes, a serous fluid or mucus issues from the nostrils, and there is a great desire for warmth. The temperature is elevated, and varies from 102·5° to 106° F., not rarely running a typical course. The cough, when present, is frequent and rattling, harsh or sometimes dull in tone. On auscultating the chest, wheezing, rattling, blowing, rubbing, or splashing sounds may be heard. Emaciation is either gradual or rapid, thirst is generally well marked, but the appetite is diminished or absent.

The breathing is either quickened or the inspiratory and expiratory efforts may be prolonged, and accompanied or not by a moan or grunt. These symptoms are sometimes associated with fluid in the pleural cavity, which is generally evidenced by the pumping, lifting, or heaving action of the flanks. This effusion into one or both pleural sacs is either of a clear amber, greenish-tinted, bloody, dirty greenish or yellowish appearance, and sometimes of a fœtid odour. Besides pleurisy, which is occasionally encountered even in an enzoötic form, the croupous type of pneumonia, broncho-pneumonia, capillary bronchitis, or bronchitis, may be present, depending on the pulmonary structures involved in this form of distemper.

The pulmonary lesions may be slight, but yet the symptoms may be very severe. On the contrary, the symptoms may be comparatively slight, but the lesions may be very extensive. If the fever remains high, and the appetite is totally abolished, the pupils dilated, the breathing plaintive and very rapid, and prostration great, death soon takes place from failure of the heart, due to the action of toxins. In many cases, however, the fever may be very slight, yet death supervenes. In cases of pleuro-pneumonia in kittens and young cats, an inexplicable comatose condition sets in before death arrives.

4. The Abdominal, Gastric, or Gastro-Enteric Form.—This is more frequently met with than either the pharyngeal or pulmonary form, and may occur as a very acute and rapidly fatal manifestation, or as a subacute or chronic disease. It often accompanies the other forms.

Symptoms.—In *very acute* cases there is sudden vomiting of food, quickly followed by a frequently repeated ejection of a thick, slimy, and frothy mucus, and eventually by a thin, watery, serous fluid, which is often of an olive-green or greenish-yellow appearance. The thirst is intense, and no sooner is water sipped than it is expelled. There is frequent diarrhœa, the stools at first being pultaceous, then watery, leaden in colour, sometimes bloody, and very fœtid. The appetite is absolutely suppressed, and the animal becomes cold, semicomatose, and indifferent to its surroundings ; the facial expression is pinched, the eyes are semiclosed, the pupils dilated, the coat dull and open, and generally soiled by vomit or fæcal material, and on pressure over the region of the stomach or intestines pain is evinced by a semidelirious moan or cry, and death usually takes place in a few hours. A discharge from the eyes and nostrils is usually absent in this acute form.

In the *subacute* cases, in addition to a slight catarrhal discharge from the eyes and nostrils, there may be either vomiting or diarrhœa present, often both ; and in other instances vomiting and constipation. When the intestine is the principal seat of the lesions, vomiting is rare ; but diarrhœa is generally persistent. Thirst is well marked, and food is refused or partaken of sparingly. The animal is dull, and cries if moved or if the abdomen be manipulated. Emaciation is rapid, and the animal dies in a state of exhaustion.

In the *chronic* cases there may or may not be any catarrhal symptoms, but there is a chronic and persistent diarrhœa, and sometimes vomiting. The appetite is capricious or sometimes ravenous, the thirst moderate, emaciation gradual, and liquid fæces may be expelled on the least effort, as by coughing ; the fur or pelage around the tail becomes soiled, and, in consequence, the animal gives off a very offensive fæcal odour.

In some instances the breath becomes fœtid ; the teeth, gums, tongue, and lips, are covered with a dirty brown or greenish slimy material, and often undergo ulceration ; frequently the gum around the necks of the teeth is spongy, and bleeds on the slightest touch ; and occasionally the bony alveoli into which the teeth are inserted become exposed and undergo necrosis. Ulcers are at times found on the lips, inside the cheeks, on the tongue, and at the internal angles of the mouth formed by the meeting of the gums of the upper and lower jaws.

5. The Chronic, Cachectic, or Wasting Form.—This is sometimes encountered as a chronic wasting malady, not showing many symptoms beyond gradual emaciation, great weakness, intense thirst, ravenous or capricious appetite, and occasionally diarrhœa. At other times the animal goes off its appetite, sits about in a mopish manner, is disinclined to move, and has a staring and open coat ; the visible mucous membranes are pallid ; the membrana nictitans protrudes over the front of the inner portion of the eyeball, and the body becomes very light in weight.

Rarely in these wasting cases the skin becomes the seat of parasitic mange, and, in consequence, gives off an offensive mousy or mouldy odour. If treatment is not adopted early, death takes place, and on post-mortem examination the appearance of the cadaver simulates that of an animal having died from starvation. It may follow on the other forms of distemper.

Skin eruptions are rarely noticed in feline distemper, but sometimes young Persian kittens have a scabby eruption resembling ecthyma, the discharge of which mats the hairs together, and when the scabs fall off bare circumscribed abrasions remain, which soon heal.

Jaundice is sometimes observed, and then the case nearly always succumbs.

Abortion in the pregnant cat is frequently observed when suffering from distemper. In large catteries, where distemper may be enzoötic, nearly all the pregnant cats abort or have their offspring prematurely. (Fleming says abortion is very rare in the cat. This is not so ; when they are kept under unnatural conditions, no animal so readily aborts as the cat.)

The *ears* occasionally become the seat of acute catarrh or ulceration, and give rise to an offensive discharge. This complication is generally associated with the pharyngeal form.

The *cornea* of the eye is sometimes the seat of ulceration, which generally disappears without leaving any blemish or opacity. However, *deep ulceration*, with *permanent opacity*, *perforation of the cornea* and *prolapse of the iris*, and *acute panophthalmitis*, with *rapid destruction* of one or both eyes, occur, but far less so than is the case in canine distemper.

When the *eyes of young kittens* become the seat of catarrh, even before the eyelids have opened, or soon after, the eyeball is generally destroyed, and consequently the sight is lost. This form of catarrhal distemper occurring in kittens is enzoötic, and many she-cats are permanent carriers of the infection ; every litter they have becomes affected within three or four weeks after birth, although the parent is to all appearances healthy. It is a troublesome affection.

The *nervous* type, showing itself in the form of cerebral excitement, of convulsions, of chorea, of meningitis, or of paralysis, although sometimes seen, is very rare.

Death either occurs suddenly from convulsions, or rapidly from toxæmia, or slowly from exhaustion. When due to toxæmia, the animal either dies quietly in a comatose condition, or manifests clonic convulsive or twitching movements of the temporo-maxillary, shoulder, and hind-limb muscles, preceding death, or they may even be seen shortly after it. Frequently death takes place without any symptoms of the disease having been observed.

COURSE.

When this complaint is not interfered with by complications or death, it usually takes three to eight weeks before the animal recovers. However, in some cases the disease seems to have become aborted, and the affected creatures manifest very slight symptoms, and are well again within a week or ten days.

DURATION.

This, like the period of incubation, varies according to the degree of potency of the virus, the resistance of the animal, and the treatment adopted during the course of the malady. Some cats die quite suddenly, without much or anything being observed amiss with them before death ; others are ill for two or three weeks ; and still others linger much longer before they die or recover. In some cases the disease shows itself in so mild a form that it appears like an ordinary catarrh, and recovery is established within a few days.

MORTALITY.

This varies according to the age, the breed, the surroundings, and the degree of virulence of the infection. During certain seasons and certain epochs the mortality is greater than at other periods. The adult common-bred cat, nearly always out in the open, rarely dies from it ; but the same animals confined to a cage, cattery, or a house, perish at the rate of 25 per cent. The long-haired cat or cross-bred animals are less resistant to it, and as many as 50 per cent. succumb. Of the Siamese breed of cats, the mortality may reach 80 to 90 per cent. The younger the animal, the greater the death-rate ; yet, on the other hand, if old, very fat, or anæmic animals are deprived of exercise and fresh air, the mortality is quite as high. At some periods, however, nearly all the animals affected recover, whereas at other times most of those attacked die.

DIAGNOSIS.

In many instances this disease is mistaken for a simple catarrh, ordinary diarrhœa, " worms," or sore throat—a mistake, unfortunately, too often made where other cats are concerned. It is quite true that the first stage of distemper frequently resembles any of these simple complaints, which are not contagious, generally affect one out of several animals kept together, and run their course in a few days. In distemper, however, the disease runs a prolonged course, is very prostrating, in many instances fatal, and, beyond all doubt, is contagious. On the other hand, it resembles, at least in some of its features, *diphtheria* (see p. 317), which is also contagious, but shows the presence of false membranes on the soft palate, pharynx, larynx, and tonsils ; these lesions, however, are absent in distemper. Both distemper and diphtheria may be mistaken for a bone or needle or a piece of grass in the fauces, pharynx, or posterior nares, but a simple inspection of the throat in a good light will settle this. It may also be mistaken for some of the manifestations of tuberculosis (see p. 184).

PROGNOSIS.

Feline distemper is a most treacherous disease, and one cannot foretell the result of its effects. Many instances occur in which an animal appears to be on the right road towards recovery, when a relapse suddenly sets in and carries off the patient. If, however, the appetite is moderate, the emaciation not rapid or extreme, the diarrhœa not intense or too frequent, and no complications appear after the end of the first week, recovery may be anticipated. On the contrary, should the weakness be progressive and prolonged, the emaciation rapid and well marked, an offensive odour be given off from the body, the eyes sunken in their orbits, the pupils dilated, and the facial expression be haggard, death is to be expected. Again, relapses, which are so commonly encountered, early youth, obesity, complications, the breed of the animal, such as Siamese and long-haired varieties, especially very light-coloured animals, are generally unfavourable towards a recovery.

Chronic nasal catarrh, chronic pneumonia, phthisis, and persistent diarrhœa, may also give trouble after distemper has run its ordinary course, and will have to be reckoned with.

PROPHYLAXIS.

The old maxim, " Prevention is better than cure," should be carried out as far as possible by isolating all those animals that have been in contact with the infection. Animals coming from homes for lost and stray cats, from cat-shows, cat-shops, or from the stud, should be kept apart from those in the cattery for at least a fortnight or three weeks, in order to observe the early symptoms of the complaint. The place of isolation should not have any communication with the building or house in which the majority of healthy cats are kept. The baskets, cages, brushes, etc., should be thoroughly washed and disinfected before they are used again for sound cats. It should not be forgotten that persons attending to the sick animals may carry the infection on their hands, if they have not been previously well washed before tending the healthy.

When the inmates in a cattery have recovered, the place should be *thoroughly* scrubbed, disinfected, and, if possible, afterwards lime-washed or painted. Boiling water, soap, and soda, used with the aid of a scrubbing brush and "elbow grease," is a much more reliable method of removing infection than many of the so-called disinfectants, which frequently do not destroy the virus, but often injure the cats. After the habitation has been thus scrupulously cleansed, it may be well to disinfect it with a solution of chlorinated lime (1 pound to the gallon of cold water), which should be brushed all over the floor, walls, partitions, etc. Baskets, hampers, etc., should be served likewise. Metal and earthenware utensils may be boiled in a strong solution of soda. Before the cats are reintroduced into the place, the doors and windows should be opened for at least a week, and fresh air and daylight admitted, as they are most destructive to micro-organisms.

But whatever precautions may be taken with the surroundings, one cannot disinfect the living cat, which may, although apparently well, give off the virus indefinitely, even for years, as observed in the case of the cat which contaminates every one of her litters. This mode of infection cannot be too strongly emphasised.

TREATMENT.

The sick animal should be kept in a well-lighted, well-ventilated, but draughtless room, which should be dry and kept at a temperature never below 60° F. The floor should be covered with a thick layer of fresh pine sawdust, heaps of which should be placed in old tin trays, boxes, or old lidless coal-scuttles, for the convenience of the animals. Instead of sawdust, peat, ashes, gardener's peat, or dry mould, may be used.

Easily digested and nourishing food, such as milk, should be offered, and, if refused, gently administered in small and repeated doses to the animal. When the appetite is fairly good, $\frac{1}{8}$ to $\frac{1}{4}$ grain of calomel may be given two or three times a day, but should be stopped if vomiting or intense diarrhœa sets in. When the appetite is poor, $\frac{1}{2}$ grain of sulphate of quinine may be given three times a day for a lengthened period, until the desire for food returns. Should there be very high fever present, aspirin may be given in $2\frac{1}{2}$ to 5 grain doses. The eyes and nostrils should be bathed three times a day with a chinosol lotion (1 in 1,200), and then smeared with vaseline or boric ointment. If the throat be very much inflamed, the outside should be painted, after removal of the hair from ear to ear, with tincture of iodine or ethereal tincture of capsicum, three times a day, until soreness is produced. And as it is difficult and troublesome to paint the inside of the cat's throat, the following powder dropped on the tongue will act in a similar manner, and also as a tonic: quinine sulphate ½ grain, borax 1 grain, chlorate of potassium* 1 grain, powdered wood charcoal 1 grain. To be repeated morning, noon, and night.

In the case of pleurisy or pneumonia, or both combined, the sides of the thorax should be painted in a similar manner to that of the outside of the throat, and the chest then wrapped up with a binder, under which a layer of thermogene wool is placed.

Should there be repeated vomition, bismuth subnitrate, in 2½ to 5 grain doses, should be shaken on the tongue every three or four hours until twenty-four hours have elapsed since the last act of vomition took place.† Persistent and profuse diarrhœa must be moderated or suppressed by giving bismuth subnitrate, tannigen, or tannalbin, in 2½ to 5 grain doses, three times a day.‡ The mouth should be cleansed with peroxide of hydrogen solution several times a day, in order to prevent or combat ulcerative changes.

Light and easily digested food, such as peptonised milk, Brand's Essence of Beef or Chicken, Benger's Peptonised Food, etc., should be given in small and repeated quantities during the earlier or active stages of the disease. Later on, in the convalescent stage, scraped raw beef, boiled fish, boiled rabbit, rice-pudding, etc., may be offered, but *not forced down the animal's throat*, as is so commonly done.

Parrish's Chemical Food, cod-liver oil, cream, and other nauseous agents, as given by cat fanciers during the acute stage of distemper, when there is little desire for food, are harmful, destroy what appetite is left, and tend to induce vomiting.

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* The cat is very susceptible to chlorate of potassium, large doses causing hæmoglobinuria.

† In cases of persistent vomiting, we have found that chloretone, administered in a similar manner, has succeeded when all other agents have failed. It must, however, be given in small doses (1 to 2 grains), as we have observed that larger doses are liable to induce serious symptoms in the cat, such as convulsions, followed by a comatose condition.—Editor.

‡ Compounds of the tarry series, such as benzonaphthol, β-naphthol, salol, etc., must be given with extreme care to the cat, as this creature is very susceptible to such agents.

DISTEMPER IN OTHER ANIMALS.

THE FERRET.

In this creature there is a disease having all the clinical characters of distemper in the dog and in the cat. It is, however, usually manifested in the catarrhal form.

Eberth and Schimmelbusch in Germany (1887), and Nocard and Leclainche several times in France, encountered and studied an epizoötic occurring in ferrets and wild rabbits at the same time. The clinical characters and lesions resembled those of canine and feline distemper, and probably the disease was of a similar nature to those mentioned. A bacterium of the colon group was isolated. It was supposed that the affected wild rabbits, when in their burrows, contaminated the ferrets.

As to the etiology of the affection, gamekeepers, farmers, and sportsmen maintain it is contracted from distempered dogs, which are often brought into contact with ferrets. Sewell and the writer have observed transmission from the dog to the ferret. Mégnin mentions that his " la grippe " or malignant catarrhal fever of the dog is contracted by the ferret.

M'Gowan records that he obtained pure cultures of his organism of distemper from the trachea and nose of ferrets contracting distemper from dogs and other animals in his laboratory in Edinburgh.*

As regards treatment, the animals affected should have a liberal amount of warm bedding material in their hutches, so that they may coil themselves up in it and keep themselves warm, which they desire so much. They should be offered small and repeated quantities of warm milk and boiled rabbit, into which may be introduced small doses of quinine, aspirin, or calomel. The eyes and nostrils should be washed with chinosol lotion, and then smeared with vaseline or castor oil to prevent excoriation of the cuticle.

The disease should not be confounded with sarcoptic mange, termed by gamekeepers " canker."

THE RABBIT.

The rabbit is affected by a disease having all the clinical characters of canine and feline distemper, and probably it is due to an analogous virus, although several authorities have described diseases having similar characters, but alleged to be due to various micro-organisms. Youatt, as far back as 1832, described the distemper of the rabbit. M'Gowan* found his microbe of canine and feline distemper in rabbits dead of an epizoötic attacking the dog, cat, ferret, rabbit, guinea-pig, goat, monkey, and man.

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* *Journal of Pathology and Bacteriology,* 1911, vol. xv.

THE GUINEA-PIG.

A similar malady to the distempers of the dog, cat, ferret, and rabbit, occurs in the guinea-pig. A disease in this animal having identical clinical characters and morbid lesions has been studied and described by Phisalix as due to a pasteurella, and termed by him *septicémie du cobaye*, and by Nocard and Leclainche as *Pasteurellose de cobaye*. Weaver, in the United States of America, has also studied a spontaneous hæmorrhagic septicæmia in the guinea-pig caused by a bacillus.

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* *Journal of Pathology and Bacteriology*, 1911, vol. xv., and *Veterinary Journal*, January, 1912, p. 7.

CANINE TYPHUS, OR CONTAGIOUS GASTRO-ENTERITIS

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Stuttgart distemper, plague, or epizoötic; New dog disease; Contagious, epizoötic, or hæmorrhagic gastritis, with ulcerative stomatitis; Canine influenza or hæmorrhagic septicæmia; Abdominal typhus; Malignant gangrenous distemper; Mouth distemper.

French: Gastro-entérite hémorragique, ou dysentérique; Affection gastro-intestinale adynamique et athermique; Typhus du chien; Maladie des chiens de Stuttgart; Stomatite grave infecteuse; Pasteurellose canine.

German: Stuttgarter Hundeseuche or Hundekrankheit; Abdominal-Typhus; Gastro-Enteritis Hæmorrhagica et Stomatitis Ulcerosa Contagiosa Canum.

Italian: Moria dei cani.

Definition.—An epizoötic disease of the dog, usually characterised by an initial but very temporary elevation of the internal temperature, succeeded by symptoms of a more or less intense inflammation of the stomach, and eventually a gangrenous ulcerative inflammation of the mucous membrane of the cheeks and jaws, and in the majority of cases ending in death, which is generally preceded by abnormal lowering of the temperature and a comatose condition.

HISTORY.

Until the year 1898 little attention was devoted to this disease by the veterinary profession, although it was often encountered by those practitioners who concerned themselves principally with the diseases of the dog, and was occasionally alluded to in the writings of the older authorities.

Delabere P. Blaine* mentioned it in 1817 as a malignant or putrid type of distemper, "in which the gums bleed, the tongue is covered with a dark crust, and sometimes is ulcerated." Youatt* alluded to similar cases, in which "the mouth and lips swell and ulcers begin to appear in them, the gums ulcerate, and a sanious and highly offensive discharge proceeds from the mouth." Funke† stated in 1845 that in some animals distemper, especially in summer, appeared in a very malignant gangrenous form, which manifested itself by extreme weakness, great emaciation, total loss of appetite, bleeding gums, *an enlarged tongue with brownish or ulcerous crusts*, and often by a very bloody diarrhœa. Duttenhofer,‡ who wrote upon distemper in 1847, said that when of a gangrenous type it was accompanied by a hard tongue of a brownish-black colour, covered with ulcerations. Hofer of Munich§ encountered the disease in 1850, and gave a good clinical account of it, designating it as "Typhus der Hunde." In 1860 Reynal, under the heading of "Enteritis," gave a clinical description that appears to read like that of typhus. In 1888 Pierre Mégnin,|| a distinguished French veterinary scientist, described a "malignant infectious stomatitis," which he frequently observed, not only in that year, but also several years previously; from his description the disease appears to be none other than the canine typhus of the present day. A few years after this outbreaks of a disease showing gastric lesions were encountered in various parts of Great Britain, one occurring notably in Brighton after a dog-show; these were generally ascribed to malicious poisoning or to ptomaine-poisoning. The writer of this article witnessed several such enzoötics of a milder type, not only in the dog, but also in the cat. An anonymous writer, "Nemo," in the *Veterinary Journal*, mentions that they were frequent in Edinburgh during 1896 and 1897, and presented a virulent type. According to A. M. Macfarlane,¶ the disease largely prevailed in a malignant form in Edinburgh and surrounding districts from November, 1897, up to the summer of 1898. In the early part of 1898, and again during the following winter months, especially those of January and February, the writer observed it in London, and even in dogs arriving from Scotland and from Wales, and other parts of England. In Glasgow it was encountered from May to August, 1898, by J. R. McCall. According to Penberthy, who recorded his experience of it in the *Veterinary Record*, March 18,

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* Blaine, "The Diseases of Dogs," 1817.

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* Youatt, *Veterinarian*, 1830, and "The Dog," 1845.

† Funke, *Handbuch der Spec. Path. und Therap.*, Leipzig, 1845, Band i., p. 369.

‡ Duttenhofer, *Anleitung zur Kenntniss und Heilung der Hausthier-Krankheiten*, Stuttgart, 1847, p. 637.

§ Hofer, "Der Typhus der Hunde (Typhus, sive febris nervosa canum)," *Repertorium der Thierheilkunde*, 1852, Band xiii., p. 201.

|| Mégnin, *Le Chien*, 1893-94.

¶ *Stock-keeper*, April 21, 1899.

1899, simultaneously with that of Charles Taylor, who witnessed it in Nottinghamshire, and with that of the writer, various veterinary practitioners in Bristol, Clifton, Essex, Hastings, Hull, and other places, observed it several months before. Cade of Bristol kept a record of seventy-six cases that came under his notice in a few months. Sewell, writing to the now defunct *Stock-keeper*, March 22, 1899, asserted that, so far as he was aware, it was unknown in England six months before; he considered it first visited Portsmouth and Southsea, and in all probability it was brought to these shores from some Eastern country, perhaps China. The disease, however, did not attract widespread attention until Professor Klett, of the Veterinary College, Stuttgart, who came across his first case in that city on August 13, 1898, published his observations on one hundred cases, collected from August up to December, in the *Deutsche Tierärztliche Wochenschrift*, February, 1899, a translation of which appeared at the end of March in the *Journal of Comparative Pathology and Therapeutics*, 1899. Sir John McFadyean, commenting on Klett's communication, thought it was not improbable that some British dog may have carried the disease there, as the malady appeared immediately after a dog-show held in Stuttgart. By January, 1899, it seems to have disappeared from that city. It was observed in Munich by Albrecht in October, 1898, and by Feser during the course of the summer. At Frankfurt-on-the-Maine, Scheibel, veterinary inspector at the slaughter-house, found, from September 15 to November 15, 1898, that out of 292 dead dogs, more than 150 were affected by the disease. By the end of December it had vanished. At Dessau it appeared in December, and gradually increased from month to month, so that over 300 dogs were submitted to veterinary treatment. Richter, however, computes that 800 to 900 out of 1,300 were attacked in Dessau, where it seemed confined, as he only encountered two cases outside this town. At Mödling, in Austria, Franz Mattel saw, from June 8 to October 14, 1899, 126 dogs affected with more or less typical symptoms. In Vienna, Leopold Tremmel saw a great number of dogs seized with it in a mild form during the year 1899. About the same period Gundelach observed numerous cases of it in Magdeburg. Rabur has witnessed it in Wiesbaden. Zschokke estimated that in Zürich from January, 1899, to the end of that year more than 10 per cent. of the dogs were affected. The first case was seen in January, and it gradually reached its height by March and April, and disappeared by December. In Italy a great number of dogs were attacked, and it was particularly studied by Trevisan, Cherie, and Boldrin (1899) in Venice, and by Gambarotta (1901). In France, where it was very severe during 1899, it was observed in Paris by Huet, Brun, Frégis, Guillemard and Chigot, Ducourneau, and Ben-Danou, and in Toulouse by Bîmes and Sérès,* the two last having contributed very largely to the literature upon the subject.

Since 1899 it has frequently recurred after a more or less periodical interval of quiescence, and during the early months of 1911 it was particularly prevalent. As to it being a widespread malady there cannot be any question.

GENERAL REMARKS.

It would be rational to inquire if this disease is a really distinct etiological entity from distemper. At first sight one might be led to answer in the affirmative, but on further consideration of the antecedents of the affected animals and of the general clinical history of the malady, and a comparison of it with that of distemper, one is almost forced to conclude that if they are not identical, they are at any rate closely related in their causation.

Against the theory of the unity of these two maladies it is generally urged, (1) that in consequence of typhus usually attacking aged animals, many of which have had distemper ; (2) there being generally no catarrhal discharge from the eyes or nostrils ; (3) the mortality being great and the malady not being readily conveyed by direct contact, it differs essentially in those respects from distemper.

But we must take into account that distemper varies in its clinical manifestations, not only in particular outbreaks, but also in certain seasons, and after more or less varying periods. It is now a well-accepted fact that distemper may manifest itself without showing any of the usually acknowledged classical symptoms, such as catarrh, high temperature, exanthematous eruption, cough, etc. ; that it may attack certain dogs more than once ; that a very virulent form may overcome the immunity derived from a milder attack ; that the mortality varies from one season to another ; that even during the same season the mortality is greater in some districts or kennels than others ; and that the principal vehicle of the *contagium* is the nasal and ocular discharge, especially the earlier serous discharge. All this evidence only goes to show that clinicians cannot set up a rigid standard by which to diagnose one disease from the other, or indulge in dogmatic opinions. Hence the difference is only apparent and not by any means real.

No doubt if there were a nasal or ocular catarrhal discharge containing the active virus in typical cases of typhus, the disease would spread with greater rapidity than is the case. Many authorities conclude that because several animals in a certain place are attacked and show similar phenomena, the cause was communicated from one animal to another. In order to comprehend the problem of complications or secondary infections, to which, no doubt, the principal phenomena of typhus are due, it is necessary to understand the natural history of saprophytes and their relation to the animal body. That these vary in degrees of virulence is as undeniable as that regarding many of the food and other plants, which are harmless at times and in certain localities, but which become virulent at various epochs and on different soils.

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* E. Bîmes et E. Sérès, " Le Typhus du Chien," *Revue Vétérinaire*, September to December, 1901.

A profounder study of the clinical history of a great number of cases of typhus carried over a number of years indicates that the earlier symptoms of the disease are overlooked, or, if noticed, are not usually connected with the later and more pronounced manifestations. In the opinion of the writer the gastro-enteric lesions and the gangrenous ulceration of the oral mucous membrane have just as much right to be included as a secondary manifestation of distemper as the eruptive, catarrhal, pulmonary, nervous, or any other of the secondary phenomena. At any rate, gastro-enteritis and gangrenous ulcerative stomatitis are complications seen in many outbreaks of the usual forms of distemper; they are the epiphenomena, and not the phenomena.

To isolate typhus from distemper would, at the present state of our knowledge, be as illogical as to isolate the pulmonary or nervous manifestations of the latter disease as an etiological entity, or to differentiate catarrhal fever without the presence of an abscess from strangles, or lupus from tuberculosis.

But possibly in the future, bacteriological investigation may be able to prove what clinical evidence cannot—the difference or identity of distemper and typhus. Yet if both are due to an ultravisible virus, how are we to decide the point? Can it be accomplished by the clinician or by the pure scientist?

A point in favour of the unity of the two affections is the fact that canine typhus attacks chiefly aged animals, the probable explanation being that the younger ones have recently had distemper and are immune whereas the older animals have lost their immunity. The writer has seen several young dogs in the same house ill, some affected with the usual manifestations of distemper, and the others with those of typical typhus, the fatal cases of the latter having the characteristic coffee-coloured or prune-juice-like vomit.

Although the affection is usually observed in *aged dogs*, it may occur in young animals, and even in puppies. Its mortality is very high, especially during some seasons or in certain districts. Some have placed the death-rate as high as 80 or even 90 per cent. This would no doubt be correct if only those cases showing intense gastritis and accompanied by gangrenous ulcerative lesions of the mucous membrane of the cheeks, gums, or occasionally of the tongue, were included under this disease. But, on the contrary, there are numerous instances of mild or atypical cases, not generally recognised as belonging to this disease, which mostly recover, and should be taken into account when preparing statistics.

Relapses are very common, and recurrences are not unknown even in a dog that has lost a portion of his tongue at a previous attack.

ETIOLOGY.

According to many authorities, including Klett, the disease is transmitted from animal to animal by direct contact, or by the intermediation of excrement, vomit, urine, blood, and various diseased products. Penberthy failed to convey the disease by feeding a healthy dog on diseased products. Albrecht, with dysenteric material, blood and bile, injected under the skin of healthy dogs, and with the contents of the stomach from a dog dead of typhus, given to a healthy dog, failed to obtain positive or satisfactory results. Scheibel gave two dogs—one after neutralising the acidity of its stomach with sodium bicarbonate—small pieces of the stomach and intestines of a dog dead of the complaint. The one to which the alkali had been administered manifested at the end of two days all the symptoms of the disease, and on post-mortem examination showed lesions of hæmorrhagic gastro-enteritis. Subcutaneous injection of diseased products gave negative results. Richter and Pirl injected under the skin of a healthy dog 5 c.c. of bile derived from a dog dead of typhus, and all the symptoms and lesions of hæmorrhagic gastro-enteritis were produced. They injected into a Gordon setter, aged four years, 2 c.c. of bile from the dog killed by the experimental disease, and obtained similar but milder symptoms. Bîmes and Sérès saw the disease affect one dog, and three days after another in the same home; in a second case three dogs of the same litter succumbed within the space of twenty-five days. Sewell encountered an outbreak in a house where all the seven dogs therein were attacked, the first being a pug, the disease then spreading to the others, so that within ten days they were all affected.

On the other hand, the great majority of authorities, including Richter, Albrecht, Scheibel, Wagner, Gundelach, Zschokke, Mattel, and the writer, consider the direct mode of transmission from the affected animal to the healthy animal is exceptional, as they have seen healthy dogs kept in the same kennel or room as the seriously affected ones without observing the development of the disease.

Probably the virus is similar to, if not identical with, that of distemper —viz., an ultravisible one—and, if so, it would only be found active in the blood and other fluids in the earlier stage of the malady, and in consequence of there being no virulent external discharge, as in distemper, it would account for the disease not being readily conveyed to healthy animals.

Weather seems to influence it Rarely, if ever, seen during very hot, dry, or intensely cold, dry weather, it prevails at all times of the year when the weather is changeable or wet. When canine typhus is prevalent, distemper is comparatively rare, the former appearing to take the place of the latter.

BACTERIOLOGY.

Although numerous investigations from a bacteriological point of view have been undertaken with the view of determining the causal factor of typhus, no definite conclusion has been arrived at. Lignières experimented with virulent cultures of pasteurella derived from cases of canine distemper, and was able, by introducing them into healthy dogs, to produce symptoms and lesions similar to, if not identical with, those of typhus. Almy, Leclainche, Nocard and Vallée, isolated this micro-organism from affected dogs during various epizoötics. Scheibel found in the blood obtained aseptically from the heart large immobile rods, having the dimensions of those of anthrax, without spores, and, in addition, numerous small microbes resembling the *Bacillus coli communis*. Cultures made from the splenic juice gave a small ovoid bacillus. In the digestive organs, besides the usual bacterial inhabitants, he constantly found a micro-organism like that found in the spleen, resembling also that of the colon bacillus, as well as that of swine plague and of fowl cholera. Pirl isolated a small ovoid bacterium similar to that of the fowl cholera group or pasteurella. Zschokke found in the blood of the heart in fresh carcasses of dogs dead of typhus numerous small cocco-bacilli, having coloured poles similar to those of swine plague. In four cases these bacteria were in a pure state of culture in the scrapings of the kidney and in the microscopical preparations of the renal tissue. C. O. Jensen denies that the pasteurella of Lignières has anything to do with the causation of typhus or even with that of distemper.*

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* *Zeitschrift für Thiermedicin*, 1905, vol. ix., pp. 321-399 ; and *Journal of Comparative Pathology and Therapeutics*, 1905, vol. xxviii., p. 350.

Lucet* reported that in a dog dead of typhus he had discovered in the hæmorrhagic exudate of the intestinal lesions numerous spirochætes, some of which were thin, measuring 5 to 10 μ in length and 0·4 μ in thickness; others were shorter and thicker, being 4 to 7 μ in length and 0·8 μ in thickness. According to MM. Ball and Roquet, the above spirochæte discovered by Lucet was the *Spirochæta regaudi*, which is a normal parasite found in the gastric mucosa of the dog and cat, and therefore plays no part in the etiology of the disease (*vide* Spirochætosis).

PERIOD OF INCUBATION.

This has not been well determined. Some authorities compute it at from three to seven days, but on what evidence they base this assumption is not made clear. In some of Scheibel's experimental cases symptoms of typhus were produced at the end of two days, following inoculation with diseased products.

SYMPTOMS.

As these vary somewhat in intensity and in the rapidity with which they run their course in a particular case or outbreak, it is thought advisable to divide them into five forms—viz., the *peracute*, *acute*, *subacute*, *chronic*, and *mild*.

PERACUTE FORM.—This form is the most rapid in its course, and also the most fatal in its termination, the majority of cases running their course within twenty-four hours to three days, and quite 80 per cent. ending in death. The animal may be suddenly seized with an unaccountable vomiting, or perhaps has passed some almost pure blood *per anum*, although an hour or two before this, food was readily taken, or he was playing about or hunting as usual. Many cases have occurred where the animal appeared well over-night before going to bed, but during the night has disturbed his owner in consequence of his vomiting, or crying, or passing bloody stools; or, where he has not slept in his owner's room, he has been found in the morning in a drowsy, cold, and miserable state. There is in such cases usually great mental depression, extreme muscular weakness, and loss of contractile power of the cutaneous muscle when the skin has been gripped and the hold then loosened; the animal appears in a dazed state, does not care to move, but passively and persistently assumes, in an unconscious manner, the recumbent position. The ears, mouth, and feet are icy cold, and a dirty saliva issues from the mouth. Should he voluntarily get up for water, he seems to lose, for the time being, the dazed appearance; he laps up the fluid with avidity, but rejects it immediately, and then resumes the lethargic and immobile recumbent position.

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* Académie des Sciences, Séance du Juillet, 1910.

In other cases the symptoms suddenly appear, and, as mentioned above, a few hours before the animal seemed to be in a perfect state of health, jumping and frolicking about and eating his food as usual. He becomes dull, seems greatly depressed and very weak, not caring to move, but remaining in his kennel or basket. Violent and repeated vomiting appears early—in fact, it may be the first symptom to attract attention or to indicate that anything is amiss with the animal. Thirst is intense, but as soon as water is ingested it is almost immediately rejected. The vomit at first contains particles of food, or it may consist of the whole of the meal taken a short time before the illness; this is soon followed by the rejection of a glairy, albuminous-looking material containing air-bubbles and water; a short time afterwards the vomit becomes watery, of a greenish tint, due to the presence of bile; and, finally, it is followed by a fluid more or less thick in consistence, of a bloody, chocolate, prune-juice, or coffee colour appearance, and having a very nauseous odour. As can be imagined, the *appetite* is totally in abeyance, and any liquid nourishment forced upon the animal is, like the water swallowed voluntarily, immediately vomited. In a great number of cases a profuse diarrhœa, which may be bloody in appearance and very often of a putrid odour, and coincidental with the repeated vomiting, may be observed during the earliest stage.

The *respirations* are not much disturbed, except immediately before and after the act of vomition, when they generally become disordered before and rapid after the completion of this act. As the animal becomes drowsy they may become rapid and frequent, or, on the contrary, infrequent and somewhat deep.

The *cardiac impulse* is strong and sometimes violent, but usually infrequent and slow. The pulsations, taken at the femoral artery, may be of a similar character, but as time goes on they become weak, thready, and very frequent.

The *internal temperature* may be normal or even subnormal, but it is found to be elevated at least during the early stage should the practitioner see the case at the commencement

The *external* or *surface temperature* may be evenly distributed, but it is usually lower at the ears, mouth, and feet than at the rest of the body.

The abdomen is hard and drawn or tucked up, and on pressure over the region of the stomach pain is generally evinced by groaning or grunting and resistance on the part of the animal, who often vomits immediately after this manipulation. When the rectum is examined, or the thermometer inserted, or the point of the enema syringe introduced, pain is caused, which is manifested by crying, resistance, and straining.

When the animal is made to move, he as a rule does so with great reluctance, shows great muscular weakness, arches his back, has a tottering, feeble gait, and assumes the recumbent position without any hesitation at the first opportunity, and then remains in that position in a listless and hopeless manner. Sometimes, when he is touched or manipulated, he cries out as if suffering the agony of rheumatic pains. As time progresses, various parts, or even the whole, of the body are seized with tetanic or choreic contractions, especially if the animal be touched or an attempt is made to open his mouth. When these convulsions are localised, they are usually seen affecting the muscles of the jaws or the temporal or ocular muscles; they somewhat resemble the convulsions seen in strychnine-poisoning or in suckling bitches. The skin has often a purplish colour, is cold, and is insensible to pin-pricks. The alternate contractions and relaxations of the muscles may vary from 10 to 150 per minute, or they may only be occasional and intermittent; during their activity the internal temperature ascends, only to decline as soon as they cease, when the animal is left very prostrate, and dies in a comatose condition, even without exhibiting a struggle. In some cases death takes place during vomition or during the convulsive movements; the latter may continue for some time after death.

If the animal linger for two or three days, the mucous membrane on the inside of the cheeks and lips and around the teeth, and in a few cases even the free portion of the tongue, may become bloodless, blackish, and then greenish, indicating that the parts are gangrenous. Such necrotic lesions are insensible and cold to the touch, give off an abominable and characteristic odour, and when either removed artificially or spontaneously do not give rise to any hæmorrhage. When the mucous membrane of the jaws, especially in the vicinity of the alveoli, is implicated in this morbid process, the bone underlying the mucosa also becomes necrotic; but should the animal live for a time or survive, the necrosed bone becomes exposed some time after the gangrenous gum has sloughed, and, in the case of a recovery, the affected parts of the osseous jaw become in their turn exfoliated.

When the disease is fairly developed, the facial expression is anxious or haggard, the eyeballs are drawn back into their orbits, the pupils are dilated, the eyes are expressionless and no longer transmit impressions to the brain, the conjunctival membrane is reddish or purplish, but there is not as a rule much, if any, catarrhal discharge therefrom. The nose is cold and dry, and the nostrils are often plugged with blackish dry mucus, which adheres rather firmly to the mucous membrane. The animal rarely urinates, and this is explained by the repeated vomiting, as very little fluid gains an entrance to the system. The urine is of very high specific gravity, dark in colour, acid in reaction, and emits a strong odour.

ACUTE FORM.—This form was very prevalent during the years 1889-1890, especially in certain districts. It now occasionally rages in some localities where the disease has not been seen previously. The mortality is great, but the disease runs a much longer course than the peracute form, and a shorter course, sooner ending in recovery or death, than in the subacute or chronic form.

In a very large number of instances it apparently commences in a similarly sudden manner as the peracute form, but, as just stated, it usually runs a longer course than that form. In numerous cases, however, there are premonitory symptoms lasting two or three days before the characteristic phenomena, such as repeated vomiting and bloody diarrhœa, manifest themselves.

It is quite probable that every case shows some premonitory signs of the malady, but these not being very apparent to the owner, the veterinary surgeon is not usually consulted before the more pronounced symptoms, such as constant vomiting, insatiable thirst, absolute loss of appetite, and great prostration, alarm the owner to such a degree that he seeks professional advice.

However, it would be well here to point out that it is a mistake to conclude that vomiting and marked thirst are always present in this disease, the writer having seen many cases running their course, and mostly ending in death, without either of these characteristic symptoms being manifested during life.

Under ordinary that circumstances the disease is evidenced by loss of spirits, a very sad and depressed expression, refusal of food, great thirst, disinclination to move from the recumbent position, an unwilling and difficult progression, which shows a short, tottering, but rather stiff gait; an arched back, with semipendulous head and tail; the gradual but certain wasting away; and the characteristic vomiting and diarrhœa, which may not appear before the third day.

The emaciation and the mental and physical weakness progress from hour to hour, so that within twenty-four to thirty-six hours after the above series of symptoms have declared themselves the animal can no longer be made to rise; his head seems too heavy for his body, which, in its turn, appears too heavy for the limbs to bear; his mouth becomes cold, offensive, and often swollen; the eyes sink and become fixed, the pupils dilate, and the facial expression is pinched and anxious; and various muscles or groups of muscles, or even all the muscles of the body, twitch convulsively in a short, jerky or throbbing manner. The diaphragm in certain instances is also affected with these half-choreic, half-strychnine-like convulsions; the pulse becomes thready and almost imperceptible, the breathing rapid, and death ends the scene, often without a tremor or a movement, within forty-eight to sixty hours from the apparent commencement of the clinical manifestation of the malady.

Besides the data just mentioned, there are others that require notice. The *internal temperature* at the beginning of the complaint is often elevated one, two, three, four, or five degrees, but when the general intoxication of the system is established (and this is usually the period when the veterinary surgeon comes on the scene) it is generally normal or even subnormal. The *external or surface temperature* varies according to circumstances. It may be regularly distributed during the early stages, but when the disease has become pronounced, the ears, muzzle, and limbs may be colder than the rest of the body. The coat is dry, lustreless, and open; the cutaneous muscle is not very retractile; the skin itself, especially in those animals in which it is pink, has often a purplish tint. The conjunctivæ are dry, injected, of a purplish or dirty yellowish hue; sometimes they are palish, and do not, as a rule, show any or much catarrhal discharge. In other cases the conjunctival membrane is very vascular. The nostrils, like the muzzle, become dry, and their orifices are often plugged with a more or less dark brownish or blackish discharge, which becomes dry, and gives to the nasal breathing a snuffling character. In a few instances, however, the nasal and ocular discharges are distinctly mucous or muco-purulent in character, and not distinguishable from those witnessed in distemper. Rarely one observes hæmorrhage into the anterior chamber, and more rarely still keratitis is witnessed.

In a very large number of instances the mouth, about the third or fourth day, becomes very offensive, often swollen, and discharges a thick, ropy saliva, which is in some cases mixed with blood. This condition arises from the gangrenous ulceration of the buccal and gingival mucous membranes, and occasionally from the necrosis of the anterior portion of the tongue. This infective process often extends to the bone contiguous to or underlying the mucous membrane covering the jaws, especially in the region immediately below the necks of the teeth, also to the lateral surfaces of the tongue, to the fauces, soft palate, tonsils, and pharynx. In a few instances the writer has observed a similar condition affecting the whole thickness of the membranes or tissues at the commissures or angles of the mouth (noma) and those parts of the skin subjected to pressure, also the perianal membrane. In other instances, when the fauces, soft palate, and pharynx have a dark, turgescent appearance, due to the congestion or dilatation of minute bloodvessels, and are not rarely covered with a gangrenous or purulent diphtheroid material, spasm of the œsophagus is sometimes observed, especially if food be forced upon the animal or the pharyngeal region be pressed on.

Under these circumstances the lungs may become the seat of pneumonia or œdema, which is manifested by the usual signs. As in all those cases in which vomiting is pronounced, urination is not often performed, partly on account of no fluid being absorbed into the system from the stomach, and partly to the non-elimination by the kidneys. What little urine is present in the bladder is clear, thin, amber or brownish in colour, with a strong odour, and acid in reaction. It often contains albumin and bile-pigments, but rarely, if ever, sugar and blood. Occasionally renal epithelial cells and granular casts are found, when the kidneys are undergoing structural changes. It must not, however, be concluded that this condition of the urine is always, if ever, due to this infective process *per se*, as a large number of dogs, after they have reached middle or advanced life, are affected with kidney disease without suffering any appreciable disturbance of health. Hence it should be generally viewed as a coincidental affection.

Although nervous disturbance is not a pronounced characteristic of this disease, one often observes, besides the choreic or strychnine-like convulsions, convulsions resembling those of epilepsy, turning in a circle, delirious screaming, crying or whining, and sometimes pressure of the head against an obstacle; rarely is true paraplegia present, but in a great number of cases, especially fatal ones, coma is observed.

This type of the disease rarely lasts longer than a week or ten days, but the more prolonged its duration, the more chances there are of recovery, especially if the vomiting and the gangrenous state of the mouth appear early, not late, in the complaint. Recovery is assured if the necrotic patches slough off early, the vomiting ceases, and the nourishment given is retained. Convalescence may, however, be tardy, and apparently early recoveries are sometimes followed by relapses.

Subacute Form.—In this form, which has been more prevalent for the last few years than either the peracute or the acute form, the symptoms are similar in kind, but not so urgent as in the types mentioned.

This commences by stiffness and a disinclination to move; the animal seems chilly and languid, capricious in his appetite, but has often a great thirst, which is followed in a few days by repeated vomiting and diarrhœa, that continues for several days or more than a week, to be ultimately followed by gangrenous ulceration of the cheeks, of the gum below the necks of the teeth, and rarely by gangrene of the free extremity of the tongue. The temperature is at first elevated, but soon afterwards becomes normal. The pulse is weak and increased in frequency; the vomit is ropy, and contains air-bubbles; the abdomen is tucked up, hard, and painful; there is often constipation; the fæces are dark, very fœtid, and frequently blood-stained. Urination is infrequent and scanty. After a time muscular weakness becomes pronounced, the animal moves in a tottering manner, and has a difficulty in maintaining the standing posture. Muscular twitchings and rheumatic symptoms may appear. The conjunctival membrane is in many instances of a dark purplish colour; in others it has a dirty pale yellow tint or a bright red appearance, with the sclero-conjunctival bloodvessels standing out prominently.

This type runs a long course, and the majority of cases end in recovery. When a fatal termination occurs, it usually results from the septic process setting in late, and the severity of the symptoms increasing instead of decreasing after the first week, especially in very old dogs.

Chronic Form.—The writer has nowhere seen reference made to this type other than in his article on "Canine Typhus" in Hoare's "Veterinary Therapeutics and Pharmacology," published in 1906. This appears strange to the writer, as to his knowledge the chronic form is very commonly met with. Probably the earlier stages of it are not recognised as being connected with the later stages.

The writer has often encountered dogs that have manifested rheumatic symptoms; the latter, under appropriate treatment, have disappeared, to be soon followed by an increased desire for water, diminished appetite, and a gradual wasting away for some time. In spite of this, the animals apparently were not much distressed, nor did they present a sad facial expression; rather the opposite has been noticed, a brightness of countenance. As time has gone on they have shown signs of languor, a disinclination to move, a desire for warmth, and were observed to micturate frequently, as if suffering from diabetes. The animals took notice of everything going on around them, and appeared otherwise cheerful. This continued for two or three weeks, when vomiting set in, and the emaciation and weakness then progressed rapidly. Two or three days after the appearance of these latter symptoms the mouth almost suddenly gave off an offensive odour, resembling that of a cesspool or bad ensilage. The suddenness of this symptom was so well marked that on inspection of the mouth the day before the appearance of the gangrenous lesions, perhaps beyond a little dirty-greyish or brownish slime on the buccal mucous membrane and teeth, no lesions were noticed. On inspection of the mouth, the buccal and labial mucous membrane, especially those parts coming in contact with the carnassial, canine, and incisor teeth, upper and lower, and sometimes the anterior portion or tip of the tongue, were found blackish or greenish in appearance, cold and insensitive to the touch, and giving off the nauseating odour mentioned above. After a few hours this blackish or greenish appearance changed in colour to that of a greyish-green or straw colour, and a gangrenous slough appeared, and seemed to be separating itself from the healthy or living underlying membrane. Complete separation of the necrotic parts took place in a day or two, and if the animal survived, a reddish or greyish-red granular surface was left. Very often this necrotic process, when attacking the gums around the necks of the teeth, also extended to the underlying alveolar processes and a portion of bone surrounding them, which, with the necks of the teeth, were left exposed. Sometimes these latter lesions were present without any being observed on the buccal mucous membrane.

If the vomiting continued and the rejected material became of a coffee or prune-juice colour or brownish-red appearance, and gave off a nauseous or putrid odour, and if, also, there was a convulsive action of the temporal and masseter muscles (simulating that seen in strychnine-poisoning), sometimes causing a short, sharp, convulsive opening and shutting of the jaws, death invariably took place in a very short time.

The gastric secretion in this disease is always alkaline.

Mild Form.—This is frequently encountered, but often put down to a chill, simple gastritis, or gastro-enteritis. It runs a mild course, and mostly ends by recovery resulting within a few days to one or two weeks.

The animal is usually dull, has shivering fits, very little or no appetite, and perhaps an increased thirst. The temperature at the commencement is very high, but as soon as vomiting sets in it becomes normal. He vomits everything he takes, or whatever is forced upon him, such as food, water, or medicine. In a few days the majority of cases show a slight gangrenous ulceration of the buccal mucous membrane, especially of those parts which come in contact with the canine, carnassial, and perhaps the upper and lower incisor teeth. Frequently, however, these lesions are absent. In some instances the only symptoms present are an increased thirst and perhaps the frequent passage of urine. In most cases there is at the commencement a slight diarrhœa, which is easily checked. In other instances there is a slight vomiting which lasts two or three weeks. Under rational treatment recovery is the rule.

MORBID ANATOMY.

Peracute Form.—In a great number of instances where death takes place very early in the complaint, the lesions are indicative of an acute hæmorrhagic septicæmia, extravasations and congestions being well pronounced in one or more of the visceral organs, which are more or less purple in colour or intensely arborescent in appearance. This is especially noticeable on the external surface of the *stomach* and *intestine*, which often contain in their cavities a more or less thick, dark, red fluid, containing blood, mucus, and bile, and resembling in appearance tar or prune-juice. On washing or scraping these organs, the purple colour does not disappear. The rugæ or folds of the stomach are thick and swollen, often showing abrasions. In some instances the gastric mucosa is soft, black in colour, and appears as a vast sheet of clotted blood. The most intense lesions are often found in the great curvature of the right sac and pyloric region. The lesions in the intestine are mostly confined to the duodenum, the walls of which are of an intense purplish colour. At other times they have numerous ecchymotic spots scattered here and there over the mucous membrane. In a few instances the mucous membrane is raised by a submucous extravasation of blood, which gives the organ the appearance of a firm sheet of blood-clot. The jejunum and ileum are often affected by congestions and hæmorrhages. The posterior or so-called large intestine is frequently affected in a similar degree to that of the stomach.

The *omentum* is not rarely congested, having an arborescent and purple appearance.

The *liver* is swollen and friable, and on section is often marbled in appearance. The blood contained in the organ is not disposed to coagulate, but is tarry and stains the fingers. Jaundice is rarely present. The gall-bladder frequently contains a thick greenish bile.

The *spleen* may be enlarged and contain numerous hæmorrhagic foci, which vary in extent.

The *pancreas* is swollen and congested; sometimes it is the seat of small hæmorrhages.

The *kidneys* are swollen, enlarged, and contain much blood. On section the medullary substance is often congested and of a dark red colour; the cortical substance is, on the contrary, of a paler hue than normal.

The *bladder* contains a small quantity of golden-reddish urine, the appearance of which is due to the presence of bile-pigments; it also contains albumin.

The *lungs* are often œdematous and congested, and not infrequently show small hæmorrhagic spots, of a dark purplish colour, scattered here and there; broncho-pneumonic lesions are sometimes present.

The *pleural membrane* is rarely congested and arborescent.

The *heart* is paler than usual, friable, and presents a boiled appearance; the cavities contain a tarry-like incoagulable material; the endocardium and internal lining of the large vessels have a bruised aspect. Sometimes ecchymotic spots are present.

ACUTE FORM.—In this, as well as in the peracute form, the carcass is somewhat emaciated, varying according to the rapidity of the course of the disease and the condition of the animal previous to the manifestation of symptoms. The facial expression is pinched, and the eyeballs are sunk into their orbits. The skin around the mouth, front of the neck, chest, and fore-limbs, and sometimes that of the hind-quarters, is soiled with a thick, more or less desiccated reddish material. The visible mucous membranes are congested and often ecchymotic; the superficial vessels are very pronounced and distributed in an arborescent manner, and small hæmorrhagic points are scattered here and there on the mucous surface. The gingival mucous membranes around the necks of the teeth have a leaden appearance. If the animal has survived several days after the manifestation of the disease, we find on the inside of the lips and cheeks and on the gums ulcers with circumscribed borders, and covered with blackish, greyish, or greenish necrotic patches. The soft palate, tonsils, fauces, pharynx, and œsophagus, may be congested, showing vascular arborisations and occasionally a few hæmorrhagic points. With the exception of the œsophagus, these parts are occasionally the seat of gangrenous or necrotic lesions, similar in character to those found in the mouth. All these parts may be soiled with a bloody or brownish material.

The various glands of the body may be swollen, often succulent, and occasionally congested.

The *stomach* is small and contracted, generally empty of food, but often containing a small quantity of bloody mucus or tarry, semi-coagulated blood; its contents are alkaline in reaction; its walls, usually thickened, are of a brownish-red colour, and infiltrated with a bloody material; the mucous membrane is swollen, of a dark red or chocolate colour; its folds are prominent and extremely vascular, containing incoagulable blood.

The *small intestines* are œdematous, thick, and intensely inflamed; the mucous membrane of the duodenum is swollen, injected, and covered with numerous longitudinal reddish lines, and presents numerous hæmorrhagic foci of various dimensions ; the contents of the duodenum consist generally of a thick chocolate or brownish mucus, or of a bloody material. The other parts of the intestinal tube are frequently swollen, have red spots or streaks, and contain a reddish or brownish thick mucus. At times the whole length of the intestine is thickened and is the seat of an intense, hæmorrhagic inflammation. Occasionally the hæmorrhagic process is confined to the posterior or so-called large intestine.

The *mesenteric glands* are often congested, swollen, succulent, and hæmorrhagic.

The *liver* has a congested, swollen, and boiled appearance ; on section it is of a brownish-yellow colour, and only a small quantity of blood escapes from the cut surfaces.

The *pancreas* is swollen, œdematous, or congested.

The *spleen* is somewhat swollen, congested, and enlarged.

The *kidneys* are hyperæmic and softened ; on section a reddish material exudes.

The *bladder* offers a similar appearance to that observed in the peracute form.

The *tracheal* and *bronchial* mucous membranes are sometimes vascular and covered with a clear reddish mucus.

The *lungs* are œdematous, and contain some black tarry blood.

SUBACUTE FORM.—The emaciation is well marked. The gangrenous lesions on the inside of the mouth are well advanced ; the alveolar processes are here and there denuded of membrane, so that the underlying bone is exposed, and of a dirty brownish-yellow colour ; often the teeth are loose or absent. In some instances the free portion of the tongue is wanting or undergoing the gangrenous process.

The gastric mucous membrane is of a greyish or greenish appearance. The stomach contains a small quantity of a yellowish-brown or greenish, stringy material, which is alkaline in reaction. It may be distended with putrefactive gases.

The intestine offers similar characters to those found in the stomach.

In all forms the *dermis* and *subcutaneous tissue* are congested, infiltrated, purplish, and contain a black, tarry, non-coagulable blood, which flows from the vessels when the skin is cut. The muscles of the body are very dark in appearance.

The *brain and spinal cord* are congested, the degree varying according to the stage of the disease ; the vessels of the pia mater are dilated, and contain an abundance of black blood ; in the vicinity of the circle of Willis hæmorrhages may be found in those cases that manifested nervous symptoms before death.

The *joints*, especially the shoulder, hip, and stifle, are often congested, and contain a serous or sero-fibrinous fluid ; the synovial membrane may be rough, deprived of lustre, and even undergoing ulceration, the ulcers being covered with a greyish exudate. The extremities of the bones are red and hyperæmic ; the cartilage covering them is of a pinkish tint ; the medulla is very much increased in vascularity, and contains blood having a tarry appearance.

The lesions of the chronic and mild forms resemble those of the subacute form.

Very often, on making post-mortem examinations of dogs dead of typhus, one finds lesions that have no connection with this disease. These are principally cirrhosis of the liver and kidney, old thrombi in the spleen, subacute interstitial nephritis, catarrhal and cystoid nephritis, etc.

DIFFERENTIAL DIAGNOSIS.

In the majority of the peracute, the acute, and the subacute typical cases, there is generally no great difficulty in arriving at a correct diagnosis, especially if a little time is allowed to elapse from the commencement of the malady.

The prevalence and somewhat sudden appearance of the illness, the repeated and obstinate vomiting and diarrhœa, the normal temperature, the stiff gait and great mental depression and prostration, and the subsequent gangrenous ulcerative stomatitis, especially if accompanied by gangrene of the free portion of the tongue, allow one to differentiate it from any other malady except distemper, which is, at least in one of its clinical forms, quite indistinguishable from that of typical typhus (see p. 738).

In some of its milder or more chronic phases or atypical cases, a differential diagnosis is rather difficult, and may only be arrived at in a great number of instances by watching the course of the malady until some of the most characteristic phenomena appear ; its epizoötic nature is also of assistance in diagnosis. Also, it must be remembered that in some cases the mouth lesions may not appear until the disease is far advanced.

From *ptomaine* or *meat poisoning* it may be determined by the history of the case, the course it runs, and by the presence of gangrenous ulcerations.

From *phosphorus*, *antimony*, *podophyllin*, *mercurial*, and *strychnine poisoning*, it is differentiated by the longer course it runs, by the premonitory symptoms, and by the presence of gangrenous lesions in the mouth.

From the *presence of foreign bodies in the stomach and intestines, and from intussusceptions and intestinal obstructions*, etc., one can only arrive at a correct diagnosis by the epizoötic nature of the malady, by the general history of the case, the absence of foreign bodies or sausage-like swellings discovered by manipulation of the abdomen, and by the presence of gangrenous stomatitis. In occlusion of the intestine, partial or complete, the vomit is frequently stercoral. In many respects the acute and often gangrenous gastritis or enteritis due to foreign bodies or occlusions resemble, especially in their later stages, that of typhus in its later stage.

From *malignant jaundice* and *malarial jaundice* it may be generally distinguished by the absence of the yellow staining of all the tissues which precedes the gastro-enteric and pneumonic symptoms and lesions of these complaints. Again, malarial jaundice is confined to swampy, tick-bearing districts, especially in subtropical and tropical countries.

From *uræmic poisoning* due to retention of urine, and hæmorrhagic cystitis arising from occlusion of the urethra by calculi or sabulous material, it is differentiated by the absence of urethral obstruction, by the usually contracted bladder, which contains as a rule very little urine, and by the free flow of this fluid when an attempt is made to pass it. In *urinary obstruction* the animal is incapable of urinating, although he makes frequent ineffectual attempts to do so ; the bladder is distended and hard ; the catheter or sound cannot pass through the urethral canal ; and the urine is turbid, dirty, albuminous, alkaline, or ammoniacal, and generally hæmorrhagic in cases of twenty-four to thirty-six hours' standing.

From *non-puerperal endometritis* it is distinguished by the absence of distension of the uterine cornua with liquid catarrhal material, and in consequence distension of the abdomen, especially in its lateral borders, shown by palpation ; also the absence of any elevation of temperature. Intense *hæmorrhagic gastritis* is often an accompaniment of these cases of chronic endometritis. But the milder gastric and physical symptoms abate as soon as an outlet for the uterine collection is obtained.

From *simple catarrhal gastritis*, by the prevalence of the malady in a district, and by its obstinacy to respond to the treatment usually adopted for simple gastritis, the latter generally yielding to treatment within twenty-four to thirty-six hours.

From *non-infectious ulcerative stomatitis* it is distinguished by the presence of vomiting and diarrhœa and by the marked prostration, which are usually absent in the former affection; moreover, ordinary ulcerative stomatitis resolves as soon as the offending teeth are removed and animal food is given.

PROGNOSIS.

This is generally grave in the majority of instances, especially in aged dogs, which often unsuspectingly suffer from structural changes of the kidneys, liver, and heart. The mortality is, however, very high even in young, vigorous, and robust animals; but one may take it that the younger the animal the greater the chances of recovery become. The mortality varies according to the district, being higher in places where it has never prevailed before; it also varies according to the virulence of the outbreak. In mild visitations greater hope of recovery may be anticipated, especially in young or adult healthy and vigorous animals, provided they have been suitably fed, and not too much confined to the house, or in an unhygienic enclosed kennel. The mortality is very high in those dogs suffering from neglected and long-standing putrid periodontitis, with loosening of the teeth.

The prognosis should always be guarded, even in the apparently mild cases, or at least in their earlier stages, as a great number of such are very deceptive, and end up by succumbing. Again, relapses are to be feared, notwithstanding an apparent improvement, or perhaps the commencement of the convalescent stage. Breed plays no part in the mortality, beyond the conditions under which the animals are kept.

Prognosis is always unfavourable if the gangrenous lesions appear late in the course of the disease, if the vomiting continues, and the animal reaches the comatose stage. It is also unfavourable when the vomit has a putrid odour, and a coffee-like or prune-juice appearance. The half-tetanic, half-clonic convulsions are usually, but not always, a fatal sign, yet the sunken eyeballs, the widely dilated and immovable pupil, the small, thready, running-down or imperceptible pulse, and the subnormal temperature, may be regarded as evidences of a fatal termination. Pneumonic complications are unfavourable to a recovery.

The favourable signs are—the rapid spontaneous separation of the gangrenous sloughs, cessation of vomiting, the desire for food, the bowels acting normally, the animal being inclined to notice what is going on around him, and having a tendency to walk about as if asking for food, the temperature rising a degree or two, and the surface of the body feeling warm.

COURSE.

The course of the malady is usually a long one, but in the rapidly acute or acute form it is often short, because death either speedily sets in or recovery takes place, but the latter is the exception in peracute cases. As a general rule it terminates within one to six or even eight weeks; sometimes it may be prolonged to three months, and then end fatally.

TREATMENT.

The experience of every authority on this malady has shown that there is as yet no reliable method of treatment, and that the majority of cases, in spite of the various remedies used or the care bestowed, have succumbed. This result is, without a doubt, partly due to the fact that the greatest number of cases are not brought to the notice of the practitioner until they are far advanced, partly to the ignorance of its true nature, and partly to the view generally adopted of taking the lesions in the stomach and intestine as the primary situation or starting-point of the disease. Probably, when a full and serious investigation has been undertaken to arrive at its correct pathology, an exact and rational therapy may be revealed. Very often, however, practical therapy is in advance of etiology and pathology.

The writer, however, believes the primary seat of the infection is in the blood-stream, and that the gastric, enteric, and oral lesions are set up by a saprophytic or secondary infection acting on an already weakened defensive system. The lowering of vitality is brought about by the primary infection of the blood, induced by some living agent or figured element, probably an invisible one at present unknown. In fact, he regards the disease as a septicæmia of unknown origin. Furthermore, he views the symptoms, especially the nervous ones occurring during the later phases of the affection, as of alimentary origin, and due to the absorption of toxic principles generated in the disorganised tissues of the alimentary tract, and probably often assisted by the coincidental structural changes existing in the kidney, liver, and gastro-intestinal tube, which cannot cope with, modify, or destroy the poisons as soon as they are taken up by the circulation. He has frequently observed that dogs which are predisposed to recurrent attacks of gastro-intestinal or alveolar disturbance are more liable to be attacked by canine typhus.

As soon as a case is brought to the notice of the practitioner he should at once enjoin abstinence from exercise, exposure to the open air or draughts and dampness, and order the animal to be comfortably housed, and kept warm and quiet.

If he should be fortunate enough to be called in early in the complaint, before the gastric or enteric symptoms appear, he might adopt measures likely to lessen the effects of the virus circulating in the blood, and to prevent, perhaps, secondary infections arising in the digestive tube. For this purpose, and especially if there be an accompanying high temperature and possibly rheumatic symptoms, which are not unusual in the early stage, he may give quinine salicylate 1 grain, and aspirin 10 grains. The amount stated here is suitable for a dog weighing from 15 to 20 pounds,* and should be dropped as a powder into the buccal pouch, and repeated three or four times a day. As a rule, however, attention is not called to the affected animal until its owner has become alarmed at the obstinate vomiting, the insatiable thirst, and the absolute loss of appetite, when gastritis or enteritis; or both combined are in evidence.

The treatment generally recommended and adopted for simple gastritis or gastro-enteritis, such as the administration of various preparations of bismuth, dilute hydrocyanic acid, sodium bicarbonate and other gastric sedatives, aerated soda-water, and Brand's Essence of Beef in jelly form, is in this epizoötic form quite a failure. And from the fact that the above treatment does not check the vomiting nor bring about an improvement in the general condition of the animal within thirty-six to forty-eight hours after its adoption, one may safely conclude, in almost 99 per cent. of the cases, that it is a question of epizoötic gastro-enteritis, and not of the simple form one is dealing with.

As was pointed out in the symptomatology, the gastric secretion, as indicated by the vomit in this affection, is always alkaline, whereas in normal health it is strongly acid in reaction—a condition unfavourable to the majority of pathogenic and saprophytic microbes. And it is a well-ascertained fact, which was, it is believed, first pointed out by the immortal Koch, that many animals under natural conditions are refractory to certain infections by the alimentary canal; but when the normal acid state of their stomachs is overcome by neutralising it with alkalies, infection then takes place readily.

Therefore, in this disease, in which the normal acid gastric juice has become replaced by an alkaline secretion, it seems to the writer not to be an unreasonable supposition that the normal saprophytic microbes of the alimentary tract, having the opportunity, take it to invade the depth of the gastric and enteric mucous membrane and set up inflammatory and very often ultimately gangrenous changes, and allow absorption of toxic material from the digestive tube.

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* The doses mentioned in this article are suitable for animals of the weight approximately given here.

Consequently, with a view of acidifying the gastric secretion so as to destroy, or at least to modify, the injurious action of the micro-organisms contained within the alimentary tube, a 1 per cent. dilution of hydrochloric acid, given in dessertspoonful doses every three or four hours, should be administered. To aid in its potency, 1 grain of quinine sulphate should be added to each dose. This fluid also forms an excellent mouth-wash or gargle, which is so necessary in this complaint. The above measure is not of much use once structural changes have taken place.

Should the vomiting and diarrhœa become well established, other treatment should be instituted. The mouth, stomach, and intestine should be washed out a few times during the first twenty-four hours with a ½ to 1 per cent. solution of potassium permanganate; or a 2 per cent. dilution of hydrogen dioxide; or a 1 in 10,000 solution of biniodide of mercury and methylene blue, this latter in the same proportion as the mercury; or a solution of acetozone (5 grains to a pint of water).

It is generally futile to give medicine by the mouth when vomiting has set in, because it is almost certain to be rejected as soon as swallowed. Therefore it is advisable to depart from this traditional but nevertheless useless method, and administer medicaments as far as possible by the subcutaneous method; this is also the surest route to introduce fluid into the system—a most important element in the treatment of the disease. For this purpose nothing in the writer's experience surpasses the normal saline solution, composed of 49 grains of sodium chloride to 16 ounces* of boiled or distilled water. This solution is in the proportion of 7 to 1,000; it should in using be raised to the temperature of the normal body, or slightly higher. The easiest and best way to introduce it under the skin is by allowing it to gravitate from a graduated glass douche (a uterine douche answers every requirement) elevated several feet above the patient, and provided with long rubber tubing, to the free end of which is attached a hollow needle arrangement (slightly larger than the ordinary slot serum needle), to pass through the skin. From 2 to 10 ounces of this fluid should be allowed to infiltrate into the subcutaneous areolar tissue, when it produces a swelling varying in dimensions according to the amount of fluid allowed to gravitate. This swelling rapidly disappears by absorption of the fluid into the circulation, without leaving any ill effects upon the tissues. It should be repeated several times a day, but the needle should never be passed in the same spot—at least, until a few days have elapsed—else irritation and pain in the part are likely to be set up. It is surprising how the animal improves by this treatment, even when bordering on an apparently moribund state; and even if it does not actually bring about a recovery, it prolongs life for a time often amounting to several days. On this account the writer considers it is worthy of an extended trial. If it is deemed necessary, alcohol, acid hydrochlorate of quinine, strychnine hydrochlorate, digitalin, caffein, or ether, may be given along with the saline solution.

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* Sixteen ounces or one pound of water weighs exactly 7,000 grains.

As a nutrient, fresh serum obtained from healthy dogs, horses, or other animals, may be administered in a similar manner. In lieu of this agent, equal parts of well-strained sterilised milk and the saline solution may be tried.

But if any prejudice should be raised against this plan of therapy, the sensibility of the stomach should be blunted by the oral administration of *chloralose;* it not only soothes the gastric mucous membrane directly, and by its hypnotic action indirectly, but also acts as an antiseptic. *Chloretone* is very useful for similar purposes. Either of these should be given in the form of a powder dropped into the buccal pouch, or in a gelatine capsule, in the proportion of ¼ to ½ grain to the pound of the animal's body-weight, twice a day or as required. When the animal is well under the hypnotic influence of one of these drugs, *formidene* in the proportion of ½ to 1 grain per pound of the body-weight should be administered in a capsule. The alkaline secretion of the stomach, notable in this disease, splits up this agent into its component parts, and enables it to act as a powerful internal antiseptic. If it be deemed desirable for it to reach the intestine in its combined state, it should be given in a capsule composed of keratin, which is not dissolved by the gastric juice. In lieu of this, the following formula may be prescribed in capsule form:

℞.	β-naphthol	5 grains.
	Bismuth salicylate	5 ,,
	Powdered wood charcoal	2 ,,

This is sufficient for one dose, which should be repeated every three or four hours.

No nourishment should be given by the mouth, as it would generally be only rejected after increasing the irritation of the stomach. Equal parts of peptonised milk and the normal saline solution may be administered *per rectum* in small and repeated doses. The saline solution introduced under the skin not only washes the blood and acts as a diuretic and a cardiac stimulant, but it also does away with the necessity of giving the animals liquids to drink. As an alternative to the milk, one of the various beef extracts or meat suppositories may be introduced by the rectum. If the body should be cold, a hot-water bottle should be applied, and renewed as soon as it becomes cool.

After a few days of this treatment the sensibility of the stomach should be tested by small and repeated quantities of Brand's Essence of Beef or Chicken in jelly form; if it is retained for twenty-four hours without rejection by the mouth, barley-water or peptonised milk may be added to the dietary for three or four days longer, when, if there still be an absence of vomiting, *scraped* raw beef should be substituted, but only in very small and repeated quantities. The compound solution of bismuth containing strychnine* and pepsin is useful in this stage of the complaint. Sanatogen may be given a trial.

When there are any gangrenous ulcerations inside the cheeks, lips, or on the gum surrounding the teeth, or gangrene of the tip of the tongue, the sloughs should be encouraged by painting the surrounding living tissues with tincture of iodine, or applying nitrate of silver, sulphate of copper, chloride of zinc, or any other suitable topical stimulant; and as soon as the necrotic parts become detached they should be removed, and the remaining ulcerated granular surface dressed with hydrogen dioxide solution, tincture of iodine, or solution of permanganate of potassium.

Should there be any loosened teeth, they should be removed, and the surrounding exposed necrotic bone of the alveolar processes mopped with dilute hydrochloric acid two or three times a day, and allowed to exfoliate. It is not advisable to surgically remove the dead bone, as general infection, great suffering, and even death, are likely to ensue; if the animal survive, the bone will soon separate of itself.

Hygienic and dietetic precautions should be rigidly observed for some time after the patient has apparently recovered from typhus. Experience teaches one that a too speedy return of the animal to his normal diet, too much food given at one time, exposure to chill or fatiguing exercise, are far too common causes in bringing about fatal relapses from an apparent recovery of a mild attack of this malignant disease.

FELINE TYPHUS OR EPIZOÖTIC GASTRO-ENTERITIS OF THE CAT.

This disease is widely spread in the various varieties of the domestic cat, in which the mortality is even greater than in the dog. Its acute form sometimes kills off nearly all the inmates of a high-class cattery within twenty-four or thirty-six hours. It is manifested in all those forms to which the dog is subject.

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* Great care should be taken in prescribing this agent, as carnivora are very susceptible to its cumulative toxic effect, especially after repeated doses.

It is not known if this disease encountered from time to time by the writer is the same as that described by Boucek* in 1910, and which was confined to cats, and did not appear in other domesticated animals. It was characterized by lassitude, desire for air, vomiting and convulsions, death taking place in a few hours. On post-mortem examination small subserous hæmorrhagic spots were observed in the heart; the lungs were slightly congested, and the intestinal mucous membrane was œdematous and reddened. Smears from the cardiac blood, spleen, liver, and kidneys showed numerous bipolar ovoid bacteria in pure culture. The bacilli were ovoid, Gram-negative, very motile, contained neither spores nor flagella, and showed bipolar staining. They were 0·5 μ to 1 μ in length, and 0·3 μ in breadth. Two cultures did not liquefy gelatin; only showed a light yellow growth on potato. Rendered bouillon turbid. Did not form indol or change milk. The organism was very pathogenic for cats, rats, rabbits, mice, and doves. Boucek considered it belonged to the group of hæmorrhagic septicæmias.

Treatment.—Permanganate of potassium in solution, or hydrogen dioxide given by the mouth, has given some success. The cat is very susceptible to the toxic influence of β-naphthol, as well as to all of the coal-tar products, which should not be prescribed for this animal.

EPIZOÖTIC ENTERITIS IN THE CAT.

The writer has frequently observed a disease which he believes to be a specific morbid entity in the cat. It attacks chiefly kittens or young adults. The inflammatory lesions are confined to the jejunum and ileum, the mucous membrane of which often appears as if it had been scalded, and frequently becomes detached from the deeper, highly inflamed, submucous and muscular coats, which have a very reddened or congested aspect. In many instances the bowel has only a pinkish appearance. In the contents of the affected portion of bowel almost pure cultures of a large streptococcus have been observed. The *mortality* is as high as 90 per cent. Phisalix has found coli bacilli in cats dead of acute enteritis.

The principal **symptoms** are great nervous depression, repeated vomiting of a thin yellowish serous fluid, pain, evidenced by moaning or crying when the abdomen is manipulated, and the animal passes into a comatose state before death takes place.

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* Boucek, *Centralblatt für Bakteriologie und Parasitenkunde*, 1910, Band liii., s. 279.

Treatment.—A solution of permanganate of potassium or hydrogen dioxide may be tried, but, as already remarked, the disease is a very fatal one.

It is not known if this malady corresponds to the one described by Professor Zschokke under the name of "epizoötic croupous enteritis," but it is apparently related to the one studied and described by Skrzynski.

Z. Skrzynski,* working in Danysx's laboratory in the section of Agricultural Bacteriology at the Pasteur Institute in Paris in 1908, investigated an enzoötic which annually affected the cats during the winter months on an estate in Gastine (Eure). It killed off the cats, especially the young ones, in a few days. The proprietor thought the disease was due to the Danysx virus put down to destroy the rats. Very few symptoms were observed beyond evidences of the animals suffering pain. The post-mortem examination did not show any well-marked lesions beyond a slight intestinal congestion.

A cocco-bacillus belonging to the coli bacilli group was obtained from the blood, but was easily differentiated from the coli typhoid group by its immobility, its aspect of the cultures on agar, the fact that it did not ferment the saccharose in sucrosed agar, and especially its specific pathogenic action on cats.

The microbe appears in the form of rods with more or less rounded ends. It is not rare to find them in an ovoid or almost round form. They never form filaments. They are immobile, although animated with very active Brownian movements. They readily stain with the ordinary basic aniline dyes, but are Gram-negative. When the staining is somewhat pronounced they show a clear central space. They have no capsule, and do not form spores. They do not liquefy gelatine. But on potato they readily form an abundant thick yellowish layer, having a plicated and shining surface. This does not change colour. Milk is coagulated with a strong acid reaction. Indol is formed.

The cultures given in the food or injected under the skin of adult and young cats produced the disease, which ended in nearly every case in death. One kitten manifesting an abscess at the seat of inoculation rapidly recovered. The blood of every cat dead of the disease gave in abundance an identical microbe, which in cultures preserved its virulence for a lengthy period.

It was not pathogenic when the cultures were given in the food to mice, rats, guinea-pigs, and pigeons. It forms in a culture in bouillon of three weeks toxic products. A culture of twenty-four to forty-eight hours is absolutely deprived of soluble toxic properties.

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* Skrzynski, "Nouveau Microbe Pathogène pour les Chats," *Annales de l'Institut Pasteur*, tome xxii., Août, 1908.

When a culture is heated to 100° C. for ten minutes, a vaccine is obtained that, when injected into the peritoneum will give immunity to a virulent dose given by the mouth. Those animals that resist a subcutaneous or an intraperitoneal injection of a culture are firmly immunised. The cultures passed through a Chamberland bougie give products which also confer immunity.

Animals that are immunised show agglutinating powers. The coli bacillus and the *Bacillus typhosus* are not agglutinated by this serum or by normal serum. Reciprocally the antityphoid serum is not affected by the microbe in question.

Serotherapy.—The serum of a prepared animal possessed preventive and curative properties.

Cats can be immunised against this disease by the use of an attenuated virus, or treated by a curative serum when affected in the earlier stage. M. Skrzynski kindly informed the writer that both the serum and vaccine for this disease could be easily prepared.

FOOT AND MOUTH DISEASE

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Aphthous fever; Epizoötic aphtha; Eczema epizoötica; Eczema contagiosa.

French: Fièvre aphteuse.
German: Maul- und Klauenseuche.

Definition.—An acute infectious and inoculable disease, affecting chiefly cattle, sheep, and pigs; also goats, and occasionally horses, dogs, deer, and other animals. It is characterised by the presence of vesicles on the mucous membrane of the mouth and tongue. Similar lesions occur on the skin of the coronets and the interdigital spaces of the feet, more especially in sheep and pigs. In milch cows the udder and teats may be affected. It spreads with great rapidity, and in some outbreaks assumes a malignant type, associated with a high mortality and serious complications or sequelæ. The malady is also communicable to man.

HISTORY AND GEOGRAPHICAL DISTRIBUTION.

According to some writers, the disease was prevalent in Central Europe and in England in the middle of the eighteenth century; but, judging from the literature on the subject, one would be led to believe that it was not until the latter part of the year 1839, when it reached England, Scotland, and Ireland, that there was definite knowledge of its existence in the British Isles.

Simonds was said to be the first to recognise the disease in England. It occurred in a herd of cattle near Twickenham. Subsequent to this identification it was realised that cases of the disease had occurred previously in Smithfield Market, and its existence in Norwich Market was also recorded. The disease existed from 1839 to 1886, and this was its longest visitation. In 1852 it became very widespread in England, Scotland, and Ireland. In 1861 it declined to very narrow limits, but again increased from this date to 1865. The disease was scheduled in 1869, but it was not until 1878 that local authorities were compelled to put the regulations in force.

In 1870 the disease was reported in ninety-two counties ; 52,164 outbreaks were noted, and 691,560 animals were attacked. From 1877 to 1884 it was prevalent, some years showing a far greater number of outbreaks than others. Thus in 1883 there were 18,732 outbreaks, while in 1884 only 949 were recorded. In 1885 the number dropped to thirty, and in 1886 only one was reported, in which ten cattle were attacked. From 1886 to 1891 the disease was absent. In 1892 it reappeared, and ninety-five outbreaks were reported ; in 1893 there were two, and in 1894 three outbreaks. From 1894 to 1899 the disease was absent, and it reappeared in 1900, 1901, and 1902, in the last year only one outbreak being reported. From 1902 to 1907 it was absent. In 1908 there were three outbreaks ; it was absent in 1909 ; and in 1910 two outbreaks occurred in the West Riding of Yorkshire.

In 1911 there were several minor outbreaks in various parts of England, including Yorkshire, Middlesex, Derbyshire, and Somerset. Recently a second outbreak has occurred in Somerset. Owing to the stringent precautions adopted, these outbreaks have been quickly stamped out, and the spread of the disease prevented.

The disease is said to have reached America from England in 1870.

It has always been prevalent on the Continent, and in 1845-46 and 1855-1857 it extended all over Europe. A great epizoötic of the disease appeared in Germany in 1890-92. At the present time the disease shows no signs of decreasing on the Continent, as in August, 1911, 37,737 outbreaks were recorded in Germany ; and in July, 1911, there were 12,358 outbreaks in Holland, 4,097 in Belgium, and 16,027 in France. The disease is also very prevalent in India. According to Bang (1911), " During the past season a tremendous wave of the disease has passed over the whole of Central Europe, probably the greatest epidemic that has ever taken place."

ANIMALS SUSCEPTIBLE.

Cattle, sheep, and pigs, are the animals chiefly affected. Horses, goats, dogs, and cats, are occasionally attacked. Deer, reindeer, buffaloes, yaks, antelopes, aurochs, zebras, chamois, llamas, giraffes, elephants, and camels, are also said to be susceptible to the disease. Experimental evidence shows that of all animals cattle are the most readily infected by artificial means, pigs less readily, and it is sometimes difficult to infect sheep artificially. According to some authorities, the rabbit, guinea-pig, rat, mouse, fowl, pigeon, and duck, are refractory to artificial infection ; while the Berlin Commission (1898) found that in the small animals and in the birds mentioned, and also in the dog and cat, artificial infection by inoculating fresh lymph into the mucous membrane of the mouth, the peritoneal cavity, and the limbs, also by feeding experiments, did not prove successful.

Friedberger and Fröhner, however, state that birds may be attacked by the disease, while Law asserts that fowls are susceptible to inoculation, and also become affected if fed upon infected milk or other contaminated products.

ETIOLOGY.

The disease is conveyed from an affected animal to others, and the virus from animals of one species is capable of inducing the malady in animals of a different species. The virus exists especially in the lymph contained in the vesicles, and when the vesicles in the mouth rupture it is present in the saliva. It also occurs in the milk, in the blood up to the time of formation of vesicles, and, according to some authorities, it exists in all the excreta, in the exhalations from the body, in the expired air, in the tears, and in the nasal and vaginal mucus.

Nature of the Infecting Agent.—No specific micro-organism has been discovered for this disease. The consensus of opinion in the present day is that the disease depends on the presence of an ultravisible virus.* The results of experiments would indicate that only very minute quantities of lymph are usually required to induce the malady, according to Bang, the inoculation of $\frac{1}{5000}$ of a c.c. of the contents of a vesicle being sufficient. But variations occur in this respect. The vitality of the virus, according to laboratory experiments, is not great. Desiccation proves very destructive to it, as twenty-four hours drying at 87·8° F. rendered the lymph ineffective. Nevertheless it has also been found that it may remain active in an attenuated condition for three to four months in hermetically sealed glass tubes. It resists the action of cold to a marked extent, as was demonstrated by keeping the virus in an icehouse for nine or even sixteen weeks, and finding that it remained active. The virus is destroyed by boiling, and if the lymph be added to milk the infecting power is destroyed by heating to 185° F. Weak solutions of disinfectants render the virus inert. Thus, a solution of carbolic acid (1 per cent.) is said to destroy it after exposure for an hour.

As to the length of time that the virus can remain active outside the animal body under natural conditions there is little definitely known.

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* Siegel, at the recent Annual Congress of Prussian Veterinary Surgeons at Berlin, announced that he had discovered the causal organism of the disease in the blood and lesions of affected animals, and cultivated it in artificial media, and that in experimenting with it he had been able to satisfy all the postulates of Koch. Up to the time of writing further details are not available.

In the Annual Report of the Board of Agriculture (1899) it is stated that the expert advisers of the French Government were of opinion that in sheds in which affected animals had been housed, if not thoroughly cleansed and disinfected, the virus may retain its activity for several months. In the Edinburgh outbreak of 1908 the probable source of infection was traced to imported Dutch hay, and it was calculated that if such were the medium of transmission the virus must have remained active outside the animal body for at least three and a half months.

Penberthy proved by experiment that hay, straw, etc., on which the "slaver" from the mouths of affected animals had been deposited was capable of infecting animals four days afterwards.

The fæces of infected animals* caused an outbreak in a team of oxen that were employed in carrying the manure away from the farm and ploughing it into the ground, even in four weeks after the disappearance of the disease from the premises (Rosenkranz).

Zundel recorded instances in which stables occupied by affected animals, and left vacant for fifteen days, infected healthy animals when they were housed therein.

Fleming† drew attention to an instance in which twenty-four young cattle were purchased at a fair, and sent to a farm isolated from all cattle traffic, and there quarantined in a yard, so as to obviate all danger of introducing the disease by means of new cattle, as the owner had suffered considerable loss from the malady some months previously. The animals were quarantined for more than a month, and showed no symptoms of the disease. Then a number of feeding-troughs were introduced from a field a long way off. These had been employed to hold mashes and other soft food for affected cattle about four months previously, and had been allowed to remain in the open field for that period, exposed to the snow and rain of the winter. Within three days after the new stock had been feeding out of them the first case of the disease appeared, and in a short time all the animals were attacked. The probable source of the infection was traced by Mr. J. B. Martin, of Rochester.

Another outbreak of the disease was traced to ordinary field hay-racks out of which affected animals had been fed five months previously.

According to Bang,‡ who met with five recurrences of the disease on estates, at the end of six months to a year, when every precaution was taken to prevent the introduction of infection, and there was not a trace of the disease elsewhere in the country, "the virus may possess much greater tenacity than is usually supposed." He also observed in the neighbourhood of Aarhus, in Jutland, that the disease returned about three months after an outbreak, and a few days after carting out the manure left from the stock which was first attacked. He believes "*it is therefore advisable in practice to assume that the virus may persist for a very long time.*"

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* Fleming's "Veterinary Sanitary Science and Police." † *Ibid.*

‡ Paper read before a meeting at the Royal Veterinary and Agricultural College, Copenhagen, October 16, 1911. Translation in *Journal of Comparative Pathology and Therapeutics*, March, 1912.

MODES OF INFECTION.

Experimentally it has been demonstrated that the disease can be produced by rubbing the virus into the abraded buccal mucous membrane or into the skin at the coronets after scarification. Injection of the virus into the blood-stream will also produce the disease, and as the result of all the methods mentioned characteristic lesions appear simultaneously in the mouth and feet, and occasionally on the udder. There is no evidence to show that the virus can enter through the unbroken skin, but it has been demonstrated that subcutaneous injections of lymph in the case of all animals usually give negative results. In one outbreak in Germany, where an attempt was made to infect the animals not yet attacked, with a view to producing immunity, some saliva from the affected cases was placed between the lips of the healthy, and in the majority of instances the disease developed in three days afterwards. But in 55 out of a total of 130 the disease did not develop, and these had suffered from an attack the previous year.*

According to Friedberger and Fröhner, and other observers, the infection is not air-borne, and McFadyean asserts that "experience both at home and abroad has shown that when the agency of human beings and other living things as carriers of the contagion is excluded, the disease does not spread to other animals within even a short distance." †

Some authorities believe that the nasal mucosa, the bronchi, the conjunctivæ, the teats, and the anus, may be possible modes of entrance.

The virus, administered *per os* in gelatine capsules, has produced the disease, and the alimentary tract is believed by many to be the more common point of entrance. Slight abrasions of the skin at the coronet may also enable the virus to enter the system.

Direct infection from diseased to healthy animals may occur by licking, and in the case of sucking calves, from the udder and milk of cows suffering from the disease. New-born calves may show evidences of the affection, indicating that intra-uterine infection is possible.

Although the living affected animal is a most important factor in the introduction and transmission of foot and mouth disease, the infection may be carried by other media, such as contaminated fodder, litter, feeding utensils, water, etc. Pastures, the floors of buildings, railway-waggons, etc., may become the medium of infection when contaminated by the feet of animals suffering from the disease. In the case of pigs, the castrator may be the means of transmitting the disease from farm to farm. Flocks of sheep and droves of pigs may transmit the infection; also drovers, slaughter-men, attendants, dogs, cats, and fowls. Rats, mice, birds, and flies, are possible carriers. Fleming pointed out that animals which had been in contact with the diseased, even for a short period, were able to infect others, though they themselves remained healthy. Again, others apparently quite recovered were capable of transmitting the infection. Animals driven along roads which have lately been traversed by affected subjects are liable to become infected. Fairs and markets, watering-troughs, also buildings lately occupied by affected animals, are fertile sources of infection.

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* *Journal of Comparative Pathology and Therapeutics*, 1901.

† *Ibid.*, 1896.

According to Bang,* observations were once made in Sweden which seem to indicate that the infection can remain for a long time with an animal which has passed through the disease. He quotes an instance in which a Dutch bull. after undergoing the prescribed period of quarantine, was imported into a stock far north in Sweden, and several months later this stock was visited by the disease. It was then found that this bull had a deep slit at the back of the hoof, and that this slit (which probably resulted from a previous attack of foot and mouth disease), just at the time that the disease broke out in the stock, had grown so far down as to release, presumably, the virus hidden in it.

In milch cows, when the udders and teats are affected, the disease may be conveyed by the hands of the milkers to other animals; the milk also proves infective, especially to sucking calves.

The outbreaks which have occurred in various parts of England from time to time present some peculiarities which merit attention, prominent among these is the fact that since 1892 the source of the infection could not be traced in the great majority of instances.

Another interesting point is that after serious outbreaks have occurred the disease dies out for a variable number of years, and again reappears in a mysterious manner, and spreads to counties that are widely apart. A reference to the short history of the malady given (see p. 764) will show that from 1839 to 1885 the outbreaks were of a very formidable character, while in 1886 only one outbreak was reported. From 1887 to 1891 inclusive no outbreaks were reported, but in February, 1892, the disease was detected among foreign cattle at the Metropolitan Cattle Market, and in spite of all precautions it spread to many places widely apart, nearly 5,000 animals in ninety-five separate outbreaks having been attacked before it was eradicated in June of that year.

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* Paper read before a meeting at the Royal Veterinary and Agricultural College, Copenhagen, October 16, 1911. Translation in *Journal of Comparative Pathology and Therapeutics*, March, 1912.

As the importation of foreign animals except for slaughter at the port of landing was prohibited in 1892, the source of the outbreak of 1893, and of those which subsequently appeared, could not be attributed to imported animals.

In 1893 and 1894 only two and three minor outbreaks respectively were reported, and there were none for the following five years ; then the disease reappeared in 1900, with twenty-one outbreaks, and this visitation was characterised by the long intervals between the separate outbreaks, by the erratic manner in which it appeared in places far apart from each other, and by the fact that the first outbreak occurred in January, while at irregular intervals the disease cropped up in various counties, and was carried into the next year. In 1902 only one outbreak was reported, and for the following five years the disease was absent, but reappeared in 1908.

Various theories were put forward to account for the source of infection, but up to the present time a satisfactory explanation is not forthcoming. The fact that so many outbreaks have first occurred in the Eastern Counties, in most direct communication with the Continent, suggests that infection is probably introduced by some intermediary bearers from Continental countries, especially those nearest to our shores. The importation of hay and straw from countries where the disease is rife is a probable source of the introduction of infection, but this does not explain how outbreaks occur in counties widely apart. Thus in June and July, 1900, no fresh outbreaks were reported, but in August of that year cases occurred again in places as far apart as the East Riding of Yorkshire and Flintshire in North Wales. It seems hardly likely that separate sources of infection from the Continent were accountable for each of these outbreaks.

McFadyean* pointed out a striking analogy between the incidence of the outbreak in England and in those which occurred a few years previously to 1901 in Denmark. In spite of the insular position of the latter country, and of the fact that all foreign cattle were excluded, repeated separate outbreaks of foot and mouth disease occurred. The source of infection could not be traced, and almost the whole of the outbreaks occurred near the coast exactly opposite North Germany.

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* *Journal of Comparative Pathology and Therapeutics*, 1901.

McFadyean was of opinion that "of all the possible means by which the infection can be introduced from abroad at the present time, human agency appears to be the most probable." He pointed out that a person who had recently been near an affected animal or in contaminated premises is capable of spreading the infection, and "that the virus may by simple contact be transferred from such a person to another, who may thus become the innocent and unconscious agent in starting an outbreak." He also quoted examples of this indirect spread of the contagion, and drew attention to the restrictions that were imposed by some of the local authorities in Saxony, where children from infected farms were not allowed to attend school, and in some places the movements of all persons residing at places where outbreaks had occurred were restricted.

It was suggested that birds might be the carriers of contagion from the Continent to Great Britain, and McFadyean was of opinion that "the degree of its probability must depend mainly upon the frequency with which sea-fowls are in the habit of crossing from shore to shore in one single flight, for it is not at all likely that much danger would attach to a bird which, in crossing, had spent much time in the water."

German authorities believe that sparrows, starlings, etc., can convey the infection from one country to another.

Bang* is of opinion that the mysterious outbreaks of the disease which appeared in Denmark might be due to birds, such as gulls, crows, rooks, etc., which might conceivably fly across from Germany and carry infection in their feet, or possibly in their intestines after having collected it from infected manure. He does not think it impossible that the infection may have been carried by the wind. "It might be a question of particles floating in the air in a free condition, or attached to the legs of insects, or possibly spiders' webs, called in Danish the "flying summer," which just at this time of the year are seen flying about in profusion." Bang does not believe much in the carriage of infection through foreign fodder or packing, as the outbreaks in Denmark since 1892 were with very few exceptions restricted to estates in the southern part of the country, and an estate close to the coast had more frequent recurrences of the disease than any, while the fodder from foreign countries was distributed throughout Denmark. He thinks that this fact points distinctly to the infection being carried hither from Germany, where the disease has existed continuously, and that it "must be a natural mode of transport."

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* Paper read before a meeting at the Royal Veterinary and Agricultural College, Copenhagen. Translation in *Journal of Comparative Pathology and Therapeutics*. March, 1912.

It is interesting to note that in a recent outbreak of foot and mouth disease in the United States of America the source of infection was traced to young cattle which had previously been used in a certain laboratory for the manufacture of calf lymph. It appeared that the calf lymph employed to inoculate these cattle had been obtained from Japan, and was believed to contain the virus of foot and mouth disease. The symptoms of the latter disease were masked by those of vaccinia, yet these animals, when sold out of the laboratory, were evidently able to transmit foot and mouth disease to other cattle.

IMMUNITY.

One attack does not confer permanent immunity. The period during which animals are immune after an attack varies considerably. Some authorities regard the average period as probably six months. Bang* states: "I firmly believe that immunity is the rule. . . . In Germany the disease diminishes appreciably after very widespread outbreaks, and I can only conclude that this is largely due to the fact that the cattle have in many instances acquired immunity for a more or less protracted period." Instances are recorded where animals were affected on two occasions within a few months; but, according to Bang, these are rare exceptions.

It is possible for immunity to last for two years. This was demonstrated by the above authority in four large farms in Denmark, where three outbreaks occurred in consecutive years, and the survivors from the first outbreak were found to be immune after a period of two years.

Some observers state that second attacks are generally of a milder character.

MORBID ANATOMY.

The lesions in the benign form of the disease will be described with the symptoms (see p. 774).

In the malignant form the lesions are very variable. In some cases evidences of acute gastro-enteritis are observed, also intestinal hæmorrhage. Vesicles may be found on the pharyngeal, laryngeal, tracheal, and bronchial mucous membranes; also on the mucosa of the abomasum and duodenum. The gastric and intestinal lesions often consist of well-defined ulcers in the centre of discoloured patches; also congestion and ecchymoses may be observed on the intestinal mucosa, and ulcers on Peyer's patches and Brünner's glands (Fleming). In the abomasum acute inflammation associated with œdema of the mucosa, which may be denuded in patches, are amongst the lesions met with; and ulcers have been observed in the other divisions of the stomach, especially along the pillars of the rumen (Friedberger and Fröhner). Congestion and œdema of the lungs, fatty degeneration of the heart, and multiple embolic myocarditis, are occasional lesions.

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* Paper read before a meeting at the Royal Veterinary and Agricultural College, Copenhagen. Translation in *Journal of Comparative Pathology and Therapeutics*, March, 1912.

The lesions in the feet may be very extensive. The suppurative process may extend downwards under the wall, and the hoof may be shed. The articulations in the vicinity may show purulent arthritis, numerous small abscesses may be found on the limb, and evidences of general pyæmia may be present. Occasionally the skin around the base of the horns is the seat of lesions, and the horns become loosened and may be shed.

In very young animals of all species, especially in the sucklings of affected mothers, no lesions may be found beyond congestion of the vessels of the alimentary tract. In other instances the intestinal canal is found to be the seat of the eruption.

PERIOD OF INCUBATION.

This is usually brief, and varies from thirty-six hours to six days. Some authorities state that the first symptoms generally appear within a week after exposure to infection. Instances are recorded in which it has been as short as twenty-four hours and as long as ten and twelve days. The maximum, according to Friedberger and Fröhner, is from seven to fourteen days. Nocard and Leclainche, by administering the virus in capsules *per os*, induced the disease in two or three days. According to Fleming, by the use of the thermometer the commencement of the primary fever may be revealed ten to twelve hours before any other symptom is apparent.

SYMPTOMS.

Two forms of the disease are recognised—viz., (1) The *Simple or Benign Form*; (2) the *Malignant or Septicæmic Form*.

SYMPTOMS OF THE BENIGN FORM IN CATTLE.—The premonitory symptoms are a rise in temperature and a varying degree of constitutional disturbance. The temperature is generally raised to 103° or 104° F., but in some instances it may reach 105° F., or even higher. The constitutional symptoms observed are slight dulness and depression, shivering, "staring" coat, and abeyance of appetite. If the animals are at pasture they will usually be found isolated, disinclined to move, and showing stiffness in progression if forced to walk. In milch cows the supply of milk is decreased. When the internal temperature is high, constitutional symptoms are more marked, the appetite is lost, rumination is partially or entirely suspended, the abdomen has a "tucked up" appearance, grinding of the teeth may be observed, and constipation is present. Fleming states that the stage of premonitory symptoms usually lasts from twenty-four to forty-eight hours, according to the intensity of the fever.

The characteristic features of the disease next make their appearance, and with the advent of the eruption the temperature generally falls, and returns to the normal soon after the vesicles have ruptured. The lesions in the mouth usually precede by a short time those occurring in the feet, but exceptions to this are met with. Berry* drew attention to a very early symptom which may be detected in connection with the oral phenomena—viz., "a sucking of the lips and frequent deglutition of saliva."

During the next twenty-four hours a peculiar sound is heard, due to "smacking of the lips." This is a symptom of considerable clinical value, as it is usually present, and can be heard at some distance from the affected animal. The presence of vesicles in the mouth causes irritation of the mucous membrane. A flow of saliva is excited, and the animal is induced to smack its lips and slaver. Law attributes the peculiar sound to a "smacking of the tongue on the hard palate." In a short time after the vesicles have ruptured this symptom disappears, or may only be heard at irregular intervals.

Profuse salivation accompanies this "smacking sound." The saliva is more or less frothy in character, and at first tends to accumulate round the lips and to drop from the mouth in small masses; or, if the animal be at pasture, portions of saliva may be carried some distance by the wind. Later on, the saliva is of a ropy character, and often mixed with shreds of epithelium. It dribbles from the mouth or hangs in stringy masses from the oral commissures.

On palpation, the oral lesions in the early stages can be felt as minute hard bodies, resembling millet-seeds, the favourite seats of which are the inside of the lips and the point or sides of the tongue. A more or less diffuse redness of the buccal mucous membrane is also observed.

The lesions also appear on the incisor pad, or immediately behind it, and along the surface of the hard palate, but usually towards its posterior portion. The mucous membrane covering the inside of the cheeks is less frequently involved, and only occasionally are lesions found at the line of demarcation between the upper or lower lip with the skin proper. The small elevations mentioned above, quickly enlarge to form vesicles; these vary in size from that of a large pin's-head to a pea, or even to that of a walnut or larger. They are of a yellowish-white colour, and contain a light, straw-coloured, limpid fluid, which gradually becomes turbid. It is not uncommon for several small vesicles close together to coalesce and form one of large size.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xiv.

After two or three days the vesicles rupture, and shallow erosions result, having a bright red base and ragged edges composed of loose epithelium. Necrosis of the loose epithelium next occurs, and a slough is formed over the lesion. This is soon removed, and the base of the erosion becomes of a light yellowish-brown colour, due to a covering composed of a thin opaque material. Except in the case of extensive lesions, repair is generally prompt. The epithelium grows from the periphery to the centre of the affected part, and healing is usually complete in from ten to fourteen days. In some instances the process of repair may be delayed, and ulcers form which cicatrise slowly. But suppuration, as a rule, does not occur in connection with the mouth lesions, and healing by the formation of granulations is very seldom observed. When healing is complete, no evidences of the lesions remain on the buccal mucosa.

On the *tongue* the vesicles may be of small size, and contain but little exudate in many instances; but vesicles of large size, containing abundant exudate, are also met with, which rupture readily during mastication of the food or when the tongue is manipulated, and red erosions result. The commonest seats of the lesions on the tongue are the middle and lower or anterior portion. They often occur on the tip and sides of the organ, and may occasionally extend to its under surface. When the formation of vesicles is extensive, the mucous membrane may be absent from the lower fourth of the tongue, or it may hang out of the mouth, being attached to the sides and the tip of the organ, the denuded portion presenting a raw, painful surface.

Healing takes place in a variable period, according to the extent of the lesions. In severe cases it may require several weeks before complete healing results. In ordinary cases little or no traces of a cicatrix can be discovered.

On the incisor pad either a single or several vesicles may be found, or a number of these may coalesce. In some instances the entire surface of this structure is implicated, and the epithelium is cast off in the form of a large crust.

The *foot* lesions may be absent in some cases in cattle. According to Berry,* mouth lesions predominate in some outbreaks ; but when several cattle are affected, lesions in the mouth and feet will generally be present, and often individual animals are affected in both these regions, and in the case of milch-cows the udder is also involved.

The primary indications of the foot lesions are the presence of increased heat, tenderness on palpation, and slight swelling of the skin in the region of the coronets. The animal is disinclined for movement, and, if forced to walk, shows tenderness of the feet and lameness—symptoms which are well marked even prior to the appearance of the characteristic vesicles.

The vesicles are smaller than those seen in the mouth. They commence as small, hard elevations on the surface of the coronet close to the junction of the skin and horn, and usually towards the heel. They vary in size from that of a small pea and upwards, and soon form vesicles, usually of a greyish-white colour. They may remain discrete, or several of the smaller ones may coalesce so as to involve a large extent of the skin between the hair and the hoof. In a period varying from one to three days the vesicles rupture, and the result is that red bases are exposed. In the case of the smaller lesions the base may appear as a red spot.

There is no tendency to suppuration or ulceration unless complications arise, due to exposure to dirt, microbic infection, or injury, and in favourable cases healing takes place readily.

When the lesions are extensive, a separation of the horny and the sensitive foot may be observed. This, when present, always extends in a direction from above downwards.

Other locations in which vesicles occur in connection with the feet are the interdigital space close to the superior border of the wall, or several smaller vesicles may coalesce and form a large vesicle between the anterior and posterior commissures of the foot, or vesicles may be observed close to the anterior commissure or in the region of the coronary band, and occasionally in the vicinity of the ergot. The lesions described may appear in one, two, three, or all four feet.

The clinical picture when mouth and foot lesions are present in the same animal may be described as follows : In addition to the symptoms in connection with the mouth already mentioned, when the vesicles rupture the irritation is much increased, so that mastication becomes painful, and if the lesions be extensive there is inability to feed ; also, rumination is suspended. Pain and difficulty in deglutition are also observed. The saliva trickles from the mouth, and may be streaked with blood.

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* *Op. cit.*

The early symptoms of foot lesions are uneasiness and frequent shifting of the weight from one leg to the other. As the lesions progress, the animal picks up the fore-feet constantly, and frequently snatches up the hind-limbs, shakes them, and often kicks as though endeavouring to remove some adherent irritating foreign body.

Lameness is well marked. When both fore-feet are involved, the animal often draws the hind-feet under the body, so as to take the weight off the fore-feet; while in cases where all the feet are affected distress is well marked; the back is arched, and the animal endeavours to bring the feet under the body as much as possible. When at pasture, loss of condition is soon apparent, and emaciation frequently occurs in consequence of difficulty in prehension, painful mastication, and also because of the acute lameness, which prevents the animal from moving about and seeking for food. In severe and advanced cases the animal may constantly assume the recumbent position, and if forced to rise does so with difficulty.

Marked depression is present. The head is held down, the breath may have a fœtid odour, the ears droop, the eyes are dull, the eyelids may be half closed, and a catarrhal discharge may be observed from one or both nostrils or eyes. Evidences of pharyngitis, laryngitis, and bronchitis, may be present; also a hoarse cough and a fœtid diarrhœa may be observed in some cases towards the later stages.

Occasionally vesicles may appear on the anterior surface of the muzzle, and just within the interior of the nostrils. In the latter region they may be large in extent, and interfere more or less with respiration. The borders and outside of the lips may be the seat of vesicular formation in some instances, and as the result of the constant action of irritating catarrhal discharges, the muzzle may be denuded of its skin. Occasionally the muzzle and lips may be swollen.

In milch cows it is not uncommon to find vesicles on the udder. The lesions are found most frequently on the teats, but may also occur on the surface of the udder. The eruption is preceded by a red, tense condition of the organ, and the teats are swollen and painful. The vesicles may be small and isolated, or large and well defined, extending over a large surface of the teat. Occasionally they may be grouped in a circle around the orifice of the teat, and cause occlusion of the external orifice of the duct. The vesicles on the udder, when undisturbed, rupture at a later stage than those on the mouth and feet, and small red surfaces are left, which become covered by coagulated exudate and epithelial cells. In some instances the vesicles on the udder may not rupture, but their contents become absorbed, cicatrisation takes place, and the thin layer of epidermis is removed in the form of scales (Fleming).

The process of milking aggravates the condition of the teats, and healing is retarded; large ulcers may be formed, and abscesses may occur, due to the entrance of pyogenic organisms. The secretion of milk is greatly diminished, or may be almost arrested. The milk is of a yellowish-white colour, mucoid in consistency, and coagulates readily. According to Brown, it is of low specific gravity, and tends to decompose within twelve hours.

In young animals, especially sucking calves receiving the warm milk from cows affected in the udder, the disease is very fatal, and death may occur without the manifestation of any distinctive symptoms or local lesions. In some instances gastro-intestinal symptoms have been observed, and the mucous membrane of the alimentary canal was found to be the seat of the eruption.

SYMPTOMS OF THE MALIGNANT OR SEPTICÆMIC FORM.—This form is met with in some outbreaks of the disease, and may be accompanied by a high mortality. In some cases death occurs so suddenly, with symptoms resembling those of hæmorrhagic septicæmia or apoplexy, that an erroneous diagnosis of anthrax may be made. It is believed by Friedberger and Fröhner to depend on paralysis of the vital nerve centres induced by the toxins formed in the infective matter of the disease, and may occur during the height of the pyrexia or during convalescence.

Local lesions may also be met with. The udder may be acutely inflamed, from extension of the disease by way of the milk-ducts. This is accompanied by a sero-sanguineous discharge from the teats; abscesses may form, and induration or atrophy of the gland may result. Gangrenous mammitis may be observed in some cases.

The eruption may extend to the respiratory or to the intestinal mucous membrane. High fever, fœtid diarrhœa, and respiratory symptoms, are then manifested. Vesicles may appear on the vulva, the vaginal mucous membrane, on the skin of the abdomen and chest, and occasionally on the cornea. Abortion may occur in the case of pregnant animals, either during the attack, or, should the animal recover, during convalescence.

Brown observed cases of the malignant type in London in 1870, characterised by "effusions into the areolar tissue beneath the skin of the legs and some parts of the body, and when the enlarged parts were punctured the discharged fluids were always extremely fœtid. Great prostration accompanied this form of the malady, and death frequently resulted." *

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* Fleming's "Veterinary Sanitary Science and Police."

COURSE AND PROGNOSIS.

Benign uncomplicated cases, placed under favourable conditions, generally run their course in from eight or ten to fifteen days, and the prognosis is favourable. In about a week's time from the commencement of the malady the constitutional symptoms commence to subside; and although the mouth lesions have not yet healed, the animal looks for food and starts to eat, especially if grass or other soft diet be at hand. The lameness also gradually declines and the foot lesions heal, so that within the period mentioned above, all the visible symptoms of the disease have disappeared.

But when the affected animals are subjected to unfavourable conditions, such as filthy cowsheds, wet pastures, long journeys on hard, rugged, or muddy roads, voyages by sea, especially in rough weather, improper treatment, etc., the lesions become aggravated, and the serious foot complications already mentioned are induced.

The duration of an outbreak in a herd is generally from four to six weeks.

Mortality.—Under favourable circumstances the mortality is slight, but its rate varies considerably. Thus, for six weeks ending August 3, 1883, of 30,985 animals attacked, 82, or 2½ per 1,000, died. For five weeks ending September 29, 1883, of 129,036 animals attacked, 1,075, or 8½ per 1,000, died.

Of course, under the adverse conditions mentioned above, the percentage of deaths is likely to be increased.

In the malignant type of the disease the mortality is high, and varies from 5 to 50 per cent. In young animals, especially milk-fed calves, it may reach 50 to 80 per cent.

The losses attributable to the malady are very heavy. These include the rapid loss of condition, especially in fat stock, the interference with the supply of milk, the tendency to mammitis, the occurrence of abortion in pregnant animals, the deleterious effect of the milk from the affected animals on young stock, the tendency to complications in connection with lesions in the feet which occurs in some outbreaks, and, finally, the paralysis of the cattle trade which ensues when an outbreak occurs in a district or in a country. Bang estimates the economic loss from the disease in milch cows at an average of 34s. per cow. He states that it is not unusual for a cow when attacked by the disease whilst dry, to yield very little or no milk after calving, although the udder is to all appearance healthy. The rapidity with which the disease spreads, in many instances following the course of traffic, must also be taken into consideration when judging of the effects produced by an outbreak of this malady; also the obscure manner in which infection may be carried from one district or country to another.

I. FOOT AND MOUTH DISEASE IN SHEEP.

In sheep the lesions are often confined to the feet, and when the mouth is affected the vesicles are by no means so extensive and well marked as in cattle. The constitutional symptoms are severe, the pain and lameness arising from the feet are very acute, and the loss of condition is rapid. Usually all the feet are affected; the animals walk with difficulty, picking their steps, and they soon tend to assume the recumbent position. Marked distress is shown, and hurried respirations or "panting" are observed, especially during hot weather or if the sheep are forced to walk.

When at pasture, the affected animals generally lie persistently, remain isolated from each other in various parts of the field, and cannot be got to rise, in consequence of the painful condition of the feet. When feeding, they may rest upon the knees or on the sternum, and in searching for food may drag themselves along the ground. From the pain that is present and the inability to obtain sufficient food, they soon become emaciated and exhibit evidences of debility.

When standing, the animals are restless, the fore-feet are picked up alternately, and they kick with the hind-feet in a similar manner to affected cattle.

In consequence of the small size of the foot in the sheep, the major portion of this structure is involved, and a partial separation of the horn and skin is observed in most instances.

The commonest seat of the vesicles is the heel, but they may also occur at any point in the vicinity of the coronary band, at the anterior part of one or more digits, and in the interdigital space. The vesicles are small, being about the size of a large pin's-head, and a number of them may become confluent. After they have ruptured, lesions resembling small punctures in the skin result. Some writers state that the formation of vesicles in connection with the feet is not very common, but Berry points out that in cases showing lameness without vesicles being apparent, if the heels be taken between the finger and thumb and pressed, a few drops of fluid will often spurt out, and on relaxing the pressure the small ruptured lesions appear as well-defined punctures in the skin.*

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* *Op. cit.*

If the animals are subjected to unfavourable conditions, such as wet pastures or filthy surroundings, suppuration generally occurs in the lesions, the hoof is cast, and the sensitive laminæ are exposed, such cases proving very serious. When mouth lesions are present, they are best marked on the incisor pad; but the vesicles are relatively smaller than those occurring in cattle. The incisor pad is prominent, and the mucous membrane may be exfoliated in the form of a large crust. Healing is usually rapid. Vesicles may also appear on the mucous membrane of the lips, tongue, cheeks, and hard palate. After these rupture, small red spots are observed, which quickly heal. Some authorities have met with vesicles on the udder and teats, and occasionally on the inside of the thighs, the fore-arms, and on the vulva and the vaginal mucous membrane. In rare instances only, is the skin on the outside of the lips and face affected.

The symptoms in connection with the mouth lesions are not well marked. Fleming states that "the animal makes a peculiar smacking sound with its lips, which are kept moving as if it were sucking;" also that "the mouth is filled with a viscid saliva." Berry, however, points out that "the exudate is always small in amount; any appreciable salivation or 'smacking of the lips' is absent, but there is a frequent snapping motion of the lips and grinding of the teeth; a fœtid condition of the breath and diarrhœa are often present."

The symptoms and lesions of the disease in goats are similar to those observed in sheep.

II. FOOT AND MOUTH DISEASE IN THE PIG.

In this animal the lesions generally occur in the feet, but the vesicles may form on the snout and on the mucous membrane of the mouth in some instances. One source of infection is believed—in some cases, at least—to be the ingestion of milk from affected cows, but there is evidence to show that pigs may be the first animals attacked on a farm.

The symptoms observed, in addition to those of constitutional disturbance, fever, disinclination for movement, etc., are a tendency to assume the recumbent posture, acute lameness, the animal hides itself under the litter, and, if disturbed, utters sharp cries. If forced to move, the animal walks on its toes, there is marked pain and tenderness in the feet, and the limbs are swollen to above the fetlock-joints. The seats of the vesicles in the feet are around the coronary band and the interdigital space close to the pads of the feet. Either one foot or all the feet may be involved. If the animals be kept in dirty surroundings or driven long journeys on hard or muddy roads, complications are likely to occur, and the hoofs may be shed.

In some instances vesicles of large size have been found on the tongue, also in the pharynx, and in the latter situation they have been known to cause death from asphyxia.

Occasionally the udder is involved, the entire surface of the organ being covered by numerous small vesicles.

According to Fleming, there is sometimes observed in this disease in the pig "a vesicular or papular eruption on different parts of the skin, as well as tumours which become abscesses, or œdema of an erysipelatous character affecting the limbs."

Sucking pigs may become infected from ingesting the milk of sows suffering from the malady, and may succumb without manifesting any symptoms.

III. **FOOT AND MOUTH DISEASE IN THE HORSE.**

Some British authorities doubt or even deny that this disease can be transmitted to the horse. Although instances are rare, still, cases have been recorded in England, while on the Continent it has been observed by a number of writers.

Armitage* described a case that occurred in 1869 in a mare that occupied the same paddock as several cattle affected with the disease. The symptoms observed were loss of appetite, salivation, and the presence of typical lesions of the malady on the inside of the lips and on the tongue. An anonymous writer in the *Veterinary Review*, vol. iii., recorded several cases in the horse.

According to Fleming, cases were observed at the Turin Veterinary School, and others were described by Hurtrel d'Arboval, Levrat, Levigney, Villeroy, Dubos, Lafosse, Lemaire, Röll, Serres, Stockfleth, etc. All were due to contact with affected cattle. In 1873 Janné reported three horses affected with the malady from eating forage contaminated with saliva from diseased cattle, and the veterinary surgeon attending these horses became inoculated through handling them.

The disease may occur in a sporadic form, but, according to some observers, it may be epizoötic in character. The symptoms resemble those met with in cattle, but the eruption is usually confined to the mouth. The eruption of vesicles may recur, in which instance the course of the disease may be prolonged to two or three weeks, and emaciation becomes well marked in consequence of the inability to feed by reason of the mouth lesions.

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* Clater's "Cattle Doctor," revised by Armitage,

De Jong* recorded three cases of foot and mouth disease in foals. The animals were just weaned, and were placed in a field occupied by cattle suffering from the disease. In five days the following symptoms were observed—difficulty in prehension of food, salivation, and the presence of aphthæ on the buccal mucous membrane. The aphthæ occurred at different points on the buccal mucosa ; some were intact, and others were ruptured. In ten days after the first appearance of the symptoms one of the foals still showed salivation, and traces of vesicles on the tongue and other portions of the buccal mucous membrane. No lesions were found in other parts of the body.

According to Röll,† severe cases may occur. The mucous membrane of the inner surface of the lips and cheeks, of the gums, also of the upper surface and sides of the tongue and adjacent parts, is intensely inflamed and covered with patches of somewhat thick grey-coloured exudation of variable size, which often coalesce. These are adherent to the subjacent membrane, which is eroded and bleeds easily.

The tongue may be involved, and often the nasal mucous membrane shows a croupous inflammation. Ulcers may appear in the vicinity of the nostrils and mouth, the lymphatics of the cheeks may be inflamed, and swelling of the glands may be present ; also pharyngitis, bronchitis, and acute catarrhal inflammation of the gastro-intestinal mucous membrane. In rare cases a vesicular eruption has been observed on the posterior part of the hind pasterns when the skin was white.

IV. FOOT AND MOUTH DISEASE IN THE DOG, CAT, AND BIRDS.

Hering and Adam have observed cases of the disease in the dog, the source of infection being the ingestion of milk from affected cows. In the cases recorded the lesions occurred in the mouth, and on the skin between the toes and between the pads of the feet.

In the *Cat* the lesions found in the cases reported were in the mouth, tongue, and upper lip, and constitutional disturbance, vomiting, and lameness, were present.

In *Birds* the eruption was observed in various regions. In the fowl the vesicles generally appeared around the nasal openings and on the crest, but also occurred in the mouth and nostrils. In the goose the feet and interdigital membrane were the seats of predilection (Fleming).

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* *Tydschr. v., Veeartsenijkunde,* translation from *Annales de Méd. Vét.* in *Veterinary Record,* February 17, 1912.

† Fleming's "Veterinary Sanitary Science and Police."

DIFFERENTIAL DIAGNOSIS.

In CATTLE it is generally admitted that the diagnosis of foot and mouth disease seldom presents any difficulty. In the case of a single animal in which typical symptoms are not developed a doubt may arise, and it may be considered prudent to defer giving a definite opinion, and to isolate the case until its real nature can be determined.

When, however, a considerable number of cattle are affected, and present the symptoms we have described, the diagnosis is generally clear.

McFadyean* points out that the marked contagious character of the disease, and the fact that it affects simultaneously both cattle and sheep, are important factors in connection with diagnosis. He also states that, although the symptoms presented by a single animal may resemble those of the disease in question, the case "may be set down as not foot and mouth disease when, in spite of abundant opportunities for infection, it does not spread after a few days to other cattle or to sheep."

Penberthy† states that "in the case of cattle there is no recognised contagious malady in which fever is followed by vesicles in the mouth and feet."

Sporadic Aphtha, also known as Pseudo-Aphtha or Vesicular Aphtha.—In this affection fever is absent, the vesicles are smaller and more numerous than those of foot and mouth disease, healing is more rapid, foot lesions are absent, and the disease cannot be transmitted to healthy animals.

Bacillary Necrosis in Calves.—Although lesions may occur in the feet as well as in the mouth in this affection, their character is altogether different from the eruption of foot and mouth disease (see Bacillary Necrosis, p. 284). Moreover, the former disease does not usually attack adult animals.

Foul in the Foot may affect a number of cattle in the same herd, but mouth lesions and vesicles are absent, and the lesions in the feet bear no resemblance to those occurring in foot and mouth disease.

Vaccinia.—The eruption in cow-pox is characteristic, and is not likely to be confounded with the lesions on the udder in cases of foot and mouth disease.

The *malignant form* of foot and mouth disease, with obscure symptoms and sudden deaths, can, of course, be distinguished from anthrax or hæmorrhagic septicæmia by a bacteriological examination of the blood.

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* *Journal of Comparative Pathology and Therapeutics*, vol. xiv., p. 95.

† *Ibid.*, vol. xiv., p. 25.

Pseudo Foot and Mouth Disease.—On the Continent, cases of stomatitis in cattle occasionally occur which simulate foot and mouth disease in certain respects. Müller * recorded three cases, the first in a calf aged fourteen months, which showed several erosions on the gums of the lower jaw and on the incisor pad. These erosions presented the appearance of flat superficial ulcers, varying in size between a grain of linseed and a horse-bean, and the base of each showed reddish granulations. Salivation and other mouth symptoms were absent, and the feet were healthy; but the appetite was interfered with, and the animal lost condition. Four other animals in the same shed showed no symptoms. Two similar cases were observed in other districts. The cause was ascribed to the presence of a thorny plant in the fodder (*Ononis spinosa*), the spicules of which punctured the mucous membrane of the mouth and permitted the entrance of micro-organisms.

Ostertag and Bugge † described a specific papular stomatitis in Bavarian oxen which occurred as an epizoötic and simulated foot and mouth disease. Clinically it started as a circumscribed stomatitis, leading to the formation of small hard swellings surrounded by a red area. After the altered epithelium was shed, sharply-defined depressions remained. Fever was absent, and the course of the disease sometimes lasted for months. The affection was capable of spontaneous transmission from animal to animal, and was also conveyed by inoculating calves under the tongue with small fragments of diseased mucous membrane from the mouths of affected animals. The blood and filtered blood-serum also proved infective. It was distinguished from foot and mouth disease by the absence of vesicles and of foot lesions.

In SHEEP the following diseases simulate foot and mouth disease, and special care is necessary in the differential diagnosis.

Foot Rot is differentiated as follows:

1. Although contagious, it does not spread with anything like the same rapidity as foot and mouth disease, and it is never communicated to cattle that are pastured with the affected sheep.

2. By the character of the lesions. In foot rot the lesions commence either on the solar aspect of the foot or in the interdigital space, and there is a marked tendency to the formation of growths of a fungoid nature in all parts of the foot. In foot rot, when horn is detached from the sensitive foot, portions still remain attached at the upper

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* Müller, *Deutsch. Tierärztl. Wochens.*, July, 1906.

† *Zeitschr. f. Infektionskr. par. Krankh. u. Hyg. d. Haust.*, vol. i., 1905.

border. Again, in this disease there is an absence of the characteristic vesicles.

In foot and mouth disease, on the other hand, the lesions commence in the coronary region, and in a typical case there is no disease of the horn tissue. When the claws separate and are shed, the separation commences at the coronary junction, generally towards the heel, and the shed claw is not diseased *per se*.

Occasionally foot rot may be associated with foot and mouth disease, in which case the horn will be found diseased.

3. In foot and mouth disease all the feet are usually affected, and there is general stiffness and lameness. In foot rot the lameness usually commences in one or two feet, or the disease may for a long period be confined to one foot.

Contagious Pustular Dermatitis, also known as "Orf."—This disease was mistaken for foot and mouth disease in Scotland in 1901. It will be found fully described on p. 300.

Berry,* who has studied the disease and written an excellent article on the subject, differentiates it from foot and mouth disease by the following features :

1. Contagious pustular dermatitis "is a pustular disease, and not vesicular. It affects the skin proper, and not the mucous membrane of the mouth, unless from unusual complications, and then the lesion is ulcerative, never vesicular."

2. "The soft parts of the foot—*i.e.*, the heels and interdigital space—are but rarely affected, except from extension of disease, and the lesions of the foot in their early stage are always associated with pus, which is not the case in foot and mouth disease. Separation of the horn is not frequent. The gait and appearance of the animal during locomotion is not as if all the feet were affected, for the acute lameness generally arises from lesions affecting one foot only."

3. "The spread of the disease is not so rapid, and cattle, although in direct contact, remain unaffected."

4. "Emaciation is not well marked, the sheep on the whole feed well, and the general appearance of dejection and ill-health is absent, except, perhaps, in a very few instances."

5. "There is no constant snatching and picking up of the fore-limbs or kicking out of the hind-limbs. The sheep are not found lying scattered about the pastures, as in foot and mouth disease, or at most only a few individuals in a flock are thus separated from the others."

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* *Journal of Comparative Pathology and Therapeutics*, December, 1901.

6. "Lameness is not well marked, but naturally it is increased with the severity and aggravation of certain cases; it is entirely absent in others. There is more evidence of pain from pressure than pain from irritation and soreness."

Epizoötic Fibrinous Aphtha.—In Roumania an affection termed "epizoötic fibrinous aphtha in sheep" was described by Vigadi.* The outbreak occurred in a flock numbering 760. It spread rapidly, and in three or four weeks about three-fourths of the total number of animals were attacked, and subsequently only fifteen or twenty of the entire flock escaped. The lesions consisted of round, firmly attached, greyish-white patches, surrounded by bright red areolæ, on the inner surface of the lips, the incisor pad, the gums, the fauces; the back of the tongue, and the hard palate. The margins of the lips were covered with small brownish crusts. The deposits gradually became thicker, assuming at first sight the appearance of vesicles. In three or four days the centres showed signs of softening and breaking down, resulting in the formation of depressions and converting the bladder-like lesions into rings. Later on the deposit disappeared, leaving a reddish superficial loss of substance which soon healed. The duration of the disease was eight or ten days, and no deaths occurred. Five healthy sheep introduced from a distant pasture contracted the affection in from three to six days. It could not be conveyed to oxen or horses. No mention was made of the etiology of the disease.

This affection, although simulating in some respects the mouth lesions in foot and mouth disease, lacked many of the characteristic features of the latter. Moreover, foot lesions were absent, and the affection could not be transmitted to cattle.

PROPHYLAXIS.

This consists in isolation of all affected animals, and prohibition of the movement of cattle, sheep, pigs, and goats, from all infected areas. Thorough disinfection of boats, cattle-trucks, cattle-pens, hides and skins from infected countries, etc., should be carried out, and all fodder, litter, etc., from infected districts should be excluded from non-infected districts.

The hands, clothes, and boots of attendants or those engaged in the slaughtering of affected animals should also be thoroughly disinfected.

Foot and mouth disease is scheduled under the Diseases of Animals Act, so that all outbreaks are compulsorily reported to local Authorities, and dealt with by the Board of Agriculture.

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* "Allatorvosi Lapok, 1905," *ex Deutsch. Tierärztl. Wochens.*, 1906; translation in *Journal of Comparative Pathology and Therapeutics*, December, 1906.

Owing to the many obscure ways in which the agents of infection may be carried, the most efficient and economical method in dealing with recent outbreaks in Great Britain has proved to be the destruction of all affected animals and those in contact therewith, and the quarantining of the district or county in which the outbreak has occurred. The various rules in connection with this subject will be found on referring to the Diseases of Animals Act. Bang adopts the radical measure of burying the slaughtered animals in a large pit, to which they were led and where they were shot. By this means the services of slaughter-men are dispensed with, and he regards these as very dangerous carriers of infection.

Protective Inoculation.—Various serums have been tried by Continental authorities, but it cannot be said that such have proved of practical utility. According to Friedberger and Fröhner, "the problem of a reliable general protective serum inoculation against foot and mouth disease remains unsolved at the present day."

TREATMENT.

Attention, when treatment is permissible, should be directed to the floors of buildings, in order to prevent the occurrence of serious lesions in the feet of affected animals. Perfect cleanliness is essential, and the floor should be kept dry by covering it with sawdust or tan, and sprinkling these with a disinfectant powder. The food should be soft, such as boiled or pulped roots, gruel, mashes, etc.

In simple cases little medicinal treatment is required. Chlorate of potash or nitrate of potash may be given in the drinking-water. If the mouth symptoms are severe, applications of borax, chlorate of potash, and glycerine, will be advisable. Boric ointment is a useful application for the udder and teats.

The feet will require attention, and may be dressed with mild astringent applications. If suppuration occurs, the detached horn should be removed, and antiseptic agents applied. The late Professor Williams advised a lotion composed of 1 part of pure carbolic acid to 12 parts of white lotion. Pledgets of tow soaked in this lotion are to be applied to the affected parts and secured with a bandage. When extensive sloughing occurs, surgical measures will be necessary, and the employment of agents such as solutions of sulphate of copper or chloride of zinc.

In the malignant form of the disease internal antiseptics, such as salicylic acid or carbolic acid, are indicated, and stimulants may be necessary. It is very doubtful, however, if such cases repay the cost of treatment.

PUBLIC HEALTH.

The disease can be conveyed to man by drinking the milk of affected animals, by butter made from infected milk, and by the milking of cows whose udders are affected. It can also be conveyed by the virus coming into contact with wounds, sores, etc. The eruption shows a predilection for the fingers, the roots of the nails, and the mouth. Constitutional symptoms are also present, such as fever, gastric disturbance, and diarrhœa.

The vesicular eruption may extend to the fauces and pharynx. The disease in man usually runs a favourable course, but in one epidemic reported by Siegel the mortality reached 8 per cent. Young children are especially susceptible, and fatal results may ensue. We have no evidence of the disease being transmitted by means of the meat from affected animals.

The milk from infected animals should not be used by human beings. Boiling the milk destroys the infection therein. Attendants should be careful to wash their hands in antiseptic solutions after handling affected animals, and to cauterise any wounds or abrasions of the skin that may have come into contact with the mouth, udder, or feet of affected animals.

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Report of Board of Agriculture, 1910.

STRANGLES

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Infectious rhino-adenitis; Febris pyogenica; Morbus glandulosus; Distemper of horses.

French : Gourme.
German : Drüse.
Italian : Cimorro, adenite equina.

Definition.—An infectious febrile disease of the equine species, believed by some authorities to be due to the *Streptococcus equi*, and characterised in its benign form by diffuse catarrhal inflammation of the upper respiratory passages, with or without the presence of abscesses in the submaxillary lymphatic glands and surrounding connective tissue. In its irregular form abscesses may occur in any region or organ of the body. Various types of the disease are met with and complications are common.

GENERAL REMARKS.

In the present day our knowledge of this disease, from a clinical and pathological point of view, has been considerably modified.

Although for a long period it was recognised by many authorities that the presence of tumefaction and abscess formation in the submaxillary lymphatic glands was not an essential feature of strangles, still the fact was by no means universally accepted. Unless abscesses appeared in the above region, or in other parts of the body, many practitioners regarded the catarrhal symptoms presented as depending on acute coryza, pharyngitis, laryngitis, or some form of influenza. Further clinical observation, however, combined with pathological investigation, has demonstrated that strangles may manifest itself by catarrhal symptoms attributable to affections of the upper respiratory passages alone, such as acute coryza, laryngitis, or to pharyngitis, etc., and that a horse so affected may infect another, and produce in the latter what is regarded as the typical feature of the disease—viz., the formation of abscesses in the submaxillary lymphatic glands.

The recognition of this fact is of the greatest importance, as it assists in explaining the occurrence of the disease in stables where a typical case was not previously observed, and it also proves of marked value with reference to the adoption of prophylactic measures.

Many authorities are doubtful as to the propriety of classifying strangles as a disease distinct from influenza and allied affections.

The lack of definite knowledge on the bacteriology of the disease, and the similarity of many of the symptoms to those of the above affections, has led many observers to the belief that all these diseases have a primary origin in common, and that the *Streptococcus equi* is not the sole etiological factor in the production of strangles.

This matter will receive due attention later on.

History.—Strangles received attention from many of the early writers.

In 1609 it was described by Blundevill ("On Horses"), and in 1623 by Markham ("Markham's Master-Piece"), under the heading of "The Strangle." In 1664 Solleysell gave an accurate description of the clinical characters of typical cases of the disease, and pointed out its contagious nature. In 1790 Lafosse conducted experiments, and proved the contagious nature of the disease by inoculation. These were confirmed by Viborg in 1802 and by Toggia in 1823. Other authorities, including Charlier, Reynal, Damalix, and Martin, arrived at similar conclusions.

Percivall, in his "Hippo-Pathology" (1834), gave an excellent clinical description of the disease, and pointed out that the presence of abscesses was not an essential feature. Reynal (1866) expressed a similar view.

In 1873 Rivolta discovered microccoci in the pus obtained from the abscess of strangles, which were either isolated, or in the form of chains composed of from three to five organisms.

In 1888 Schütz discovered and described the micro-organism which is now regarded by many authorities as the definite cause of strangles—viz., the *Streptococcus equi.*

Sand and Jensen, Poels, Lupke, and Hœhne, isolated and described a similar micro-organism in the same year.

Animals Affected.—The disease is very common in horses, less frequently met with in mules, and is rare in donkeys. We have no evidence of its occurrence in cattle, sheep, swine, dogs, or birds.

ETIOLOGY.

Although the *Streptococcus equi,* also known as the *Streptococcus of Schütz,* is accepted by many authorities as the specific micro-organism of strangles, there are several eminent investigators who express doubts on the subject, and certainly the evidence on which their scepticism in this direction is founded is very convincing.

Hence, in considering the etiology of the disease, it will be desirable to describe the characters of the *Streptococcus equi,* and to state briefly the grounds on which it is claimed to be specific. Then it will be necessary to consider the objections that have been brought forward with reference to the acceptance of the view that this micro-organism is the specific cause of the disease.

The *Streptococcus equi* can be obtained from the nasal discharge, from the pus contained in the abscess, from the pleural and pulmonary exudate when the pleura and lung are involved, and from the skin eruptions.

According to Nocard and Wiart, the streptococci can be isolated from the blood in cases of strangles of the hæmorrhagic type.

Champetier has isolated cocci united in a chain of two, four, or five, from the blood, urine, and œdematous swellings of horses suffering from the exanthematous type of strangles, and Jacoulet has made similar observations.

The usual form of the organism is an undulating chain of cocci, each chain being made up of thirty to sixty organisms.

The length of the chain varies according to the situation. In the throat abscesses longer chains are found than in the nasal discharge; in the latter they are short, and individual cocci and diplococci are chiefly met with. The streptococcus is found pure in unbroken abscesses, while in the nasal discharge it is associated with other micro-organisms. It is both aërobic and anaërobic, non-motile, grows on most media at ordinary temperature, and can be stained by all aniline dyes, and also by the method of Gram. Reproduction occurs by transverse division.

Vitality.—According to Nocard and Leclainche, it is destroyed by sunlight in eight hours, and by desiccation in three weeks. Hutyra and Marek found that solutions of perchloride of mercury (1 to 1,500), carbolic acid (1 to 200), and cyllin (1 to 80), killed it in fifteen minutes.

According to Cadéac, a horse inoculated with pure cultures develops an attack of strangles, with all its clinical characters.

Intravenous inoculation does not produce any apparent general infection, but may cause an acute phlebitis at the point of injection, according to Jensen and Sand.

Cattle, sheep, pigs, dogs, and birds, do not contract the disease when inoculated with cultures. Cadéac states that the *Streptococcus equi* is a saprophyte, that it does not differ from other streptococci, and cannot be regarded as specific. He believes that under unknown causes it is rendered virulent and capable of producing epidemics of strangles.

"Numerous passages of the micro-organism from animal to animal of the same species exalt continually their virulence for this species and for certain tissues, so that the ordinary streptococcus finishes by assuming the rôle of a specific microbe." *

It is now well known that a streptococcus indistinguishable from the *Streptococcus equi* exists in the upper air-passages of normal horses, also in the alimentary canal and in the ingesta, while in cases of ordinary nasal catarrh similar micro-organisms occur.

Again, Jensen and Sand have discovered the streptococcus of strangles in the lungs of horses that have died of pneumonia, and Cadéac has observed a similar micro-organism in the most varied affections of the respiratory organs.

According to Lignières,† the streptococcus of strangles cannot be distinguished by the appearance of the chains or by the length of the latter. The chief distinguishing features are—(1) Its *pathogenic* action, when cultures are inoculated into certain animals; (2) the *effect of serotherapy* on the affected subjects. He differentiates the streptococcus of strangles from the pyogenic streptococcus, or that of erysipelas, by the different effects produced on mice and rabbits when inoculated with cultures, and also demonstrates that subcutaneous injections of pyogenic streptococci in foals are well borne, and do not show a marked reaction; while injections of the strangles streptococci in these animals have a powerful reaction, especially in the region of the sternum, a large swelling resulting, followed by an abscess, the pus of which is white, homogeneous, and creamy, and teeming with long chains easily stained by the method of Gram.

Again, he states that the serum produced from one of these types of streptococci is *active against all the streptococci of the same type*, but is practically inactive against streptococci of another type. Thus antistreptococcic serum obtained from pyogenic streptococci has no effect on strangles streptococci.

Labat (1909), in a paper on the same subject, states that it is impossible to differentiate the strangles streptococcus by the behaviour of cultures on different media, or by the character of the cultures and length of the chains. He believes that in cases of virulent strangles the streptococcus of Schütz is associated with the pyogenic streptococcus.

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* "Pathologie Interne."

† Paper on "The Pathology and Therapeutics of Streptococci in Domestic Animals," read before the International Congress of Veterinary Medicine at the Hague, 1909.

Some authorities are of opinion that the strangles streptococcus is identical with the micro-organism on which contagious pneumonia is believed to depend.

The frequent association of strangles and pneumonia is well known to clinicians, and further investigation is necessary before the question can be settled from a pathological point of view.

Others advance the theory that the primary cause of strangles is an ultra-visible virus, which has the effect of weakening the general resistance, especially of the upper air-passages, by setting up a condition of catarrh, and thus permitting the entrance and development of the *Streptococcus equi.*

From the various opinions which we have quoted above, it is apparent that further investigation is required before the *streptococcus of Schütz,* also known as the *Streptococcus equi*, can be accepted as the definite cause of strangles.

McFadyean* expressed doubts as to the *Streptococcus equi* being the actual cause of strangles. In the following lucid manner he pointed out that the evidence brought forward to establish the specific character of the micro-organism was not convincing:

" The mere fact that the strangles streptococcus produces suppuration is not sufficient to prove that it is the cause of strangles.

" What would satisfy one that the streptococcus is the actual cause of strangles would be a series of experiments in which it was shown that, by using a culture of the streptococcus, one could infect a horse, and that the artificially infected horse would then hand on the disease to other susceptible horses placed in contact with him; but so far as I am aware that has never been done, and I very much doubt whether, if the experiment were tried, it would have a positive result. . . .

" I admit that, in the absence of such crucial experiments as these, one might find in another direction evidence to convince us that the streptococcus is the actual cause of strangles. If, for example, it should be shown beyond question that one can immunise horses against the natural disease with the streptococcus or its products, then I think that would be proof that the streptococcus is the actual cause of the disease."

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* Discussion on Strangles, National Veterinary Association, London, 1910.

PREDISPOSING CAUSES.

Conditions Favouring Infection.

Cold, Chills, and Fatigue.—These, by reducing the natural powers of resistance, are recognised as predisposing causes of strangles. Debility from any cause also renders a horse more susceptible to the infection.

No one in the present day believes in the spontaneous origin of strangles, although it is often difficult to trace the source of infection in certain instances. McFadyean points out that too much importance should not be attached to the above causes, as any young horse not previously attacked by the disease may not be capable of resisting infection, no matter how vigorous his condition may be at the time he is exposed to infection.

This opinion is supported by clinical experience, as we are fully aware that horses may be exposed to all these unfavourable conditions without contracting the disease, while, *per contra,* horses kept under the best conditions and in good health may be attacked, provided they are exposed to infection.

The Influence of Ill-ventilated, Overcrowded Stables.—The subjection of horses, especially young animals from the country, to the above influence no doubt interferes with the natural powers of resistance; but we cannot overlook the fact that such animals are exposed to infection, not only from cases of the disease that have recently recovered, but also from infected stables, fodder, etc.

Age.—Young horses are most susceptible to infection, but in many epidemics of the disease we find that horses of all ages are attacked.

Major Todd finds that in horses over six years of age, and in those suffering from secondary attacks, the atypical form of strangles is that usually met with.

This is not our experience, as we have seen the so-called typical forms of the disease in a large number of outbreaks in horses of all ages.

The irregular or virulent type of strangles is more commonly met with in foals and yearlings.

The Influence of Coryza and Allied Affections.—Catarrh of the upper air-passages, so common an affection in town stables, no doubt predisposes a horse to strangles when he is exposed to the infection. The altered condition of the mucous membrane offers a ready entrance to the micro-organism of strangles.

It must be remembered, however, that nasal and pharyngeal catarrh may be themselves premonitory symptoms of strangles, and that they also depend on a streptococcus which cannot be distinguished from the

Streptococcus equi. Cases of influenza which develop so-called typical symptoms of strangles during their course are not uncommon. Probably the infection of strangles has been introduced into the stable by some means, and the subjects of influenza, having their vitality lowered, are rendered more susceptible to the secondary infection. But a mixed infection from the start is not improbable, as in some outbreaks we may find in the same stable cases of ordinary influenza, of so-called typical strangles, and of pleuro-pneumonia, or a combination of strangles with either of the two affections named.

IMMUNITY.

Some difference of opinion exists among authorities with reference to the immunity conferred by an attack of the disease.

According to Cadéac, animals may be attacked several times, and recurrence may take place at different periods of the animal's life. The first attack has no immunising effect, but the recurring attacks are usually of a benign character. If the first attack be a severe one, recurrence is less likely to take place, but after a time the animal is again liable to be infected by the streptococcus. "Absolute immunity never exists."

The relative immunity of older horses is probably due to the fact that they have been affected on a previous occasion.

According to Friedberger and Fröhner, "one attack confers immunity for a couple of years at least, and frequently for the remainder of the horse's life." Major Todd states: * "A natural attack of the disease confers a varying amount of immunity, the duration of which is difficult to ascertain." General Smith, in discussing the subject of strangles at the National Veterinary Association meeting (1910), remarked: "As to repeated attacks of strangles, I know nothing whatever about them. I have seen horses attacked a second time, but so very few that I should think probably not two or three in my lifetime." According to Nocard and Leclainche, "a first attack only confers a partial and temporary immunity; the disease may recur several times in the same animal at irregular intervals."

From the above opinions of competent observers it is apparent that further investigation is necessary in order to obtain definite information with reference to the question of immunity in this disease. In private practice it is very difficult to obtain statistics on the subject, as we cannot be aware of the previous history of the horses coming under our observation. In army horses, however, the opportunities are far more favourable for deciding the question.

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* Paper read before the National Veterinary Association, 1910.

It is by no means necessary that every horse should suffer from an attack of strangles, more especially in the present day, when attention is directed to isolation and disinfection.

When an outbreak of the disease occurs in a stable, it is impossible to ascertain whether the horses attacked had previously suffered from the affection or otherwise, or whether those that escaped infection were rendered immune by a previous attack. Judging by the large number of cases we meet with in adult horses during an epidemic, it would seem as if the views of Cadéac were fairly correct. Probably the degree of virulence of the infecting agent may exert some influence in such cases.

INFECTION AND MODES OF INFECTION.

As already stated, what would appear to be sporadic cases of the disease are met with in which no obvious source of infection can be determined. Remembering, however, that horses recently recovered from the disease may act as "carriers" of the infection, although all traces of the affection have disappeared, also horses in the incubative stage of the affection, some at least of these cases can be accounted for, as it is rare for horses doing daily work not to come into contact of some kind with other horses. Infected fodder must be considered in connection with this subject, also infected water. Experiments in connection with the vitality of the organism have demonstrated that it can resist desiccation for three weeks; hence a possible source of infection may exist in recently-purchased fodder contaminated within the above period with the virulent material of strangles. We have no evidence with reference to the length of time that the organism may remain virulent in water.

In connection with the occurrence of cases in which even the above etiological factors can be eliminated, Cadéac's view that the *Streptococcus equi* may exist as a saprophyte and become pathogenic from unknown causes is accepted by some as a possible theory.

The following *modes of infection* are recognised, but opinions differ as to which is the commoner and more important, so far as those by inhalation and ingestion are concerned.

1. **By Inhalation.**—Friedberger and Fröhner regard this mode of infection as the most common.

They state: "The contagium is absorbed chiefly through the respiratory mucous membrane. The high initial internal temperature, which is manifested even prior to the swelling of the glands, and the experimental observations of Schütz, Jensen and Sand, prove that the infective matter may be taken into the blood directly from the mucous membrane. In its further progress the contagium keeps to the direction of the lymph-passages, and enters by preference into the lymph glands, in which it excites extensive suppuration. As was shown by Schütz, the cocci of strangles can also pass through the walls of the capillaries without the help of the blood or of the lymph, and can penetrate between the tissue cells of the affected organs." *

Cadéac regards the respiratory passages as the principal route of infection in strangles.†

McFadyean expresses the following view on the subject: "Until experiments have been conducted to show what is the minimum amount of strangles infection that will certainly infect by the air-passages and the minimum amount that will certainly infect by the digestive tract, our opinion as to what is the common method of infection in strangles should be suspended."‡

Major Todd, in the paper already referred to, is of opinion that it is unusual for the disease to be transmitted by inhalation, and points out that it would necessitate the organism entering the body in a desiccated state.

With reference to the entrance of the virus by way of the nasal mucous membrane, Cadéac draws attention to a number of important experiments, as follows: Viborg found that simply rubbing the nasal mucous membrane with the pus of strangles produced the disease. Moussis (1822-1832) confirmed these results by forty experiments, consisting of a simple impregnation of the nostrils of healthy foals with the nasal discharge from a case of strangles, or in applying friction to the nasal mucous membrane so as to insure inoculation. C. Martin, in a series of experiments, found that by rubbing or producing erosions or excoriations of the nasal mucosa with objects infected by strangles pus, muco-pus, or nasal discharge, strangles was produced, or an acute inflammation of the upper respiratory passages. It was found that these experiments were negative when carried out on horses that were previously attacked by the disease. Joly conducted experiments with similar results.

Schütz was of opinion that a solution of continuity of the mucous membrane was not necessary for infection to occur. According to his observations, the micro-organisms are introduced with the inspired air, and colonize in the folds of mucous membrane; they induce irritation of the membrane, and penetrate slowly into the deep lymphatic areas.

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* "Veterinary Pathology," 1908. (Translation.)

† Cadéac, "Pathologie Interne."

‡ Discussion on Strangles, National Veterinary Association, 1910.

Injection of a culture of the streptococcus into the nostrils or larynx of a foal is followed after some days by catarrhal inflammation of the mucous membrane and the formation of an abscess in the submaxillary glands.

Cadéac found that direct penetration of the streptococci into the bronchi and lung is exceptional; the pulmonary lesions are generally the result of a progressive and descending infection, and are secondary.*

An important point in favour of inhalation being the common mode of infection in strangles is that the primary lesions in most cases occur in the upper respiratory passages.

Major Todd carried out experiments similar to those mentioned above, but the results were negative. As the subjects were chiefly aged horses, the experiments do not in any way negative the conclusions of the authorities mentioned. However, as he points out, "they show that aged horses in good health and living under favourable conditions possess an extraordinary high degree of resistance to enormous quantities of virulent infective material."†

2. **By Ingestion.**—This method of infection of late years has received more attention from both pathologists and clinicians. Up to the present time it has not been definitely decided as to whether inhalation or ingestion is the more common mode of infection.

Solleysell observed that the disease could be communicated to horses by drinking from the same buckets to which affected animals had access.

Cadéac does not favour the view of infection by ingestion, and points out that the lesions of the stomach, intestine, and neighbouring lymphatic glands, are intimately associated with a secondary localisation of the virus circulating in the blood or carried by the lymphatics. He quotes the opinion of Bermbach, that in the large majority of cases the streptococci are absorbed in the vicinity of the mouth and pharynx, and subsequently reach the respiratory passages.

Friedberger and Fröhner state: "It is probable that the contagium may also enter the body from the intestinal mucous membrane."‡

General Smith, in testing the value of certain sera from a preventive point of view, administered strangles virus mixed in the food to a number of horses, and found that the animals almost invariably contracted the disease.§

Major Todd is of opinion that ingestion is by far the most common method of infection.

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* Cadéac, "Pathologie Interne."

† Paper on Strangles, National Veterinary Association, 1910.

‡ "Veterinary Pathology."

§ Discussion on Strangles, National Veterinary Association, 1910.

Further research is necessary before a definite opinion can be given as to the frequency of infection by ingestion. From a consideration of the opinions expressed by authorities, and also from clinical observation, we are led to believe that infection may occur both from inhalation and ingestion, and, in directing prophylactic measures, it is important to keep this fact in view. Hence the probable vehicles of contagion, such as infected food, litter, manure, water-troughs, mangers, buckets, etc., must receive attention in our efforts to prevent the spread of the disease.

3. **By Abrasions or Wounds of the Skin.**—From 1857 to 1863 C. Martin conducted experiments in connection with the production of strangles by inoculation. Inoculation of muco-pus obtained from a case of strangles, carried out by means of punctures around the nostrils and in the upper lip of a four-year-old horse, were followed by local lymphangitis, cough, nasal discharge, fever, submaxillary and parotid abscess. In further experiments twelve out of fourteen horses contracted the disease after inoculation, and of this number seven transmitted strangles to healthy horses placed in contact with them.

According to Joly and Leclainche, inoculations of material obtained from the cutaneous eruptions of strangles, carried out by means of scarifications of the skin, produced in a healthy horse local manifestations, followed by a generalised eruption.* Surgical operations, especially castration, may be followed by strangles. In such circumstances it may result from *direct* inoculation, such as when the infection is introduced into the wound by the hands of the operator, the latter having recently been engaged in the surgical treatment of a strangles abscess; or the instruments may have been contaminated from placing them in infected vessels; or the attendant, in dressing the wounds, may convey the infection by his hands, buckets, sponges, etc., should he be attending a case of strangles on the premises.

The usual lesion produced in infected castration cases is the formation of an abscess in the inguinal region prior to the appearance of other phenomena (see p. 806).

In some instances a series of typical strangles abscesses are found superficially situated, such as in the region of the sternum and extending towards the abdomen, without any direct history of a wound and no other evidence of the disease. It is highly probable, however, that some abrasion of the skin was present. In one instance we have seen a case of this kind which infected the other horses in the same stable, and produced in them an ordinary attack of strangles.

* Cadéac, "Pathologie Interne."

A horse, if recently castrated and exposed to infection, may develop a severe form of strangles, with the formation of secondary abscesses in the inguinal region and at the seat of the operation wounds. It must be remembered, however, that as the result of all operations of any severity the vital resistance of an animal is lessened; hence he is more receptive to infection. Again, it is quite possible for an animal to be in the incubative stage of the disease prior to operation; hence we cannot attribute all cases of strangles occurring subsequent to operations as depending on direct inoculation by way of the operation wounds. These latter should be limited to those instances in which the first indication of infection is found at the seat of operation. Cadéac points out that the streptococci tend to accumulate in regions which are the seat of acute or chronic inflammation.*

Congenital Infection.—Infection from the dam to the fœtus may take place during intra-uterine life. This subject has been studied by Martin, Choisy, Cadiot, Pecus, and Nocard. According to Nocard, one of the results of this infection is the occurrence of abortion. If the fœtus is close to full development, it possesses more vitality and resisting power, and may show the usual evidences of strangles. At an earlier period death usually results. The lesions are chiefly met with in the digestive system, and in the lungs, liver, and kidneys.

Infection by Coitus.—An infected stallion may convey the infection to a mare, and produce what is termed "genital strangles." The lesions are found on the vaginal mucous membrane, on the vulva, perineum, and internal surface of the thighs (see p. 808).

Of course, infection may occur from stallion to mare and from mare to stallion during the process of "trying" a mare when either of the animals is suffering from the disease; in such instances the infection would be conveyed in the usual manner.

Infection by the Udder.—A foal suffering from the disease may communicate it to the dam during the act of sucking. The streptococci gain entrance through the openings of the teats, reach the interior of the udder, and produce mammary abscesses. *Per contra,* a mare suffering from a strangles abscess in the udder can infect her foal. Of course, infection from nasal discharge, etc., may also occur from the dam to the foal, and *vice versâ.*

Mode of Extension.—In ordinary mild cases the streptococci do not extend far from the point of entrance, but in others extension takes place chiefly by the lymphatics, less often by the blood. In most instances the upper air-passages and pharynx are the primary seats of infection, and from these points the streptococci are carried to other regions by the lymphatics. Occasionally the swelling of the submaxillary glands may precede the symptoms of coryza and pharyngitis, and lead us to suspect a primary infection of the lymphatics of this region.

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* "Pathologie Interne."

Indirect Methods of Infection or Vehicles of Contagion.—It is of importance to recognise the indirect methods of infection in considering the prevention of the disease. The facility with which horses suffering from strangles can contaminate any material or objects in their vicinity is readily recognised when we consider the extensive nasal discharge, the fits of coughing, and the flow of pus from the abscesses when the latter are surgically treated or rupture spontaneously. Thus food, water, feeding utensils, watering troughs, etc., become carriers of infection, and prove a fertile cause of spreading the disease unless special precautions be taken. Mangers and the woodwork in stables, grooming utensils, clothing, halters, bandages, etc., become contaminated and act as carriers of contagion. The contagion is more likely to remain in dark, ill-ventilated, badly-drained stables, as in such there is absence of sunlight and less chance of desiccation of the infective material occurring. Livery stables are a fertile source of contagion, as, in consequence of horses coming thereto from various districts, the chances of infection are largely increased. Litter also becomes contaminated, and may produce the disease by inhalation, or by ingestion when it is eaten by the animals. Attendants on horses suffering from strangles may convey the disease to susceptible animals through the medium of their hands or clothes. According to Cadéac, dogs and cats may carry the infection from one stable to another.

PERIOD OF INCUBATION.

According to Cadéac, the period of incubation is from three to five days as the result of experimental inoculation; but in the case of natural infection it may extend to eight days, or even more.

Major Todd places it at from three to eight days, and Friedberger and Fröhner at from four to eight days.

In must be remembered that in some cases the early symptoms of the disease may be so slight as to be overlooked.

FORMS OR TYPES OF STRANGLES.

In former times two varieties of the disease were recognised—viz., a mild, regular, or typical, and an irregular or malignant form.

In the present day, as our knowledge of the affection has been greatly extended, we find that various forms, in addition to those mentioned above, may be met with, and a classification is extremely difficult.

The form once recognised as typical, in which swelling and abscess formation in the submaxillary glands was a prominent symptom, is not now regarded as such; as already mentioned, this phenomenon may be absent in many cases.

The irregular or malignant form may present a variety of lesions in addition to the occurrence of abscesses in various regions of the body.

The disease may start in a stable in a simple or benign form, and, after passing through a varying number of horses, it may assume the irregular or malignant type.

A septicæmic form of the disease is also recognised; this corresponds to the hæmorrhagic variety described by Wiart (see p. 814).

MORBID ANATOMY.

There are few diseases which show such widely-spread, diverse lesions and complications as the irregular or malignant types of strangles. The lesions may be localised, when the micro-organisms do not extend far beyond the point of entrance and the ordinary type of the disease is produced; or they may be generalised to a greater or less extent, according to the spread of the micro-organisms throughout the system. The leading pathological processes are catarrhal inflammation and abscess formation. The latter may be primary or secondary. The structures chiefly affected are the lymphatic glands, lymphatic vessels, and parts well supplied with connective tissue.

It will be convenient to describe the lesions in each system as follows:

Respiratory System.—The upper air-passages show most frequently the lesions of the disease.

The Nasal Mucous Membrane is the seat of a catarrhal inflammation which usually extends to the naso-pharynx, pharynx, and larynx. Desquamation of the epithelium occurs, and a free secretion of muco-pus and pus is observed. Occasionally small erosions may be met with in the nasal mucosa.

The larynx may show inflammatory infiltration and an œdematous condition of the mucous membrane, also submucous abscesses may be observed.

The air-sinuses may be involved by extension of the inflammation resulting in the formation of purulent collections therein; also the guttural pouches, leading to distension of the latter with pus, but this is rare.

Bronchi and Lungs.—The inflammation may extend to the trachea and bronchi. The bronchitis may occur early in debilitated subjects, or when the streptococci are of a virulent type. In other instances it is secondary, and appears in cases progressing towards convalescence, especially when they are exposed to chills and fatigue. Bronchopneumonia may occur under similar conditions. The lungs may show congestion or various forms of pneumonia. Swelling and suppuration of the bronchial glands may be present. The pulmonary lesions may depend on direct infection when the latter extends from the upper respiratory passages, or pus from pharyngeal abscesses may enter the bronchi, or they may arise indirectly through infection carried by the blood.

According to Cadéac, the toxins formed by the streptococci exert a vasodilatatory effect, resulting in the production of congestion and the localisation of the micro-organisms in the lungs, pneumonia being thus induced.

The pneumonia may be unilateral or bilateral. When the pulmonary lesions are secondary, abscesses in the lung and necrotic centres are frequently met with.

Pleura.—Pleuritic lesions may coexist with pneumonia, or the pleura may be involved by extension of the infection along the lymphatics at the entrance to the thorax, or from infection carried by the blood, or from rupture of an abscess of the bronchial glands, or of a mediastinal abscess. In the latter instances purulent pleurisy results.

Occasionally pus from an abscess in the region of the throat may burrow behind the trachea, and gain an entrance to the pleural cavity, inducing purulent pleurisy.

Digestive System.—The *pharynx* is one of the commonest seats of primary infection in strangles; hence pharyngitis is a constant symptom of the disease, and is generally associated with laryngitis.

In the submucous tissue of the pharynx small abscesses are often found, which contain thick creamy pus; in some instances the latter is caseated. Occasionally abscesses may occur between the pharyngeal muscles, which may become confluent and rupture through the skin externally, or burrow into the pharynx. In rare cases a pharyngeal fistula may result. The buccal mucous membrane shows congestion, and the submucous tissue may be infiltrated with a hæmorrhagic exudate.

Acute glossitis may be observed in some cases. The soft palate in some instances shows marked swelling, and either disseminated abscesses or a solitary abscess. Occasionally necrotic patches are present.

Stomach.—Gastritis may result from extension of infection from the upper digestive and respiratory passages, or occurs as a secondary infection by way of the lymphatics and blood-stream. Ecchymoses may be observed on the gastric mucous membrane, and abscesses may be present involving the mucous follicles and the submucous connective tissue, sometimes resulting in rupture of the stomach.*

Intestines.—Infection results in a similar manner to that already mentioned as occurring in the stomach. The lesions observed may be enteritis, suppuration of the intestinal follicles, and abscesses in the submucous coat of the jejunum or ileum, the terminal portion of the floating colon, the walls of the rectum, and the peri-rectal connective tissue. These abscesses may be large in number and of various sizes, and in their vicinity the intestinal tube is narrowed, ulcerating, and may be gangrenous. Abscesses of large extent may be found at the terminal portion of the floating colon and in the peri-rectal connective tissue.†

Liver.—The lesions are secondary, and consist of suppurative hepatitis and the formation of abscesses, which may attain a large size.

Pancreas.—Multiple abscesses may be found in this organ.

Peritoneum.—Peritonitis of a septic character occurs when abscesses rupture into the peritoneal cavity. It is also met with in cases where an animal develops strangles shortly after the operation of castration has been performed (see p. 806).

Spleen and Kidneys.—Congestion, hypertrophy, and abscesses of the spleen may be present. Abscesses may be found in the kidneys. According to Cadéac, peri-renal abscesses are not rare; they may contain a large amount of pus. He records one case in a foal in which the abscess burrowed through the flank and discharged externally.

Lymphatic System.—This system is the special seat of lesions in strangles, and it is through the lymphatic vessels that extension of the disease in the body generally occurs. In ordinary cases the submaxillary lymphatic glands are those most frequently affected; as already remarked, this lesion is not an essential one in strangles. Probably the infection is carried to these glands through the lymphatics of the nasal cavities.

In the early stages these glands are enlarged and hard, due to exudation occurring therein. In a variable period suppuration occurs, and an abscess is formed containing white creamy laudable pus. Either one or both glands may be affected. The surrounding connective tissue may be involved, resulting in an extensive swelling that fills up the intermaxillary space. In other cases the infection spreads farther, and the parotid lymph glands, the retropharyngeal, and the lymph glands just below the base of the ear, become involved. In the irregular type the lymphatic vessels and glands in any part of the face, head, and neck may become affected, and abscesses are formed. A lymphangitis occurs, and along the course of the lymphatic vessels nodules of various sizes form and suppurate. Superficial abscesses may form in the subcutaneous tissue. These phenomena may occur on the lips, nostrils, face, eyelids, inferior maxillary region, the withers, shoulders, and thoracic region.

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* Cadéac, "Pathologie Interne."

† *Ibid.*

The infection of the lymphatics and lymphatic glands may occur in any region of the body. Extension to the bronchial and mediastinal glands is common, and abscesses are formed in these regions. Mediastinal abscesses may attain a large size.

Infection of the mesenteric glands may occur through extension from neighbouring lymphatic glands or through the blood-stream. These abscesses are of various sizes and slow in development. They may cause pressure on the intestines, the posterior vena cava, or on any of the organs of the abdominal cavity, or they may rupture into the peritoneal cavity.

When strangles occurs as the result of infection following the operation of castration, the abscess develops in the inguinal region. According to Cadéac, "the micro-organisms are deposited in the connective tissue at the seat of operation, and increase slowly, so that evidences of the lesion are not observed until all inflammatory symptoms due to the operation have disappeared. The region of the sheath shows an œdematous swelling, which rapidly increases in extent and becomes invaded by purulent centres."

"An abscess also forms between the internal face of the thighs and the scrotal region. After being treated surgically, fresh swellings soon appear, and new abscesses are formed. These abscesses may take four or six weeks to develop, and they may contain a large amount of pus. The abscess generally develops in the cellular tissue surrounding the spermatic cord, and frequently involves the cord itself, forming in its interior or in the vaginal sheath. The process spreads by the lymphatics to the abdominal cavity and to the sublumbar region. These abscesses sometimes open on the inferior region of the flank."

"A large number of cases of inflammation of the spermatic cord, erroneously described under the term 'champignons,' are in reality external local abscesses, or abdominal or erratic abscesses, and should be attributed to strangles infection. In this form of strangles the neighbouring lymphatics may become involved, as in ordinary cases of the disease. Hence secondary abscesses may be met with in the hind-limbs, especially in the region of the hocks, also metastatic abscesses in the lungs, etc. The metastatic abscesses which develop in the course of strangles depend on the transport of the micro-organisms by the blood-stream and their colonization in various organs." *

Pericardium and Heart.—Pericarditis may be associated with the pleurisy of strangles. Cases are recorded in which abscesses have been found in the ventricular walls or in the interventricular septum of the heart. According to Zschokke, endocarditis may occur in connection with strangles. Vegetations have been observed on the tricuspid valve, and generalised abscesses in the heart.

Nervous System—*Cerebrum.*—Cerebral congestion, meningitis, cerebral abscess, evidences of cerebral hæmorrhage, encephalitis, etc., may be observed. According to Cadéac, the majority of cerebral abscesses originate from the infection of strangles.

Spinal Cord.—Spinal meningitis and abscesses in the spinal canal and in the spinal cord have been observed. Meningitis has resulted from an abscess perforating the walls of the spinal canal in the cervical region, also in the lumbar region.

Various observers have described lesions of the spinal cord, especially well marked in the lumbar region (lumbar enlargement), consisting of acute congestion, capillary hæmorrhages in the spinal meninges, engorgement of the venous sinuses, effusion into the arachnoidean space. The lesions may assume the chronic type (Cadéac).

Muscular System.—Congestion, œdema, hæmorrhages, and abscesses may occur, especially in the intermuscular connective tissue, and also in the muscles themselves.

Joints, Synovial Sheaths, and Bursæ.—Arthritis and synovitis occur in some cases. The infection is probably carried by the blood. Streptococci have been found in the synovial exudate. The lesions may be of an exudative or purulent nature.

The great sesamoidean sheath is frequently affected; the carpal sheath, the stifle, shoulder, and fetlock joints may also be attacked, either singly or together. Cadéac has observed that the streptococci have a tendency to invade joints which had previously suffered from various chronic affections. Open-joint and necrosis of tendons have occasionally been observed as sequelæ. Instances are recorded in which laminitis has resulted from infection of the keratogenous membrane, carried thereto by the blood-stream.

The Bones occasionally show lesions, especially at their peripheral ends, in the joints. Periostitis, caries, and necrosis have been observed.

Mauri records an instance of caries of the inferior maxillary bone, and

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* Cadéac, "Pathologie Interne."

Wiart records cases of periostitis of the knee, hock, and of the metacarpal bones.

Ophthalmic Lesions.—Iritis, effusion into the anterior chamber, posterior synechiæ, and atrophy of the eye, have been observed as complications by Joly.

Cutaneous Lesions.—These are believed to depend on the action of the micro-organisms and of their secretions. Various forms of skin eruption are described in connection with the disease. In some cases a generalised eruption, resembling that occurring in purpura hæmorrhagica, may be observed. In others vesicles and pustules occur, especially on the lateral surfaces of the neck, shoulders, and sides of the thorax. An eczematous eruption is met with in some instances, located especially on the hind-limbs, between the thighs, on the sheath, around the anus, also in the vicinity of the head, neck, throat, and around the eyes, nostrils, and lips.

The eruption may affect various mucous membranes, such as that of the lips, nasal passages, and also the conjunctiva, etc., in the form of vesicles of various sizes. These may rupture, shallow granulating patches resulting, which heal without the formation of a scar. The eruption may also appear in the hollow of the heel and posterior aspect of the fetlock of one or of all the limbs, and extend upwards, becoming generalised. Chronic lesions of the skin may result in some cases. Fissures and ulcerations occasionally occur, and a series of attacks may be observed.

Lesions in Genital Organs.—These occur in the mare as the result of infection from the stallion during coition. According to Nocard and Leclainche, the lips of the vulva become œdematous, and a discharge, first of a yellowish serum, then of muco-pus, from the inferior commissure is observed. According to Cadéac, " vesicles and pustules develop on the vagina, mucous membrane, and on the fine skin of the lips of the vulva, perineum, and internal aspect of the thighs. " The pustules rupture, and form small circumscribed ulcerous patches. The swelling of the perineum may extend to the udder. Lymphangitis occurs, and superficial and deep abscesses form, the pus from which contains the streptococci. The abscesses in the udder persist and recur."

SYMPTOMS.

Strangles being a disease that assumes such a variety of types, it is apparent that the symptoms presented will vary accordingly.

Although attempts have been made to classify these types for the purposes of clinical description, we find it difficult to ascertain in many instances what form the disease is likely to take. Thus a case may start with symptoms which are usually regarded as characteristic of the regular or benign form of the disease, and, without apparent cause, may assume the characters of the irregular form.

Again, a horse suffering from the regular form may infect another horse, and produce in him a severe or irregular attack of the disease. Even the regular forms of the disease show marked variations in severity.

As already mentioned, the presence of swelling and abscess formation in the submaxillary glands was at one time regarded as a characteristic symptom of ordinary strangles. More careful clinical observation, however, has demonstrated that this symptom may be absent, the animal only showing what might be regarded as evidences of acute nasal catarrh, pharyngitis, or laryngitis, and still be capable of infecting another horse, and the latter may develop the lesions in the submaxillary glands in addition to the catarrhal symptoms.

Some writers describe mild cases of the disease, showing an absence of submaxillary or parotid lesions, as "atypical." If we accept the latter term, then typical cases are those in which the following symptoms are shown—viz., fever, nasal catarrh, swelling terminating in the formation of an abscess in the submaxillary glands and surrounding connective tissue, or in the region of the parotid gland, and a varying degree of pharyngitis or laryngitis. In this class of cases variations are found as regards mildness or severity of symptoms. Very frequently the early symptoms presented are: nasal discharge, indicating coryza; difficulty in swallowing, due to pharyngitis; cough; loss of appetite; and a varying degree of constitutional disturbance.

The swelling of the glands in the intermaxillary space may appear concurrently with the above symptoms or some days afterwards, or the swelling of the submaxillary glands may be the first symptom to attract attention.

In very mild cases slight nasal catarrh, a moderate swelling in one or both submaxillary glands, a slight cough, and little or no constitutional disturbance, are the only symptoms observed.

Such cases are often met with amongst young horses at pasture, and recovery without any treatment is frequently observed, the abscess in the submaxillary gland rupturing spontaneously. In some instances the swelling disappears without abscess formation.

Fever.—In cases of a severe type fever is a prominent symptom; the temperature may rise to 103° or 105° F., and oscillations are common. As a rule it reaches its height when suppuration occurs in the submaxillary gland, and tends to decline when the abscess either ruptures spontaneously or is surgically treated. If, however, another abscess appears, the temperature again rises. In the mild cases the temperature may not exceed 102° F. Occasionally even with abscess formation the temperature may remain normal.

Nasal Catarrh.—This is evidenced by a nasal discharge, at first serous in nature, subsequently becoming muco-purulent. The amount of discharge varies according to the severity of the coryza and the degree of pharyngitis and laryngitis present.

Swelling and Abscess Formation in the Submaxillary Glands.—In the early stages either one or both glands are swollen, firm, hot, and painful on palpation. Suppuration and abscess formation may occur in a comparatively short space of time, or in some cases this process may develop slowly. In the centre, or at various points on the surface of the swelling, soft areas appear, and evidences of fluctuation are observed on palpation. The skin over these areas becomes tense, denuded of hair, thin, and of a bluish-red tint. The abscess "points," and may rupture spontaneously in from eight to twelve days by one or more openings. The pus is yellowish-white in colour, creamy in consistence, and may contain shreds of tissue; the abscess cavity heals by granulation.

In some cases suppuration does not occur, the exudate undergoes liquefaction and absorption, and the swelling gradually disappears. Marked variations may be observed as regards the extent of the swelling and the size of the abscesses. In some instances the swelling may extend towards the region of the throat and attain large dimensions. The abscess may be very extensive, the pus deep-seated and very large in amount; or two or three abscesses may form in the swollen area and become confluent.

The lymph glands between the lobules of the parotid gland may become involved, an extensive swelling being produced in this region, terminating in the formation of an abscess; or the lymph glands in the vicinity of the temporo-maxillary articulation become swollen and suppurate, a large abscess being formed under the ear. In such instances the pus is often very deep-seated, and considerable tension is produced on the overlying tissues, so that when opened surgically the pus issues with considerable force.

It is not uncommon to find a large abscess in the parotid or temporo-maxillary region in cases that did not present any marked evidences of nasal or pharyngeal catarrh—or, at least, these were not observed by the owners or attendants. In such instances the true nature of the case may be overlooked, unless we remember that the catarrhal symptoms have disappeared prior to our examination.

We meet with cases in which coryza, acute pharyngitis or laryngitis, high fever, and marked constitutional disturbance, are prominent symptoms; and when convalescence seems well advanced, the swelling and abscess formation appear rather suddenly.

Again, during an outbreak of influenza or contagious pneumonia it is not uncommon to find that some of the cases may develop abscesses in the submaxillary glands at any time during the course of the disease. Whether such cases are to be regarded as examples of mixed infection, or whether there is an intimate relation between the micro-organisms of these affections, is a matter that is still undecided.

In the so-called "atypical" forms of strangles there is an absence of swelling and abscess formation in the submaxillary glands, and also of external abscesses in other regions of the body, but abscesses may be present in the interior of the pharynx. The cases present only evidences of nasal catarrh, pharyngitis, laryngitis, etc., in various degrees of intensity; but they are capable of infecting susceptible horses, and producing in them the typical form of the disease.

The atypical form, according to some authorities, is more commonly met with in horses six years old and upwards, and in those which have previously suffered from an attack of the disease. As cases of ordinary catarrh, pharyngitis, etc., also depend on the presence of a streptococcus, further investigation on the atypical form of strangles is necessary in order to obtain definite information on the subject. Probably in these atypical cases some micro-organism will ultimately be discovered in addition to the streptococcus, which will enable us to distinguish them from ordinary cases of catarrh.

Pharyngitis.—This is usually associated with a varying degree of laryngitis. It is evidenced by difficulty in swallowing, popularly known as "sore throat," profuse salivation, extension of the head, the region of the throat swollen and tender to palpation, a thick purulent nasal discharge, frequent cough, which occurs in paroxysms when attempts are made to swallow either liquids or solids.

In severe cases swallowing is impossible, even of the saliva; the latter collects in the region of the lips in stringy masses containing débris of food. When attempts are made to swallow fluids, the latter are returned by the nostrils. In animals at pasture the nasal discharge assumes a greenish colour, due to the secretions from the pharynx being tinged during attempts at swallowing grass.

When severe laryngitis is associated with the pharyngitis, the symptoms are aggravated; violent fits of coughing occur, also evidences of respiratory distress. Asphyxia may result from extensive œdema of the glottis (see Laryngitis, Vol. II.). A roaring sound, varying in intensity in different cases, may accompany strangles. It may depend on œdema of the pharyngeal and laryngeal mucous membrane, abscesses in the retropharyngeal glands, or in the connective tissue around the pharynx, and occasionally from the presence of pus in the guttural pouches.

Constitutional Symptoms.—These vary according to the severity of the case. In mild cases they may be so slight as to be overlooked, and the appearance of a swelling in the intermaxillary space, followed by an abscess, may be the first indication of the affection.

In more severe cases dulness, loss of appetite, disinclination for movement, and alterations in the character of the pulse, are observed.

Loss of condition is a frequent symptom. The presence of severe laryngitis interferes with respiration; pharyngitis prevents deglutition; while the presence of toxins produced by the micro-organisms is believed to contribute to the constitutional disturbance.

Irregular Strangles.

In this form a variety of symptoms may be manifested, depending on the organs involved. In many instances the symptoms are obscure, such as in the case of the formation of abscesses in the abdominal cavity.

The ordinary or regular type of the disease may be followed by the irregular form. For example, a case which is apparently progressing favourably shows a recurrence of the febrile and constitutional symptoms and the formation of abscesses in various regions of the body. Or, the disease may start insidiously, with obscure symptoms and no external manifestations.

It is not uncommon for the existence of the disease not to be suspected, and its real nature only to be discovered after death by the presence of an abscess or abscesses in the interior of the body.

The irregular form may also show various complications, such as have been described under the heading of Morbid Anatomy.

Respiratory Complications.—These may manifest themselves during the course of strangles, or may appear when the patient is apparently on the road to recovery.

Various forms of respiratory affections may complicate strangles, such as bronchitis, terminating in broncho-pneumonia, pulmonary abscess, pulmonary gangrene, etc., and the symptoms presented are similar to those occurring in the ordinary forms of such affections (see Respiratory Affections, Vol. II.). Pleurisy may also occur, and is manifested by the usual symptoms of that affection.

When pulmonary abscess is present, the symptoms are obscure; emaciation, debility, dyspnœa, and frequent intermissions of fever, may be observed.

Abscesses in the mediastinum may not give rise to any specia symptoms until they rupture; then evidences of purulent pleurisy manifest themselves.

Broncho-pneumonia of a septic type may occur in strangles as the result of purulent discharges, saliva, etc., entering the trachea and bronchi in cases where acute laryngitis is present.

Abdominal Complications—*Mesenteric Abscesses.*—The symptoms are obscure. The patient may have apparently recovered from the primary attack, but becomes dull, has a capricious appetite, becomes emaciated, shows colicky pains, especially after feeding; or tympanites may occur, also constipation or even intestinal obstruction. The back may be arched, the abdominal region tender to palpation and "tucked-up" in appearance, and groans may be emitted when the animal lies down or gets up, or is turned sharply round.

According to Cadéac, the formation of the abscess is usually slow, and adhesions may form between it and the abdominal wall; it may remain encysted, or may perforate the flank. When rupture of the abscess into the peritoneal cavity occurs, evidences of septic peritonitis are manifested. Occasionally the appetite is good, but progressive emaciation continues. In many instances death occurs from debility and emaciation, or from intestinal complications, and the abscess is discovered at the autopsy although its presence was not suspected during life. In exceptional cases rectal exploration may enable us to detect the presence of a mesenteric abscess. Rupture of the stomach has been observed in cases where gastric lesions have been present, also in those associated with a large mesenteric abscess.

There are no characteristic symptoms in cases where the lesions of strangles occur in the liver, spleen, pancreas, or kidneys.

Nervous Complications.—The symptoms vary to a considerable extent.

When cerebral abscess occurs, evidences of meningitis may be observed in some instances, such as delirium and convulsive movements of the fore and hind limbs at first, succeeded by coma and paralysis. In other cases a staggering gait occurs, the animal gets into a sleepy condition, and has a tendency to push his head against the wall; if forced to move, he may fall, especially if turned round sharply.

These nervous symptoms may not appear until a considerable period after convalescence. When the lesions occur in the spinal cord, evidences of "shivering" are presented, also loss of power in the hind-limbs.

Various local forms of paralysis may be met with. Amaurosis has been observed in some cases.

Arthritic Complications.—These are evidenced by lameness and by pain and swelling of certain joints, synovial sheaths, and bursæ.

Symptoms of Strangles affecting the Genital Organs in Mares.—The symptoms generally occur in from four to eight days after coition with an infected stallion. The conditions produced have been described on p. 808.

Animals so affected may convey the disease in its ordinary form to other horses in the same stable.

In some instances mares affected with genital strangles may develop the usual symptoms of the disease—viz., abscess in the submaxillary gland, coryza, and nasal discharge.

Exanthematous Strangles has already been described (see p. 808). Occasionally this type of the disease has been met with in the form of an epizoötic in a stud of yearlings running at pasture.

Septicæmic Strangles.—This corresponds to the *hæmorrhagic* form described by Wiart. It is believed to depend on infection of the blood, and there is an absence of local symptoms.

The symptoms, as described by Bigoteau, are as follows: "The attack is sudden, the animal in a few hours falling into a prostrated condition; he assumes the standing posture, refuses all food, eyes wild and staring, staggering gait, cyanosis of the visible mucous membranes, sometimes ecchymoses on the conjunctivæ, action of the heart violent and tumultuous, pulse small and quick, respirations accelerated, temperature oscillates between 39·5° and 41° C. Recovery is exceptional; the animal succumbs in from two to five days, probably from asphyxia."

We have not met with any cases of this form of the disease, and, judging by the literature on strangles, this type must be rarely encountered, at any rate in Great Britain.

SEQUELÆ.

The most important are *Whistling* and *Roaring*. These are common sequels, and prove a source of great loss, in consequence of the depreciation in value that occurs in animals so affected.

Whistling, according to some authors, depends on a permanent thickening of the laryngeal mucous membrane (see Whistling, Vol. II.).

Roaring may be due to some alterations produced by the disease in the nerve-supply of the larynx, or possibly in the nerve centres. Some authorities believe that this sequel may depend on chronic enlargement of the bronchial lymphatic glands. As pointed out by Cadéac, roaring may result in cases of strangles that were neither severe nor prolonged; so that further research is necessary before the connection between strangles and roaring can be definitely explained.

Amongst other sequels may be mentioned chronic catarrh of the nasal and pharyngeal mucous membrane, also purulent collections in the facial sinuses, induration of the submaxillary lymphatic glands, and occasionally the occurrence of pus in the guttural pouches.

Purpura hæmorrhagica may develop during the course of strangles, or during convalescence.

COURSE.

The course of strangles is very variable. Few diseases show such a tendency to complications and relapses. Even cases that apparently belong to the benign type at first may develop evidences of the irregular form, or of pulmonary complications, during their course. The period at which external abscesses are formed varies to a great extent.

As we have already seen, there are many cases in which no external abscesses develop. In fatal cases the cause of death varies according to the nature of the complications.

PROGNOSIS.

This must always be guarded, as, although in the benign form recovery is the rule, one never knows when relapses or complications may occur even during convalescence. The irregular form is always serious.

In foals the prognosis is generally unfavourable, especially when acute pharyngitis and laryngitis are present; the disease is very apt to assume the irregular type in animals of this age.

The presence of pulmonary complications renders the prognosis distinctly unfavourable in animals of all ages.

DIFFERENTIAL DIAGNOSIS.

No difficulty arises in connection with the diagnosis of cases presenting swelling and abscess formation of the submaxillary glands, associated with nasal catarrh, sore throat, etc.

But in those termed "atypical," showing evidences of nasal catarrh, pharyngitis or laryngitis, and a varying degree of fever, but no external abscesses, it may be impossible to state definitely whether they are cases of ordinary catarrh of the upper air-passages or strangles until another horse in the same stable becomes attacked, and shows the so-called typical form of the latter disease.

Hence the history of the case must receive consideration, especially as regards recent exposure to infection and as to the occurrence of cases of strangles in the same stable or in the locality. Similar remarks will apply to cases in which the abscesses are situated internally and obscure symptoms are presented.

We meet with cases in which small subcutaneous abscesses occur along the course of the lymphatics, especially in the substernal region, but with no appearance of catarrhal symptoms or abscess in the submaxillary region. The real nature of such cases may be overlooked until they are followed by typical attacks of strangles in other horses in the same stable.

It is not uncommon to find instances in which a swelling terminating in the formation of an abscess occurs in the parotid region or below the ear without any catarrhal symptoms. In such it is probable that the evidences of catarrh have been so slight as to be overlooked before the case is brought for treatment; nevertheless, a horse so affected is capable of infecting others with strangles.

Cases which run a chronic course, and those in which small erosions occur on the nasal mucous membrane, must be differentiated from glanders (see Differential Diagnosis of Glanders, p. 87).

The cutaneous form of strangles and the coital exanthema of this disease may require to be differentiated from *Variola equina.* According to Jacoulet (quoted by Cadéac), "the pustules of strangles are less voluminous, less clearly marked, and are found chiefly on the mucous membranes; also they are less inclined to become confluent, they are not accompanied by lymphangitis, and they contain a smaller amount of sero-pus. The amount of secretion from them is slight, it dries quickly, and forms a crust which contains epithelium, epidermis, and hair. A very superficial abrasion results, showing little moisture and cicatrising readily."

PROPHYLAXIS.

This may be considered under the headings of measures for *Isolation and Disinfection*, and of measures for conferring *Immunity*.

The latter will receive attention in the section devoted to Immunisation as applied to Strangles (see p. 822).

Preventive measures are of the greatest importance in this disease. Although in the benign form recovery is the rule, we have to consider how frequently unfortunate sequels, such as whistling and roaring, occur. Again, a horse suffering from the benign form may infect other horses, and produce in them the irregular or malignant type of the disease. Once the affection makes its appearance in a stable, the chances are that few of the occupants will escape ; hence the losses due to incapacity for work will be very considerable in large studs of cart-horses.

Rigid isolation of the affected animals is the first step with regard to prophylaxis. In connection with this we must point out that cases showing only symptoms of coryza, pharyngitis or laryngitis, should also be isolated, as these may be sufferers from the disease in an atypical form, but are capable of infecting other horses with typical strangles.

A common source of infection in town horses is the addition of newly-purchased young horses to the stable. These may have lately recovered from the disease, or may only show slight evidences of nasal catarrh, which may be the premonitory symptoms of strangles. Whenever possible, horses newly purchased should be isolated for two weeks before being added to the stud, and carefully watched so as to discover the early symptoms of nasal catarrh, etc.

The next important step is to deal with the vehicles of contagion such as feeding utensils, fodder, litter, clothing, halters, grooming utensils, etc., and to insure that attendants on the affected animals should not feed or groom healthy horses, or come into contact with them. Contaminated litter and food left in the manger is best dealt with by burning. Thorough disinfection of the walls of the stable, floors, mangers, timber-work, etc., should be carried out. Major Todd advises the use of a blow-lamp on the walls, floors, and mangers, followed by the employment of a suitable disinfectant with a spray-pump. In farmers' stables a liberal application of lime-wash containing Jeyes' Fluid or crude carbolic acid may be used. Feeding utensils should be boiled, also the clothing; the harness that has been on an infected animal should be disinfected.

Operations of any kind should not be performed on animals at farms or establishments where cases of strangles exist ; it is even advisable to postpone operations on animals removed from such infected areas.

Veterinary surgeons should adopt stringent precautions when attending cases of strangles, so as to avoid infecting operation wounds. Rigid disinfection of the hands is essential, also of the instruments and operating-coat. Attendants on cases of strangles should be prevented from dressing wounds in other horses.

The occurrence of cases of strangles at a stud farm is a very serious matter, as mares coming for service from various districts and accompanied by their foals are likely to become infected, and to convey the disease to others on their return home, or such mares may themselves be carriers of infection in the first instance. Moreover, the mortality in foals from strangles is very high, and we are aware of serious losses resulting to breeders from infection occurring under the above circumstances. All such farms should be under veterinary supervision, so that the disease may be detected in its early stages, and all newly arrived mares and foals should be inspected, in order that isolation may be carried out if found necessary. Special attention should be devoted to the question of infection from stallion to mare, and evidences of genital strangles should be carefully sought for.

Preventive measures are more likely to prove successful when it is universally recognised that atypical cases are frequently overlooked, and prove a fertile source of infection. Cases of strangles showing pulmonary complications should be isolated from those suffering from the uncomplicated type of the disease. *Per contra,* cases showing evidences of strangles should not be placed amongst horses suffering from influenza or pneumonia.

TREATMENT.

This will be considered under the headings of (*a*) *Medicinal,* (*b*) *Vaccine Therapy.*

The latter is dealt with separately (see p. 823).

Medicinal Treatment.

In mild cases little treatment is necessary. Attention should be directed to the surroundings of the patient, and, as far as possible, proper ventilation and drainage of the stall should be secured. The diet must be easy of digestion and nourishing. Bran and linseed mashes, oatmeal gruel, hay tea, pulped roots, and grass in moderation (when in season). may be allowed.

When the abscess "points," it should be opened by a free incision, the contents evacuated, and the cavity irrigated with an antiseptic solution and packed with antiseptic gauze, the dressing being changed once daily.

Surgical interference is contra-indicated, and may prove harmful before the abscess has matured, as the glandular swelling is liable to become indolent, and may even assume a septic character, as the result of an incision at this stage.

Should the glandular swelling be large and show little or no signs of "pointing," the application of a cantharides blister will hasten the process. Some practitioners prefer the ointment of biniodide of mercury for this purpose, and state that in some cases it will cause disappearance of the swelling without the occurrence of suppuration, It is a common practice to apply poultices to the swelling after the counter-irritant, in order to assist the action of the latter. We have found that the modern substitutes for poultices—*i.e.*, preparations composed of kaolin and glycerine, etc.—are cleaner, far more convenient, and more efficacious, retaining heat and moisture for a long period.

General Smith does not advise either poultices or surgical interference with the abscess. He employs a specially-made hood so as to retain a layer of fine tow in the intermaxillary space; by this means the part is kept warm, the abscess matures and is allowed to rupture spontaneously.

When pharyngitis and laryngitis are present, inhalations of steam medicated with terebene, oil of eucalyptus, compound tincture of benzoin, etc., should be prescribed, and repeated three times daily, taking care not to cause the animal distress.

A cantharides blister should be applied to the region of the throat and to the swollen glands, followed by a thick layer of kaolin and glycerine applied hot, and kept in position by a layer of cotton-wool and suitable bandages.

An electuary composed of extract of belladonna, camphor, potassium chlorate, glycerine, with honey as a basis, is to be placed on the back of the tongue three times daily. When the temperature is high, it is advisable to add quinine to the electuary, and the drug is readily taken by this method.

On no account should drenches be prescribed, as, in consequence of the difficulty in swallowing and the paroxysms of coughing which the process induces in this condition, portions of the fluid would certainly enter the trachea and bronchi, and cause a fatal broncho-pneumonia.

In cases where difficulty in swallowing is marked, foods in the fluid form should be ordered, such as milk, oatmeal gruel, linseed tea, hay tea, barley-water, etc.

Milk is an excellent form of diet in such cases, and horses generally take to it after a little persuasion.

The food should be allowed in moderate amounts at a time, and when not made use of should be removed; also the manger, buckets, etc., must be kept thoroughly clean. Sour food covered with the nasal discharge, and dirty feeding utensils tend to nauseate the animal. A bucket containing cold water, in which may be dissolved some potassium chlorate, should be left in the stall; the contents should be renewed at proper intervals. The animal's nostrils should be cleansed at intervals, and the stall and surroundings properly cleaned and disinfected. Good nursing is a *sine quâ non* in strangles, as it is in all other diseases.

When dyspnœa becomes excessive, the necessity for performing tracheotomy must be considered. This operation should not be indiscriminately performed, as there is the risk of the resulting wound assuming a septic character. Neither should it be delayed too long when indications for its employment are present, as the animal may die from asphyxia during the night-time.

When abscesses appear in other regions of the body, they should be opened when mature. Some of these are very deep-seated and close to important structures, so that a knowledge of surgical anatomy is necessary, and surgical dexterity is requisite in order to avoid injury to important bloodvessels, nerves, etc., in the affected regions. A trocar and cannula of fine calibre should be employed to locate the abscess, the skin should be carefully incised, and the fingers used as far as possible to expose the abscess. Many of these abscesses contain a very large amount of pus, and care should be taken to insure proper drainage.

In the irregular or malignant form of strangles the mortality is high, and ordinary methods of treatment are not successful. Of late years the employment of nuclein has given good results in the hands of some practitioners. (See p. 841 for hints as to its employment.) Potassium iodide is said to exert a beneficial action in some cases. The pulmonary complications of strangles are to be treated on similar lines to those laid down for pneumonia (see p. 840).

When abscesses occur in internal organs, treatment is seldom of any avail; the large majority of such cases prove fatal. Similar remarks apply to cases showing symptoms referable to the presence of lesions of strangles in the brain or spinal cord. Destruction of such cases proves the most humane and economical course once the symptoms have developed.

During convalescence from strangles great care should be taken to avoid putting the animals back to work before recovery is complete, otherwise complications are likely to develop, especially pneumonia, when the animal is exposed to chills and fatigue.

We are not aware of any means by which we can exert influence of a preventive character on the occurrence of "whistling" and "roaring" as sequels to strangles. The internal administration of potassium iodide, and the application of strong counter-irritants to the region of the throat, have been tried, but without any benefit resulting.

STRANGLES IN OTHER ANIMALS.

We have no definite knowledge of the occurrence of strangles in animals other than the horse. According to Friedberger and Fröhner, isolated cases of a disease simulating strangles have been recorded.

These authors quote instances recorded by Peschke in which calves were affected " with multiple abscess formation in the head." Also cases recorded by Nagy of two cows in which " suppuration of the laryngeal lymphatic glands with purulent nasal discharge and general febrile disturbance were present."

In the Dog, cases simulating strangles are met with. A series of abscesses occur in the region of the head, orbit, pharynx, and neck. Friedberger and Fröhner regard it as an " original infective disease which is independent of distemper and pyæmia," and may be given the name of " canine strangles."*

It seems probable, however, that such cases in reality belong to the suppurative or phlegmonous form of distemper (see Canine Distemper, p. 686).

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IMMUNISATION AND VACCINE THERAPY AS APPLIED TO STRANGLES.

By MAJOR A. G. TODD, A.V.C.

(*a*) **Immunisation.**—Up to the present time, the serum of hyper-immunised horses, although not conferring absolute immunity, has proved itself to be of more practical value for protection against the disease than any other method.

A horse may be considered hyper-immunised when he has received 100 c.c. of virulent culture of the *Streptococcus equi* intravenously. The initial dose of the culture is 1 c.c. Succeeding doses are regulated by the febrile reaction of the preceding dose. They are gradually raised from 1 to 20 c.c. weekly, and the final dose of 20 c.c. is never exceeded.* The average thermal reaction resulting is from 1° to 4° F. It is noticeable twelve hours after the inoculation, and lasts from twenty-four to thirty-six hours. There is seldom any constitutional disturbance. Making allowance for occasional and unavoidable delays in the preparation of culture, variations in the health of the animals under hyper-immunisation, and untoward after-effects, a horse is usually ready for bleeding three to four months after the initial dose. After this, fortnightly inoculations of the maximum dose of culture are necessary to keep up the potency of the serum.

The inoculations are made into the jugular vein, but any large superficial vein will answer the purpose equally well. They require to be made with the greatest care because of the risk of phlebitis and local abscess formation. Fortified horses may be bled twice monthly, 6 litres being taken at each bleeding.

The dose of the serum is from 20 to 30 c.c.; it may be injected subcutaneously or intravenously. Remounts in the army receive four 10 c.c. doses, the first being given after purchase, and the succeeding doses at intervals of one week. The sequence of the inoculations is not broken. After the last inoculation there is an immunity of four to five weeks' duration.

Serum does not prevent the development of the disease when injected during the period of incubation, but it modifies the severity of the attack in such cases.

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* I am indebted to Dr. Poels of Rotterdam for this method, and for much advice and general instruction regarding hyper-immunisation.

In civilian practice inoculation results appear to be more successful than in the army, and they are better in regiments than in remount depôts. In the army the average number of animals contracting the disease under ordinary conditions is about 20 per cent., which is reduced to about 5 per cent. by serum inoculations.

There is a consensus of opinion in the army by those who have used strangles serum, that it markedly modifies the course of the disease, when it occurs after inoculation, in that abscess formation is less pronounced and recovery quicker than in animals not protected.

There is good reason to believe that the risk of unsoundness of the wind—which is one of the common sequels of strangles—is appreciably reduced by the use of serum. The average number becoming unsound is 20 to 25 per cent. Out of sixty-nine protected cavalry remounts in 1909-10, only one subsequently became unsound (this horse had neither strangles nor catarrh), and of 251 similarly protected in 1910-11 all remained sound. With heavier horses—artillery remounts—the results were not quite so brilliant, but the number of cases of wind unsoundness was greatly reduced.

Serum may be used in the treatment of the disease in daily doses of 20 to 30 c.c. Under its influence the affected animals become brighter, feed better, the character of the nasal and abscess discharge often alters for the better, and the throat swellings which in the ordinary way would develop into abscesses sometimes subside altogether without further treatment.

(*b*) **Vaccine Therapy.**—Although this is still in the experimental stage, the encouraging results obtained from some of the cases treated point to a bright future for this method of treatment. A strangles vaccine may be prepared in various ways. The following method was adopted for the preparation of *strangline*, which gave such hopeful results. The streptococcus is isolated from the pus of a strangles abscess, and fortified by passing it through white mice. It is then grown on inspissated blood serum, at 37° C. for twenty-four hours, transferred to 10 per cent. serum bouillon, and incubated for one month. Six per cent. sterilised glycerine is now added, after which it is exposed in an incubator at 60° C. for two days over unslaked lime. This kills the organism, and evaporates the culture down to a thick paste. It is now dissolved in ½ per cent. carbolic and sterile water, and brought back to its original bulk to make it thin enough to pass through an ordinary hypodermic needle. The dose is from 1 to 10 c.c. hypodermically, which may be repeated in seven to ten days if necessary. It has no appreciable effect on the temperature, but a local reaction sometimes results, which disappears in two to three days. The most marked effects observed were in the healing of chronic abscesses in the malignant forms of the disease. The nasal discharge also altered in character, and visibly decreased soon after the injection.

CONTAGIOUS PNEUMONIA OF THE HORSE

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Contagious pleuro-pneumonia of the horse; Bilious pneumonia; Stable or hospital pneumonia; Pulmonary influenza; Epizoötic or enzoötic pneumonia.

French: Pneumonie infectieuse, ou contagieuse, ou d'écurie; Pleuro-pneumonie infectieuse.

German: Croupösen Pneumonie; Brustseuche; Infektiose Pneumonie; Brust-influenza; Influenza Pectoralis; Lungenentzundung.

Italian: Polmonite tifica.

Definition.—An infectious disease often occurring in an epizoötic form in horses, asses, and mules, characterised by the presence of high fever and the early appearance of pneumonia and pleurisy of an adynamic type, with a marked tendency to pulmonary gangrene and to complicating lesions of other organs, such as the heart, liver, kidneys, and intestines.

GENERAL REMARKS.

In most textbooks contagious pneumonia or epizoötic pleuro-pneumonia of the horse is placed amongst the infectious diseases, while ordinary or sporadic pneumonia occurs under the heading of Respiratory Affections. This would seem at first sight to indicate that the two affections were distinct from each other, both pathologically and clinically. We must point out, however, that all pneumonias depend on microbic infection, and that the causes formerly accepted—viz., exposure to cold, chills, fatigue, etc.—must be regarded as predisposing to the disease, and not as direct etiological factors. Definite knowledge on the bacteriology of equine pneumonias does not as yet exist; hence the two forms mentioned above cannot be differentiated on bacteriological grounds. Nor can the question be decided on the *post-mortem* appearances, because differences of opinion still exist as to the characteristic lesions met with in this so-called contagious form by which it can be differentiated from the sporadic form. On clinical grounds more reason exists for describing the two forms separately ; but, as we shall show later on, there are many points of resemblance even on clinical examination.

For convenience of description, and recognising the fact that we meet with cases of pneumonia corresponding to the sporadic type mentioned by certain authors, we have deemed it advisable to follow the usual plan and discuss here the epizoötic type of the disease.

The sporadic type will be described under the heading of Respiratory Diseases (see Pneumonia, Vol. II.).

Cadéac and other authorities believe that sporadic pneumonia and the so-called contagious type originate from a common cause. According to this view the clinical differences observed depend on an increased virulency of the micro-organisms, probably from the latter passing through a number of horses. Cadéac* has observed that a case showing the characters usually ascribed to the sporadic type may transmit the disease to a neighbouring horse in the stable, and produce in him an attack with all the characteristics of the contagious form. He has also found typical instances of both forms in the same stable. This is in accordance with our own experience.

The question will be found more fully discussed under the heading of Pneumonia (Vol. II.).

The affection we are considering is to be regarded as an infectious disease in which pneumonia is one of the characteristic lesions or manifestations. Amongst its characteristics we may note the frequency of extra-pulmonary localisations and the varieties of pulmonary lesions met with, also its marked infectious nature and the tendency to implication of the pleura. The disease usually occurs as an epizoötic, and outbreaks are very common in cities, especially in large stables. It tends to spread in an irregular manner, and for this reason a considerable time elapses before the disease can be eradicated from a large stable, as cases keep cropping up at irregular intervals. Even in the same stable there is a marked diversity between the intensity of the symptoms in the various cases, this depending on the extent of the pulmonary lesions, the nature of the complications, the virulence of the micro-organisms and their association with other germs, and the degree of intoxication produced by the microbes and their toxins.

The connection between contagious pneumonia and the pulmonary complications of influenza will be considered under the heading of Differential Diagnosis. It is in reality more a question of the history of the case and the early appearance of pulmonary symptoms than of the pathology of the affections, as the lesions may be similar in both.

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* Cadéac, "Pathologie Interne."

The probable connection between epizoötic pneumonia and influenza is considered under the heading of the latter disease (see p. 844).

SUSCEPTIBILITY.

It is generally observed that young horses are more susceptible to the disease, especially those brought from the country to city stables. Accessory causes have, however, a marked influence in rendering animals susceptible to the infection. Old worn-out horses also prove susceptible subjects.

IMMUNITY.

Authorities differ with reference to the degree of immunity conferred by an attack of the disease. Some believe that immunity is conferred for an indefinite period; others state that one attack has no protective influence.

ETIOLOGY.

Infection can probably occur by inhalation and by ingestion. The most common source of infection is the presence of an animal suffering from the disease, especially in a large stable of horses. Animals at some distance in the stable from those affected may contract the disease as well as those in direct contact; hence aerial infection is probable.

Some authorities regard ingestion as playing a very important part in connection with the transmission of the disease, the infection being introduced by means of contaminated litter, forage, watering-troughs, and feeding utensils.

Stables in which cases of the disease have recently been housed are also well known to be capable of infecting healthy horses placed therein. Mr. William Hunting, F.R.C.V.S., has observed that certain stalls in which cases of contagious pneumonia had been treated became so infected that every case placed therein, even those suffering from ordinary pulmonary congestion, developed pneumonia of a gangrenous type.

Horses that have recently recovered from the disease prove a source of infection when introduced amongst healthy animals. More especially is this the case when the apparently convalescent animal suffers from a chronic pulmonary lesion, which is by no means uncommon as a sequel to this disease. Such an animal is capable of maintaining the infection in a stable for a long period, and may induce an attack of the disease in every fresh arrival.

BACTERIOLOGY.

Up to the present time the specific micro-organism on which all varieties of pneumonia depend has not been discovered.

As already mentioned, many authorities, including Cadéac, do not recognise any distinction between the bacteriology of the contagious form and the so-called sporadic type of the disease; they believe that the differences in the clinical manifestations depend on the degree of virulence of the micro-organisms present. In the present state of knowledge we can only direct attention to the principal micro-organisms which certain authorities have isolated and described as the probable cause of pneumonia.

The streptococcus of Schütz, also known as the "bacterium of Schütz," was isolated and described in 1887. This micro-organism may be in the form of diplococci or in chains, and is found in the hepatised pulmonary tissue, the fibrinous exudate, the nasal discharge, the pleuritic exudate, the blood, and in various organs. According to Schütz and Cadéac, injections of the streptococci into the trachea, the blood, and the subcutaneous connective tissue, fail to produce pneumonia. Successful inoculations only occur when cultivations are directly introduced into the lung tissue by means of a Pravaz syringe. Then the symptoms and lesions of contagious pneumonia are produced.

Lignières' cocco bacillus, also known as the *Bacillus equisepticus.* This belongs to the group of Pasteurella, and is stated by some authorities to be the specific microbe of influenza (see p. 845). It is believed that this micro-organism can be associated with the streptococcus of Schütz, and prepares the way for the entrance and development of the latter.

Some authorities believe that the micro-organism on which fibrinous pneumonia in man depends may be occasionally found in equine pneumonia. This is known as the *Micrococcus lanceolatus*, or the *Pneumococcus*, or *Diplococcus pneumoniæ* of Fraenkel and Weichselbaum.

In the more virulent form of the disease various micro-organisms, in addition to those mentioned, have been found.

For further information on this subject the reader is referred to works on bacteriology.

INDIRECT MODES OF INFECTION.

According to Cadéac, the nasal discharge contains the micro-organisms in an active state during the entire course of the disease, and, as in some cases this discharge may continue during convalescence, ample opportunity is given for the spread of the affection. The same author states that desiccation does not interfere with the virulence of the micro-organisms.

Fodder, litter, mangers, feeding utensils, clothing, halters, etc., are likely to become contaminated, and to act as carriers of the infection. The micro-organisms have also been found in the fæces, urine, cutaneous secretions, and epithelial desquamations. Hence one can understand the important part that infected buildings play in the spread of the disease.

ACCESSORY OR PREDISPOSING CAUSES.

Any circumstances which tend to lower the natural powers of resistance act as predisposing causes. Exposure to cold and chills was at one time regarded as an actual cause of pneumonia, but is now known to lessen the normal resisting powers of the animal, and thus to render him susceptible to infection. Catarrh of the upper respiratory passages has a similar effect, but in addition it facilitates the entrance and development of the micro-organisms in the respiratory mucous membrane, and their extension to the bronchi and pulmonary alveoli. Fatigue, lack of proper food, ill-ventilated, badly drained, and overcrowded stables, are also accessory causes.

The disease is more prevalent during autumn, early winter, and early spring, especially if the weather changes suddenly. In our experience sudden changes from frosty weather to damp and wet bring about an increase in the number of cases.

MORBID ANATOMY.

The lesions found in this disease are subject to great variations. The pulmonary lesions present such a variety of forms that authorities differ as to which of these should be regarded as typical of the disease. In addition to the lungs and pleuræ, various organs and structures may be involved, and in some instances the lesions in the latter may be of equal gravity and importance to those in the former. And while pulmonary lesions may be regarded as constant in the disease, with rare exceptions, the alterations in other organs are variable, and predominate in some outbreaks far more than in others.

Lungs.

Many authorities recognise two forms of pneumonia as occurring in this disease—viz., (1) *Lobar ;* (2) *Lobular* or *Disseminated.*

To these Cadéac adds a third form, which he terms *Central.* In this the lesions are found in the vicinity of the large bronchial trunks, and are thus central in position, but may extend to the superior third of the lung.

Here we may remark that, although theoretically these various forms are recognised and described, it by no means follows that we are able to differentiate them at autopsies, nor is it an easy matter to decide by an examination of the lesions whether a given case should be classified as being of the epizoötic or of the sporadic type. Usually in both instances the lesions will be found far advanced, and the fatal termination may depend on similar conditions in each.

Lobar Form.—Reference to Pneumonia, Vol. II., will show that unanimity of opinion does not exist with regard to the lesions of sporadic lobar pneumonia in the horse, and similar remarks will apply to the lobar type of epizoötic pneumonia. The latter is said to be met with in the milder attacks of the disease.

According to Cadéac, the distinguishing features in the lesions are that in the epizoötic form the area of congestion surrounding the hepatised portion of the lung is more marked, pulmonary œdema is strongly in evidence, and an abundant exudate infiltrates the alveolar walls and interlobular connective tissue. The exudate tends to assume a hæmorrhagic character, and the area of hepatisation is less firm than in the sporadic form. On section there is a free outpouring of a greyish-red liquid, and the section surface shows small hæmorrhagic spots. According to the same author, the central form starts from an infectious bronchitis, and the hepatised area appears limited by a hyperæmic and œdematous zone. The Lobar form is generally unilateral.

Lobular Form.—This is to be regarded as being more typical of the epizoötic form of the disease. It is characterised by the presence of multiple areas or centres of hepatisation of various sizes, which tend to become confluent; these lesions are at first found especially at the lower borders and at the root of the lungs. The inflammation is of a hæmorrhagic character, and there is a marked tendency to the production of pulmonary gangrene.

According to Cadéac, the exudate is fibrinous in character, and this feature distinguishes it from ordinary broncho-pneumonia. Both lungs are generally involved in a symmetrical manner. The infection is believed to gain access by the bronchi, so that the anterior portions of the organs are first affected.

Pulmonary Gangrene.—The occurrence of this condition is ascribed to various causes, such as the presence of thrombi in a large number of the capillary vessels, a tendency to which is present in this disease. Also to the entrance of septic organisms into the lungs, when the latter are attacked by a hæmorrhagic inflammation. A large number of cases are also due to the careless administration of medicines in the form of drenches (see Inhalation Pneumonia, Vol. II.). Some authorities ascribe gangrene to the presence of "tissue-destroying toxins."

The condition may occur in a *circumscribed* or in a *diffuse* form; the latter is more common in the lobular type of epizoötic pneumonia.

The *circumscribed form* does not tend to spread, and the lesion is generally found in the centre of the lung. It consists of a slough of irregular shape and size, and of a dark brown or greenish colour, with a fœtid odour; it tends to liquefy, and a communication is established with a bronchus in the vicinity. By inflammatory reaction the gangrene is limited and a cavity is formed. Pus of a sanious character may enclose the slough. Some of the cavities may be of considerable extent. The cavities generally communicate with the bronchi in the vicinity, but rarely with the pleura. The necrosis may be of a dry character, and the cavities, lined by a smooth capsule, contain sequestra, which may remain thus encapsuled for an indefinite period.

The *diffuse form* is characterised by a number of hæmorrhagic centres of various sizes distributed throughout the hepatised areas; they show various tints of colour, and some undergo a process of dry gangrene, cavities of different sizes enclosing sequestra being formed. In others the necrotic portions undergo disintegration, and are transformed into cavities containing a putrid, fœtid, purulent material, of a greenish or blackish tint; the lung tissue is of an olive or greenish-brown colour. The walls of these cavities are imperfectly formed, and communications with bronchi in the vicinity occur, so that the bronchi and trachea become involved in the process, and present a greenish tint. Pulmonary fistulæ may also result.

By many authors gangrene is regarded as a complication of epizoötic pneumonia, and no doubt is a condition frequently met with; we find, however, that in some outbreaks a series of fatal cases may occur in which this lesion is absent, death resulting from other causes.

Diffuse Suppuration and Pulmonary Abscess.—These may be regarded also as complications or secondary conditions, and they very frequently accompany gangrene.

They depend on a mixed infection, such as the entrance of staphylococci, pyogenic streptococci, and the micro-organisms of gangrenous septicæmia. The pulmonary tissue undergoes disintegration, and abscess formation occurs. The suppurative process is believed to commence in the alveolar exudate; abscesses of various sizes are formed, and these tend to coalesce, so that an abscess of very large size may result. The pus may be of a laudable character, or it may be septic and fœtid and contain shreds of tissue. The abscess may extend to the pleura, and induce a purulent pleurisy. It generally opens into a bronchus, but in rare cases may become encysted.

Pleuræ.—Diffuse pleurisy is nearly always present. It generally develops at the same time as the pneumonia. In some instances it occurs as a secondary affection, while in others it may precede the pneumonia. The effusion may be considerable in amount, and in some instances it is found purulent, septic, and putrid in character.

Pharynx and Larynx.—Pharyngitis and laryngitis are present in a large proportion of cases, and may be very acute in character. These lesions probably depend on the entrance, development, and primary action of the micro-organisms in these regions.

Heart and Pericardium.—*Myocarditis* may occur alone or in association with pericarditis. According to Cadéac, myocarditis may be the chief or only lesion present in some cases, the pulmonary alterations being little marked; in such instances he believes that the micro-organisms and their toxins attack the myocardium and its nerves, instead of the lungs. The muscular tissue of the heart is of a pallid or brownish-grey colour, and undergoes softening; in some cases fatty degeneration is present.

Endocarditis may be observed in some cases; it depends on infection of the endocardium by the micro-organisms, which reach it by way of the blood-stream.

Pericarditis.—This is also microbic in origin, and is fibrinous in character.

All the lesions in connection with the heart and pericardium are believed to depend on an exalted virulence of the infecting micro-organisms.

Liver.—This organ may show congestion and enlargement. Catarrh of the bile-ducts may be present, due to extension of inflammation from the duodenum, and the organ may show an icteric tinge. Evidences of general icterus may be present, due to the cause mentioned, but more probably depending on alterations in the blood and excessive formation of bile-pigment. Fatty degeneration has been observed at certain points, and in some cases areas of commencing necrosis are present.

Spleen.—Hyperæmia and petechiæ may be present.

Kidneys.—These organs may show congestion or nephritis due to microbic influences; in some cases numerous petechiæ are present.

Stomach and Intestines.—Gastro-enteritis in varying degrees of intensity may be observed in some cases. Occasionally we find hæmorrhagic infiltrations, and rarely ulcers.

Lymph Glands.—Congestion, enlargement, and softening of the retropharyngeal, bronchial, mediastinal, and occasionally of the mesenteric lymph glands, may be observed.

Nervous System.—Lesions in the brain and spinal cord are by no means commonly met with, but in our experience they occur more frequently in some outbreaks than in others. Cerebral congestion, cerebral meningitis, spinal meningitis, cerebro-spinal meningitis, and cerebral hæmorrhage, may be observed.

Ocular Lesions.—Catarrhal conjunctivitis, keratitis, iritis, and occasionally peri-ocular abscess, are some of the lesions that may be present.

Muscles, Joints, and Synovial Sheaths.—The muscles may undergo softening, and their colour may be altered to a yellowish-brown tint. Lesions of joints and synovial sheaths—viz, arthritis and synovitis—are met with in the more virulent types of the disease, and they depend on microbic infection, as evidenced by the detection of streptococci in the synovial exudate. The frequency of these lesions varies in different outbreaks, and they seldom appear until convalescence has set in. The great sesamoidean sheath of a fore-limb is most frequently affected; other synovial sheaths may be simultaneously or successively attacked. Chronic lesions result in some cases.

Skin Lesions.—Occasionally, during convalescence, small abscesses containing pus of a benign character may appear, especially at the base of the neck.

Occasional Lesions.—Amongst other lesions which may be met with in cases of epizoötic pneumonia are those of purpura hæmorrhagica and of general septicæmia. Cases are recorded by Adrian in which thrombosis of the saphena vein and of the lymphatics in the vicinity occurred during convalescence, as a sequel to the disease. The malady may also be complicated by strangles, in which instance evidences of the latter affection will be found.

PERIOD OF INCUBATION.

This cannot be definitely determined, as it is difficult to ascertain the exact time at which infection takes place; besides, the primary symptoms may escape attention. Some authorities give the period of incubation as from five to six days, others as from three to ten days. During an outbreak in a stable fresh cases occur at very irregular intervals, and in some instances from two to three weeks may elapse before another case appears. It is probable that in animals subjected to chills, etc., the incubation period is lessened.

SYMPTOMS.

As might be expected in a disease characterised by such a variety of pulmonary lesions and a tendency to numerous complications, the symptoms show many variations.

Lobar Type.—The symptoms resemble those occurring in ordinary sporadic lobar pneumonia (see Pneumonia, Vol. II.). This type of the disease may be met with during an outbreak, and the subjects thereof may infect other horses, and produce in them the lobular or severe form of epizoötic pneumonia. *Per contra,* horses suffering from the latter form may infect others in the stable, and produce the lobar type. Hence, from a clinical point of view, there is little distinction between the lobar form of epizoötic pneumonia and the sporadic form, except in the more infectious nature of the former.

Larval or Abortive Forms.—During an outbreak cases are met with in which general symptoms, such as fever, dulness, loss of appetite, slight acceleration of the respirations and of the pulse, are present, but no evidences of pulmonary localisations can be detected. Others may present, in addition, slight crepitant râles, and mild coryza or bronchial catarrh.

Such cases, provided they are kept from work and not exposed to chills, tend to recover rapidly without presenting pulmonary symptoms, and are regarded as abortive attacks of the disease.

Lobular Type.—Although premonitory symptoms always occur, they are not observed in all cases, as the practitioner is seldom called to attend during the early stages.

General Symptoms.—Very frequently, on our first attendance, we find the physical signs of pneumonia well in evidence. The early development of pulmonary lesions is one of the characteristics of this disease. Rigors may occur in the early stages, but are by no means a constant symptom.

Fever.—The rise in temperature generally occurs in a rapid manner, and it may reach 104° to 106° F. During the later stages, when complications occur, the fever is subject to exacerbations.

The Pulse is increased in frequency, and in the later stages it tends to become weak, soft, and irregular. In some cases in the early stages, even with a high temperature, the acceleration of the pulse is not well marked.

The Respirations are accelerated, and their character and the amount of dyspnœa present vary according to the extent of the pulmonary lesions.

Visible Mucous Membranes.—An icteric tinge is often present, especially in the conjunctivæ. In some cases marked congestion is observed.

Constitutional Disturbance.—This varies in degree according to the severity of the attack. Depression, muscular weakness, hanging head, drooping ears, and loss of appetite, are observed. In some cases the loss of appetite is subject to modifications, and we meet with instances in which the animal continues to feed throughout the attack.

The *nasal discharge* varies in extent: it may be of a yellowish or orange tint, or it may assume the rusty colour which is observed in lobar pneumonia. In very acute cases it may be tinged with blood, or hæmorrhagic in character.

Pharyngitis and *laryngitis* occur in the early stages in many outbreaks.

Cough is a variable symptom, but is always present when pharyngitis and laryngitis accompany the disease.

The presence of pulmonary lesions is ascertained by a physical examination, of the chest. When they are extensive, accelerated respirations and dyspnœa occur; but there are cases in which pulmonary alterations are present without any marked degree of respiratory disturbance.

PHYSICAL SIGNS.

Percussion.—When the lesions are deep-seated, percussion may not reveal anything abnormal. As they approach the superficial parts of the lung, dulness is found on percussion, and a tympanitic sound may be detected around the affected areas. In the disseminated or lobular type of the disease, when the lesions approach each other, irregular areas of dulness are found at the antero-inferior aspect of the lungs, especially in the region just behind the shoulder. These are in contrast to the large area of dulness found in cases of lobar pneumonia (see Pneumonia, Vol. II.). Between the hepatised areas a tympanitic sound may sometimes be detected.

Auscultation.—In deep-seated lesions, or when the foci are small in size, auscultation may reveal little beyond a coarse bronchial respiration and an inspiratory crepitant râle. With more extensive lesions, weakness of the vesicular murmur, with crepitant râles, irregularly distributed, may be detected.

For further details with reference to auscultation see Pneumonia, Vol. II.

As *pleurisy* generally accompanies or soon follows the pneumonia, the physical signs of the former are added. Percussion in the early stages shows tenderness of the thoracic walls, and after effusion occurs, dulness is present up to a line corresponding to the upper limit of the fluid. Auscultation reveals in the first stages friction sounds, and, after the occurrence of effusion, absence of respiratory sounds up to the level of the fluid (see Pleurisy, Vol. II.).

Other symptoms of pleurisy, such as the abdominal type of respiration, the pleuritic ridge, and the painful suppressed cough, may be present, but in some cases the affection develops so rapidly that these symptoms may be overlooked; moreover, they may be absent. For the symptoms of pleural effusion, see Pleurisy, Vol. II. Generally horses suffering from pleuro-pneumonia and all acute respiratory affections maintain the standing posture. To this rule there are exceptions, as in some cases the animals lie down at intervals throughout the course of the affections.

Pulmonary Gangrene.—The symptoms of this condition may appear towards the end of the second week, or they may occur during the stage of resolution. Mr. W. Hunting has observed that cases of pneumonia which have not fully recovered, if put to work too soon, may show symptoms of pulmonary gangrene in twenty-four hours, and rapidly succumb. The symptoms usually met with are those which might be attributed to a relapse. The temperature rises, the animal shows marked depression, a "wrinkled" and haggard expression of countenance, total loss of appetite, the pulse small, weak, and gradually becoming imperceptible, tumultuous action of the heart, marked dyspnœa with irregular respirations, and a fœtid greyish or almost black discharge from the nostrils. The latter symptom is very characteristic, and may be the first indication observed of the presence of the condition, the breath of the animal having a most repulsive odour.

We must point out, however, that the occurrence of fœtid breath and putrid nasal discharge in cases of pneumonia are not always to be attributed to pulmonary gangrene, as they may depend on a septic condition of the pulmonary exudate.

The *Physical Signs* are very variable. Percussion may show a tympanitic sound or a metallic *bruit*, indicating disintegration of lung tissue. Auscultation may reveal the presence of crepitant râles, and various mucous and sibilant râles with sonorous râles. Crepitations of a bubbling character are very commonly met with in this condition. Death occurs in the majority of cases of diffuse gangrene, but in a variable period of time. Some cases last for three or four days; others may succumb in from twenty-four to thirty-six hours. In circumscribed gangrene recovery may take place, with a sequestrum encapsuled in the lung. Mr. W. Hunting has found that cases of pneumonia showing fœtid breath, if removed out of town to well-ventilated stables in the country, may recover, while if left in town stables they invariably die. Evidences of general septicæmia are present in most of the fatal cases.

Pulmonary Abscess.—The symptoms of this condition are by no means well marked, and are often obscure. The fever reappears, and may be of an intermittent character; the constitutional symptoms resemble those met with in gangrene. The nasal discharge is of a greyish tint, or it may be absent; cough is also present, and pus may be expelled by the nostrils during the act of coughing. Usually the abscess gains access to the bronchi, and pus is discharged by the nostrils. Auscultation shows the presence of sibilant and mucous râles or of cavernous râles, due to the air passing through the purulent contents of the cavity; gurgling and bubbling sounds may also be heard. Such cases are nearly always fatal.

When the abscess gains entrance to the pleural cavity, an amphoric* sound is heard, also gurgling sounds. In rare cases the abscess becomes encysted, and the acute constitutional symptoms abate; but the patient remains in a feeble condition, has a constant cough, and shows respiratory distress when moved. Death occurs in a variable period from gradual exhaustion of the vital powers.

COMPLICATIONS.

Myocarditis.—This is evidenced at first by a tumultuous action of the heart and an accelerated pulse, succeeded by a feeble thready pulse, which becomes gradually imperceptible. Pulmonary œdema may occur, and aggravate the existing dyspnœa.

Cadéac points out that the symptoms produced are not always in accordance with the cardiac lesions present. The toxins exert a paralysing effect on the cardiac branches of the vagus in some cases, and produce the symptoms at first, without the presence of lesions of the myocardium. Later on organic lesions may appear, and contribute to the fatal termination.

Pericarditis and Endocarditis.—The symptoms are similar to those met with in the primary forms of these conditions (see Cardiac Affections, Vol. II.). It must be remembered, however, that the symptoms may be considerably modified in consequence of being associated with those of pneumonia and pleurisy. In most instances the presence of endocardial lesions is only discovered at the autopsy.

Renal Complications.—Nephritis may occur during the course of the disease or during convalescence. It may be evidenced by the frequent passage of small quantities of urine, sometimes hæmaturia, uneasiness, anxious expression of countenance, dropsical effusions on the chest and abdomen, straining and frequent attempts at micturition, swelling of the hind-limbs, and weakness of the latter on progression.

Diagnosis is based on an examination of the urine (see Nephritis, Vol. II.).

Hepatic Complications.—Jaundice in varying degree points to the liver being implicated. An icteric tint of the visible mucous membranes is a frequent symptom in epizoötic pneumonia.

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* A sound resembling that produced by blowing with the mouth through the neck of a bottle.

Gastro-Intestinal Complications.—In these the symptoms presented depend on the extent of the lesions in the stomach and intestines. Severe attacks of colic are manifested in some cases; in others we find diarrhœa and the passage of blood-stained fæces. Cadéac describes cases in which horses in an infected stable showed severe colic, with an elevation of temperature; and after the colicky symptoms disappeared with treatment, the temperature returned to normal, but no pulmonary symptoms developed. He regards such cases as depending on a slight degree of infection of the alimentary canal, and, as a result, the lungs escape the infection.

Nervous Complications.—These may occur during the period of hepatisation, or when resolution is proceeding. Meningitis is evidenced by a staggering gait; the animal soon assumes the recumbent position, and struggles violently at intervals with the fore- and hind-limbs; nystagmus (rolling movements of the eyes) and delirium are present, and a tendency to push the head towards the back. The delirium is succeeded by a comatose condition, and a fatal termination always results.

Milder forms of nervous phenomena may be observed instead of the above, such as stupor, great prostration, unsteady gait, relaxation of the *sphincter ani*, dilatation of the rectum. Paralysis of both hind-limbs may occur in some cases. Occasionally special nerves may be affected; paralysis of the penis has been observed, and we have met with one case of this kind in a stallion during convalescence from epizoötic pneumonia. Cadéac has observed instances of amaurosis, deafness, paralysis of the facial nerve and of the sciatic nerve, amongst the nervous complications.

Nervous complications are more common in some outbreaks than in others, and in one instance we met with a series of cases which showed symptoms of meningitis early in the course of the disease.

Complications in Joints and Synovial Sheaths.—These generally appear during convalescence, and are evidenced by lameness and by painful swelling of the affected region. In some cases the lameness is very severe and the pain acute.

Amongst other complications may be mentioned the occurrence of purpura hæmorrhagica, which is recognised by the characteristic symptoms of that affection (see p. 875). It may manifest itself at any time during the course of epizoötic pneumonia, or during convalescence. Laminitis has also been observed during the course of the disease.

SEQUELÆ.

Roaring, pulmonary emphysema, chronic cardiac lesions, chronic nephritis, etc., may occur as sequels to epizoötic pneumonia.

COURSE.

In the lobar form of the disease and in mild attacks the course is fairly regular. The fever generally continues from five to eight days, and gradually abates, and evidences of pulmonary lesions disappear, the appetite returns to normal, and the animal recovers his former spirits. The period of convalescence usually extends from two to three weeks. In the lobular form the course tends to become irregular. Relapses are liable to occur and complications to develop, even when the case appears to be progressing favourably. Death may occur from various causes, the complications present exerting a marked influence in this direction.

A chronic type of the disease is often observed, in which the necrotic portions of the pulmonary tissue become encapsuled. Such animals may recover sufficiently to return to work, but are likely to become attacked with pneumonia at any time, and almost invariably succumb to pulmonary gangrene as the result.

PROGNOSIS.

In the milder forms prognosis is favourable, provided good hygienic surroundings and an intelligent attendant can be obtained. In the severe forms the mortality is high, even with the best of care and surroundings, as complications may develop at any time during the course of the affection. An important element towards a favourable termination is that the animals should be removed from work as early as possible. As many attendants on horses are not specially observant, they imagine that nothing is amiss so long as the animals feed. We know, however, that horses in the early stages may maintain their appetite, and if they are kept at work until this fails, the gravity of the cases is largely increased. Of almost equal importance is the management of cases during convalescence, so as to avoid working the animals before a proper period has elapsed. Neglect of this precaution is responsible for many fatal cases, as relapses or secondary attacks are common under such circumstances.

DIFFERENTIAL DIAGNOSIS.

Considerable difficulty may occur with reference to the diagnosis when the first case appears in a stable, more especially if the pneumonic lesions are small in extent. As already mentioned, it is impossible to state whether a first case should be classed as one of sporadic pneumonia or one of the epizoötic type. When more cases occur, this difficulty disappears. As pneumonia is a frequent complication of influenza, the question of a differential diagnosis must be considered. The early appearance of pulmonary symptoms in epizoötic pneumonia, and their later appearance in influenza, in which they are secondary, will prove of assistance in the diagnosis. In connection with this, the history of the outbreak must be taken into consideration (see Influenza, p. 845).

We must admit, however, that it is difficult to lay down rules for the differential diagnosis of these affections, as they have many points in common. It is not unusual to find in the same stable cases conforming to the type of epizoötic pneumonia, ordinary pneumonia, influenza, and even strangles. We must remember, too, that strangles may be complicated with pneumonia, while pneumonia and influenza may, during their course, show the presence of the characteristic abscesses of strangles.

PROPHYLAXIS.

From a practical point of view, the question of a differential diagnosis is not of so much importance as the necessity for attention to preventive measures in connection with all these affections. All cases showing coryza, pharyngitis, laryngitis, the presence of fever, constitutional disturbance, pneumonic or pleuritic symptoms, etc., should be promptly isolated. In the case of large stables it is prudent to isolate all new purchases for at least two weeks whenever this is possible, and an isolation stable may be the means of saving much loss. The measures for disinfection, etc., are similar to those mentioned under the heading of Strangles (see p. 816), and precautions for the prevention of infection are to be carried out on the same lines recommended for that disease.

Serum Inoculation, as a preventive measure, has not up to the present time given definite results, and contradictory opinions are expressed with reference to its efficacy.

TREATMENT.

The primary essentials are good hygienic surroundings and careful nursing. Medicinal treatment plays but a minor part in bringing about a favourable termination, and we are not aware of any means by which the course of the disease can be modified or shortened. The principles of treatment are similar to those mentioned under the heading of Sporadic Pneumonia (see Vol. II.), where the matter will be found fully discussed.

We need only repeat the injunction here that drenching should be avoided as much as possible, as we are fully convinced that many cases of pulmonary gangrene are due to the entrance of medicines in the fluid form, associated with secretions of the mouth and pharynx, into the trachea and bronchi. Even with the exercise of the greatest care this accident is likely to occur. If it is considered advisable to administer antipyretics in cases where the fever runs very high, quinine may be given in the form of electuary, the best basis for which is honey, as this is readily taken by the patient. Salines such as magnesium sulphate and potassium nitrate may be given in the drinking-water. A plentiful supply of cold water should always be left within reach of the animal. If stimulants are indicated, carbonate of ammonia may be given in the form of a bolus. Some practitioners believe that alcoholic stimulants prove of value in the treatment of this affection, but the risks involved by drenching must always be taken into consideration.

The employment of antiseptic medicated inhalations of steam we have always found of value, provided due care is exercised so as not to distress the animal. The most suitable agents for this purpose are pure terebene, oil of eucalyptus, cyllin, carbolic acid, etc.

Mr. D. Imrie, M.R.C.V.S., Glasgow, has found the intravenous injection of formalin very useful in the treatment of pneumonia, and he has kindly furnished us with the following particulars with reference to its employment:

In cases where the pneumonia is bilateral, or the patient debilitated, with a quick, weak, frequent pulse, and the case "hanging fire," about the seventh day of the illness 2 drachms of formalin diluted with 8 ounces of warm water are injected into the jugular vein. Sometimes the injection is given earlier in the case; or it may be administered at any time during the course of the disease. Great care is necessary when introducing formalin solution into the vein.

Mr. Imrie advises that a small trocar and cannula be first introduced; the trocar is then withdrawn, and a piece of small-bore pliable rubber tubing is attached to the cannula. The solution is then slowly injected through the rubber tubing by means of a glass syringe capable of holding 4 ounces. He believes that the employment of formalin is indicated when there is reason to fear that pulmonary gangrene may ensue.

Mr. William Robb, F.R.C.V.S., Glasgow, informs us that he has had marked success with *nuclein* solution in the treatment of this disease. He finds that by its employment the temperature is often reduced from 106° to 103° F. within twenty-four hours after the first injection. In other cases, although the temperature was not reduced so quickly, a marked improvement was shown in the general condition of the animal, the respirations becoming easier, the appetite improving, and the appearance being brighter. He administers 25 to 30 c.c. of the nuclein solution once daily by intravenous injection. The solution should be heated prior to injection, as this prevents the occurrence of alarming symptoms, such as collapse, which are likely to appear if the solution be injected when cold. When improvement is observed and the temperature is reduced to about 103° F., the injections may be discontinued.

The intravenous injection of normal saline solution is highly spoken of by some practitioners as a cardiac stimulant in cases of pneumonia. Inhalations of oxygen gas have been also found of value (see Pneumonia, Vol. II.).

The complications of the disease must be treated as they arise.

The value of counter-irritation applied to the thoracic walls is a question on which opinions differ. It will be found fully discussed under the treatment of Pneumonia in Vol. II.

Affections of the joints and synovial sheaths are best treated by the application of fomentations and anodyne liniments at first, followed by a cantharides blister. Potassium iodide should be given internally.

During convalescence an excess of nitrogenous food should be avoided; otherwise there is a risk of renal affections or of an attack of azoturia being induced. Exercise should be gradual, precautions should be taken against the occurrence of chills, and the animal must not be put to work too soon.

Cases showing evidences of pulmonary gangrene should be kept in a separate building, as we learn from experience that they are likely to induce a similar condition in other cases of pneumonia. Similar remarks apply to cases showing evidences of strangles in addition to those of pneumonia.

INFLUENZA

BY E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Catarrhal fever; Horse distemper; Pink-Eye; Epizoötic cellulitis; Adynamic catarrhal fever of horses; Bilious fever; Epizoötic gastro-enteritis.

French: Fièvre typhoïde; La grippe.
German: Pferdestaupe; Rothlaufseuche; Darmseuche.
Italian: Influenza; Febbre tifoide.

Definition.—An infectious febrile disease of the equine species, often occurring as an epizoötic. Its bacteriology is not as yet determined. The affection is characterised by stupor, alterations in the blood, and in some cases ocular phenomena, also by a marked tendency to secondary infections and complications, and by the variety of clinical types it may assume in different outbreaks.

HISTORY.

Influenza appears to have been recognised from the earliest times. Some authors believe that references to the disease occur in the writings of Titus Livius, Hippocrates, Virgil, Vegetius, and others. In 1299 an extensive epizoötic affection amongst horses in Seville was described by Laurentius Rusius. In 1648 Solleysell described an outbreak amongst the horses of the French army in Germany. In 1688 the disease was prevalent over the entire continent of Europe. Various epizoötics of the affection from this date are recorded in Great Britain, the continent of Europe, and America. For a full description of the earlier history of the disease the reader is referred to Fleming's "Veterinary Sanitary Science and Police."

In the nineteenth century epidemics of influenza occurred in 1803, 1805, 1813-1815, 1825-1827, 1836, 1840, 1846, 1851, 1853, 1862.

In 1871-72 an outbreak occurred in London, and in 1891-92 the disease was extensively distributed throughout Great Britain.

One of the most remarkable and widely spread outbreaks of influenza occurred in Canada and the United States in 1872-73. It commenced in Toronto on October 1, 1872, and rapidly spread throughout Canada. It appeared in Buffalo, N.Y., on October 14; and extended to Boston, New York, Brooklyn, Jersey City, Philadelphia, and Washington. In this epizoötic the type of the disease was that known as "pink-eye."

From 1881 to 1883 a widespread epizoötic of influenza occurred over almost the entire continent of Europe, especially in the large cities. In Northern Germany and Denmark it was especially prevalent during 1890, 1891, and 1892.

In the British Isles outbreaks of the disease occur from time to time, and in different years the type of the affection is observed to alter, and also its character, as regards mildness or severity.

GENERAL REMARKS.

The term "influenza" was in former years applied to any epizoötic affection characterised by the presence of fever and presenting evidences of morbid alterations in one or more of the internal organs. In consequence of the various symptoms manifested during different outbreaks, it was usual to recognise certain forms of the disease, such as catarrhal, pulmonary, gastro-intestinal, etc.

Falke in 1862 studied the subject, and proposed that two distinct affections should be recognised—viz., (1) influenza, (2) typhus. The latter, from the description given by this author, appears to be identical with the epidemic equine pleuro-pneumonia of Dieckerhoff.

In the present day most authors follow this classification, and describe influenza and contagious pneumonia of the horse under separate headings.

Robertson,* in dealing with influenza, described the simple or uncomplicated form, the pulmonary or thoracic, the abdominal or enteric, and the rheumatic.

A separate type was recognised by Williams† under the designation of Epizoötic Cellulitis, or "Pink-Eye."

Some authorities in the present day regard influenza and epizoötic pneumonia as being only different forms due to one etiological factor. Thus, Nocard and Leclainche, also Lignières, include them under the denomination of "equine pasteurellosis." On the other hand, many authorities, including Cadéac, regard influenza as a distinct disease, in which the so-called forms are in reality complications or secondary infections due to the entrance of other micro-organisms. Thus an animal affected with influenza is rendered susceptible to secondary infections.

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* "Equine Medicine." † "Principles and Practice of Veterinary Medicine."

A comparison of the clinical phenomena and morbid anatomy of cases of influenza complicated with pneumonia and of epizoötic pneumonia shows many points in common. It is well known that cases showing symptoms of uncomplicated influenza, as well as those presenting evidences of epizoötic pneumonia, may be met with in the same stable. Again, cases coming under the designation of "pink-eye," and also of strangles, may occur in addition to the above. In many instances the practitioner may, even with the possession of considerable diagnostic skill and experience, be unable to state definitely as to which of the above diseases the outbreak should properly be attributed.

No doubt the early appearance of pulmonary symptoms would point to an epizoötic pneumonia, while the characteristic swelling of the eyes and limbs would be strongly in favour of influenza. But we must remember that in many outbreaks, cases of uncomplicated influenza may be present without showing any ocular symptoms or swollen limbs; that cases of strangles may not show the typical abscesses; while in some instances cases of epizoötic pneumonia may not present any evidences of pulmonary lesions at the first examination. In fact, so many difficulties arise in connection with the differential diagnosis of these affections that many practitioners, for clinical purposes, class them all under the heading of influenza, and diagnose the complications as they arise. According to Nocard and Leclainche, "the commonest and best known of the superadded infections to the pasteurellosis (influenza) is infectious pleuro-pneumonia."

BACTERIOLOGY.

The specific micro-organism on which influenza depends has not yet been discovered. Lignières discovered a cocco-bacillus in the blood, exudates, nasal discharge, pharynx and larynx of horses suffering from the disease, which is believed by some authorities to be the specific cause of influenza. This micro-organism belongs to the group of Pasteurella. Many authorities do not recognise this micro-organism as the cause of influenza, and bring forward several objections to the acceptance of its specific character. Cadéac points out that the cocco-bacillus cannot be discovered in many instances in the bodies of horses that have died from influenza. Also, that it is impossible to produce this disease by inoculation with the cocco-bacillus in a series of horses. "The only pathogenic cocco-bacilli for the horse are those which are rendered hypervirulent by their inoculation into the peritoneum of the guinea-pig. "These produce in the horse an intoxication, generally followed by a secondary infection due to streptococci." Horses experimentally infected are incapable of transmitting the disease to other animals in contact with them.

Cadéac regards all the micro-organisms which may be present in the body after death from influenza as being due to a secondary infection. Whatever may be the infecting agent which produces influenza, it renders the system liable to invasion by other micro-organisms, such as streptococci, similar to those occurring in strangles and epizoötic pneumonia, the cocco-bacillus, the bacillus of necrosis, the *Bacillus coli*, the bacillus of putrefaction, etc.

Poels has shown that the causal agent is an ultra-visible micro-organism (see p. 848).

Basset* has also shown that influenza is due to an ultra-visible micro-organism and is an inoculable disease. This view was held many years ago by Dieckerhoff.

Ferry† has isolated a streptococcus from the trachea in the early stages of nearly every case of acute equine influenza studied. He has also isolated an organism with the same characteristics from the blood of thirty-four out of sixty-three cases. He was unable to find any points of difference between this organism and the streptococcus of strangles, but did not feel justified in stating positively that this streptococcus is the cause of influenza in horses. He concluded that strangles and contagious pneumonia are not clinical entities, but complications of influenza, due either to secondary infections or to extension of the primary disease.

We may remark that this view has been held for several years by many clinicians in Great Britain, so there is nothing new in the suggestions made.

In the present state of knowledge it is neither necessary nor desirable to quote other views on this subject. Probably further researches will result in some definite knowledge being obtained. Although there are some points of resemblance between influenza of the horse and influenza of man, we have no evidence of the disease being communicable from one species to the other. The bacillus of Pfeiffer, which is regarded as the micro-organism on which human influenza depends, has not been found in the horse.

ETIOLOGY.

Clinical evidence points to an infected animal as being the most common source of the disease in every outbreak. The source of infection in the *first* case that appears in a stable cannot always be traced; this difficulty is also encountered in connection with strangles. Some authorities attempt to explain this on the grounds that a saprophytic micro-organism may be the cause of the disease, which from unknown influences may become virulent and pathogenic. One of the characteristics of influenza is the variety of types it may assume in different years. Some outbreaks show marked virulence, with serious and often fatal complications. On the other hand, a mild type may be met with, or, after a series of outbreaks of a mild character, the disease may appear in an acute and highly infectious form, the so-called "pink-eye." Even in one outbreak all grades of acuteness and varieties of types may be met with. We are not aware of the circumstances which are capable of modifying or increasing the virulence of the infection in different outbreaks. One fact we do know—viz., that in some years the disease spreads with remarkable rapidity from horse to horse, especially where a large number of animals are kept together. Probably infection takes place both by inhalation and by ingestion. Horses recently recovered from the disease are a probable source of infection. Mules and asses may be affected in some outbreaks.

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* *Revue Vét.*, April, 1911.

† "The Etiology of Equine Influenza," a paper read at the meeting of the Society of American Bacteriologists, Washington, D.C. (*Vet. Journal*, April, 1912).

Indirect Methods of Infection.—Forage contaminated by nasal discharge, litter, manure, nose-bags, clothing, halters, grooming utensils, buckets, mangers, etc., may act as intermediary bearers of infection. Infected stalls are well known to prove a source of infection, also horse-boxes which animals suffering from the disease have recently occupied. Attendants on cases of influenza may convey the disease to healthy horses.

Infection by Coition.—Many observers have recorded instances where the disease was transmitted from a stallion that had apparently, recovered from an attack, to a mare by coition. Cases are reported in which a stallion under the above conditions has proved a source of infection to mares even for months after convalescence.

With reference to this mode of infection, and also to the causal factor in the disease, we are indebted to Major Todd, A.V.C., for the following interesting note:

"In the spring of 1907 the Netherlands Government bought a stallion in France for stud purposes. He was examined and passed sound, and arrived at his destination in the best of health. When put to the stud, every mare mated with him developed influenza three or four days after coition. As he was a good-looking horse, with an excellent pedigree, it was decided to retain him till the following season. The result in this and the succeeding year was the same.

"Poels collected some of this horse's semen and inoculated several susceptible horses with it. A few days afterwards they exhibited the same symptoms as the mares. He then passed the semen through a Chamberland filter, and found the filtrate to be equally virulent. The blood of the infected stallion was now tested by inoculation, and gave negative results, proving that the virus was confined to some part of the generative organs. This was also supported by the fact that susceptible animals placed in the same stable as the infected stallion did not develop the disease. Poels made further observations on the blood of recently infected animals, and found that the virus was present during, and for a short time after, an attack of the disease, and that susceptible horses inoculated with small doses of virulent blood passed through a mild form of the disease. They were afterwards immune to large doses and to natural infection. Although the virus soon disappears from the blood-stream, it is apparent from the above that it may remain in a state of virulency in certain glands in the body with no ill-effects on the general health. Such animals may become "carriers" of the disease for an indefinite period, but it is only when the secretion of the infected glands comes in contact with susceptible horses that the latter become infected. Poels afterwards inoculated large numbers of horses against the disease without fatal result. A reaction is noticeable two to three days after the inoculation. It is accompanied by mild fever, inappetence, and swelling of the eyes and limbs in varying degrees of severity."

ACCESSORY CAUSES.

Chills and Fatigue.—These act by lowering the natural resistance to microbic infection. A mild attack may be converted into a severe one, with serious complications, when the affected animal is exposed to chills; or if returned to work too soon during convalescence, similar results may occur.

Impure Air and Unsanitary Surroundings.—These impair the vigour of animals, and thus render them more liable to become receptive to infection.

Age.—Young horses appear to be more susceptible to infection than older animals. It is well recognised that young horses purchased in the country and added to city stables are very frequently attacked. The probable explanation is that they are exposed to infection for the first time, and also that they do not possess the acquired immunity that a previous attack of the disease may sometimes confer.

Season of the Year.—As a rule, more cases are met with during early spring and in autumn, or in the early months of winter. In damp countries like Ireland the disease may be prevalent at any season of the year.

Other Accessory Causes.—The disease is often prevalent in dealers' stables, as young horses are constantly being added; these are purchased at fairs in various parts of the country, and exposed to chills and infection during transit by rail or boat. In some large stables cases of the disease are present at all times in varying degrees of severity, in spite of all efforts at preventive measures.

IMMUNITY.

One attack may confer immunity for an indefinite period, but marked variations are met with. We have seen two attacks in the same animal within six months of each other.

MORBID ANATOMY.

The lesions will depend on the severity of the case and on the nature and extent of the complications. The primary action of the disease is on the blood; next the digestive, circulatory, and nervous systems are affected, and in some types ocular phenomena are observed. The complications may involve many organs, but the respiratory and nervous systems are especially liable to be attacked.

According to Cadéac, the complications may be considered under two headings:

1. *Those occurring during the Course of the Disease, and depending on Alterations in the Blood, the Result being to induce Passive Congestion in Various Organs and Tissues.*

The alterations in the blood are due to toxins and to the high temperature. The effusions and hæmorrhages result from stagnation of the blood and infiltration of the tissues. Nutrition is thus interfered with, and parenchymatous degenerations of various organs are produced; also œdematous swellings of the limbs and dependent parts of the body, pulmonary congestion and œdema, and passive congestions of the nervous system, heart, liver, etc.*

2. *Complications depending on Secondary Infections.*

Various micro-organisms may enter the system of an animal suffering from influenza. The natural defences of the body are weakened by the action of the influenza infection, and by the morbid changes produced.

"The alteration of the blood, the feebleness of the patient, the elevation of temperature (which weakens the bactericidal power of the tissues and the phagocytic means of defence), the loss of appetite and the insufficiency of nutrition, the intestinal fermentations, and the accumulation of toxic products in the blood, which is deficient in oxygen and rich in carbonic acid—these are circumstances whch favour the entrance of all extraneous germs into the system and their multiplication therein" (Cadéac).*

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* Cadéac, "Pathologie Interne."

Thus is explained the occurrence of strangles, pneumonia, endocarditis, purpura hæmorrhagica, etc., in association with influenza. It is believed that the micro-organisms which normally exist in the respiratory passages may, under the influence of the influenza microbes, become virulent, and produce the various catarrhal affections which are so commonly associated with the disease.

We shall now consider in detail the various lesions met with.

Respiratory System.—Evidences of coryza, laryngitis, and œdema of the glottis, may be present. When complicated with strangles, the lesions of this affection will be observed. Occasionally pseudo-membranous patches may be found on the nasal septum (Cadéac). The trachea and bronchi may show hæmorrhagic effusion and petechiæ, and in rare cases the mucous membrane may present ulcerations.

Lungs and Pleuræ.—Pulmonary congestion followed by œdema may occur in some cases; both lungs are affected, and the lesions are similar to those met with in acute congestion of the lungs (see Vol. II.).

Various types of pneumonia may occur as complications, but the lesions most frequently met with are similar to those described under Epizoötic Pneumonia (see p. 830).

Pulmonary gangrene may be present. The pleural lesions are similar to those met with in the epizoötic form of pneumonia.

Digestive System—*Mouth.*—Stomatitis, swelling of the follicles and papillæ of the buccal mucous membrane, swelling and redness of the gums, especially in the vicinity of the incisor teeth, may be observed.

Pharynx.—Evidences of pharyngitis and swelling of the pharyngeal glands may be present.

Stomach.—In the pyloric region the mucous membrane may show congestion, ecchymoses, and desquamation of the epithelium. Deep ulcers may occur near the pylorus. The mucous membrane may be altered in colour to a reddish-brown or a greyish tint. Evidences of gastritis may be present.

Small Intestine.—Well-marked elevations may be observed on the mucous membrane, due to gelatinous infiltration of the submucous coat.

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* Cadéac, "Pathologie Interne."

Peyer's patches show congestion and enlargement, and sometimes evidences of inflammation.

Large Intestine.—The mucous membrane is thickened and infiltrated, shows ecchymoses, and is lined by a greyish-coloured fœtid mucus. Enteritis of a patchy or diffuse character occurs in some cases, and occasionally hæmorrhagic extravasation between the mucous and serous layers of the bowel; ulcers of the mucosa may also be present. The mucous membrane of the rectum may be congested, infiltrated, and prominent; and eversion of the rectal mucosa may occur as the result of straining when constipation is present.

Mesenteric Glands.—These show congestion, enlargement, and occasionally undergo softening.

Peritoneum.—Areas of infiltration, petechiæ, and a variable amount of peritoneal effusion, may be present.

Liver.—Congestion of the organ is present; this depends in part on feeble cardiac action due to degenerative changes in the myocardium. The liver is increased in size and weight, and in some cases shows superficial hæmorrhages. The gland structure may be friable and of a yellowish tint; in some cases fatty degeneration is present.

Spleen.—Occasionally engorgement may be observed, and hæmorrhagic foci may be present. In many instances no lesions are discovered in this organ.

Urinary System—*Kidneys.*—The chief lesions are: congestion; the presence of small elevations on the surface of the organs, due to serous effusion or extravasation beneath the capsule at certain points; and gelatinous infiltration of the renal connective tissue. Occasionally parenchymatous nephritis may occur; this is generally a secondary condition.

Bladder.—Areas of congestion and ecchymoses on the vesical mucous membrane may be observed, also vesical catarrh.

Nervous System.—Evidences of cerebral congestion and cerebral œdema may be present; in some cases lesions of acute meningitis are observed, with effusion into the subarachnoid spaces. A blood-stained serous fluid may be found in the lateral ventricles. Evidences of spinal meningitis may also be present.

Circulatory System.—Petechiæ or hæmorrhagic foci may occur in the epicardium, endocardium, pericardium, and even in the myocardium. Myocarditis, due to the action of toxins, to secondary infections, continued high temperature, and to alterations in the blood, may be present (Cadéac). Cloudy swelling of the muscular fibres of the cardiac walls may occasionally be observed, giving the organ a pale appearance, as if it had been subjected to the process of boiling. Acute endocarditis may occur as the result of a streptococcic infection which is secondary; the lesions are found chiefly in the vicinity of the cardiac valves. Pericarditis may also be present.

Blood.—Authorities differ with reference to the alterations that are present in the blood during life. Some claim that its power of coagulation is increased, others that it is decreased.

In that type of influenza known as "pink-eye" Williams held there was a marked tendency to firm coagulation, and that such a condition of the blood predisposed to the formation of thrombi in the heart and large vessels, and brought about the sudden deaths which are sometimes met with in this affection.

According to Cadéac, the following alterations occur: At the commencement the blood coagulates more quickly than in a normal condition; later on the separation of the constituents is rapid, but coagulation is slow. In peracute or septic cases the power of coagulation is very slight, or may be absent. In the early stages the fibrin is increased; later on it is diminished. The number of leucocytes is increased from one-third to double the normal amount, the number of red corpuscles is diminished; they become altered in shape, and tend to undergo disintegration and, just before death, to cohere in a mass by their borders. A large quantity of crystals of hæmatoidin is found free in the blood a few hours prior to death. The clot, at the outset of the disease, is firm and retractile; in the later stages it is soft. The blood-serum shows a deep yellow tint, due to the presence of the colouring matter of the bile and of biliary pigments in a state of dissolution.

According to Trasbot, the altered colour of the visible mucous membranes in influenza depends on the presence of this biliary colouring matter dissolved in the serum. After death the blood tends to early decomposition. It is of a black, tarry appearance, and does not tend to coagulate even in the cavities of the heart. Micro-organisms of putrefaction are found in the blood just prior to death and afterwards; these are the result of secondary infections.

Ocular Lesions.—These are chiefly met with in that type of the disease known as "pink-eye." They consist of infiltration and marked œdema of the conjunctivæ; the swollen upper eyelid may conceal the eyeball. The cornea and iris may be involved. Thus parenchymatous keratitis and iritis may be observed in some cases, also sanguineous or purulent effusion into the anterior chamber (Cadéac). As occasional sequelæ may be mentioned opacity of the vitreous humour, hæmorrhage into the retina, amaurosis, cataract, etc. Both eyes are generally involved.

Other Lesions.—The muscles may show morbid alterations, such as serous infiltration, a flabby texture, and a pale colour. The subcutaneous connective tissue may be infiltrated, and sometimes shows extravasations of blood, and in some areas emphysema may occur from the formation of fœtid gases. Certain joints and synovial sheaths may show lesions of arthritis and synovitis. These should probably be regarded as due to secondary streptococcic infection.

PERIOD OF INCUBATION.

Definite knowledge on this point does not exist. It is difficult, if not impossible, in many instances to decide at what time the infection enters the system, and the period that elapses prior to the development of symptoms. Again, the early symptoms may be overlooked, and the first indications presented may be those of pulmonary complications. The receptivity of the animal, and the amount and degree of virulence of the infecting micro-organisms that enter the system, will influence the period of incubation.

It is generally accepted that the period of incubation is short, and infection occurs rapidly. Some authorities state that the period is from four to seven days ; others from three to ten days. Cadéac has observed that healthy horses placed in a waggon lately occupied by an infected animal may contract the disease and succumb to it in fifty to sixty hours after their removal therefrom. Also that a healthy horse placed in a stall from which a case of influenza had been removed may show the first symptoms of the disease on the morning of the second day.

It is surmised, but not proved, that the infection may remain in an animal's system without symptoms being produced until he is exposed to predisposing influences such as fatigue or a chill.

SYMPTOMS.

The clinical picture will vary according to the type of the disease and the presence or absence of complications. Although the type varies in the outbreaks that occur in different years, it is by no means uncommon to find, even in the same stable, cases that conform to each of the types that are recognised.

As already mentioned, one of the characteristics of the disease is that a mild type may exist in one or more outbreaks, and then a virulent form may develop, which spreads rapidly, and may have a high mortality in consequence of serious complications. Some cases are of so mild a nature that they may be mistaken for ordinary acute nasal catarrh. Their true nature is not recognised until further cases occur in the stable, and present typical symptoms of influenza. Others show dulness, loss of appetite, inaptitude for work, and on examination are found to have a high temperature, but no catarrhal symptoms. If such cases are promptly kept from work and placed under good hygienic conditions, they tend to recover with simple treatment. They may be regarded as abortive or larval forms of the disease.

One of the commonest types of influenza is that known as the *catarrhal*. It is associated with acute nasal catarrh, and with a varying degree of pharyngitis and laryngitis. Rigors frequently usher in the attack, but their presence is often overlooked by the attendant. Loss of appetite, the presence of a cough, nasal discharge, difficulty in swallowing, inaptitude for work, a dull, heavy appearance, and slight weakness in the hind-quarters, are usually the symptoms which indicate to the attendant that something is amiss with the animal, and lead him to seek professional advice.

On examination, the temperature is found elevated, according to the severity of the case, from 103° to 106° F., the pulse frequent, the visible mucous membranes are injected, and in some cases show an icteric tinge; the respirations are slightly accelerated; the action of the bowels is torpid.

When pharyngitis and laryngitis are present the nasal discharge is profuse; there is marked difficulty in swallowing either solids or fluids, and when the animal attempts to drink the fluid is returned by the nostrils. The region of the pharynx may be swollen and is tender to pressure, and violent fits of coughing occur at intervals. In severe cases the head is "poked out," swallowing becomes impossible, saliva flows from the mouth, respiration is difficult, and may be accompanied by a roaring sound (see Laryngitis, Vol. II.). In such cases there is a tendency for the disease to extend to the bronchi, and if the cases be aggravated by attempts to administer drenches, broncho-pneumonia will probably be induced.

The milder cases, if treated early in hygienic surroundings, and protected from chills, tend to improve within a week, and to become convalescent in about a fortnight. In some outbreaks marked nervous depression and extreme muscular debility occurs early; the temperature runs high, and there is considerable disturbance of the heart.

The fewer generally attains its height early in the disease, and tends to abate in a comparatively short period, provided no complications arise. It may decline rapidly or gradually; in the latter instance daily oscillations may be met with.

The pulse is of a soft character, but may not be accelerated, even with a high temperature present. In severe cases it tends to become feeble and irregular.

The nervous depression is evidenced by a sleepy appearance, hanging head, drooping ears, feeble gait, weakness in the hind-limbs, difficulty in turning round. The appetite may be in abeyance, and if the animal feeds he does so slowly; thirst may be present; the mouth is dry and hot, the upper surface of the tongue "furred."

In that type of the disease known as "pink-eye" or "epizoötic cellulitis" we find, in addition to the above symptoms, a swollen condition of one or both eyelids and œdema of the conjunctivæ; the latter may be of a bright red or of a yellowish-red colour. Generally both eyes are affected; lachrymation and photophobia are present. Also, swelling of the limbs is usually present, depending on infiltration into the cellular tissue; there is marked stiffness and pain displayed on movement, and a tendency to the occurrence of pulmonary, enteric, and cardiac complications. In some outbreaks this type is the prevailing one, and the disease spreads with remarkable rapidity. The swollen condition of one or both eyelids may be the first symptom presented, and on the appearance of the first case in a stable its real nature may be overlooked until further symptoms develop or more cases appear.

In one outbreak which occurred in a cavalry regiment just as it was about to proceed to South Africa at the time of the Boer War, the first case that came under my notice was a mare that showed a swollen condition of the eyelids of one eye, and no other symptoms. The true nature of the case was not suspected, but in a few days a large number of horses were attacked in different parts of the barracks, and hardly any of the animals escaped the infection. There were no other cases of the disease in the district at the time, so the origin of the infection was a mystery. Some of the apparently healthy horses were sent to another barracks six miles distant, with the result that the disease broke out in the horses at this centre, and rapidly spread through them.

One of the characteristics of this type of influenza is the suddenness with which the symptoms develop, and its tendency to attack every horse in a large stable. We often observe in some outbreaks that, although the cases show very high temperatures and considerable swellings of the limbs, complications rarely occur. In others complications are frequently met with, and the mortality may be high in obese animals of heavy breed.

Another type of the disease is sometimes observed in which slight fever, dulness, and catarrh of the conjunctivæ, with a brick-red tint of the latter, and a gluey discharge from the eyelids, are the symptoms presented. The appetite is maintained, and recovery ensues without complications, provided the animals are taken from work sufficiently early.

The symptoms of influenza presenting complications are most conveniently studied under the following headings:

Thoracic or Pulmonary Complications.—These may be regarded as the most common of the complications of influenza.

Pulmonary Congestion.—This may occur at any period during the course of the disease. Both lungs are affected, and pulmonary œdema soon follows. It may set in early, especially in the case of cart stallions or heavy horses in high condition, purchased at shows and sent long journeys by boat or rail. Infection is probably contracted in the show-yard; the animals are tested for their wind and otherwise fatigued, and afterwards exposed to chills.

The symptoms presented are marked acceleration of the respiratory movements; great dyspnœa; in some cases slight hæmorrhage may be observed from the nostrils; extreme depression; a small, thready, gradually imperceptible pulse; a cyanotic tint in the visible mucous membranes. The temperature may be very high, ranging from 106° to 107° F.

For the physical signs, see Pulmonary Congestion, Vol. II.

These cases are usually very fatal, and death may result from asphyxia in from twelve hours to three days, or pneumonia results. In other cases pulmonary congestion may set in during the course of influenza, the symptoms may gradually subside, or the condition may pass on to pneumonia.

Pneumonia.—This is a secondary infection. It may develop insidiously, or the symptoms may arise suddenly in a case that appears to be progressing favourably. The symptoms are similar to those occurring in cases of epizoötic pneumonia (see p. 833). Careful observation and daily physical examination of the chest are necessary in many instances in order to detect the presence of pulmonary complications. Pleurisy is frequently associated with the pneumonia. In some outbreaks we find bronchitis of more frequent occurrence than pneumonia. For the symptoms of bronchitis, see Vol. II.

Gastro-Intestinal Complications.—Some authorities state that digestive disturbances of varying extent are present in all cases of influenza. Others have found that lesions of the stomach and intestine are complications occurring in certain outbreaks, so that they recognise an abdominal type or form of the disease. The latter view is more in accordance with our experience. Colicky symptoms may be present in some cases. These may depend on a paralysed condition of the stomach or intestines due to nervous influences. The ingesta tend to undue fermentation, slight tympanites and gastric and intestinal catarrh result. The symptoms of colic may be intermittent. Constipation is present, the fæces being hard, dry, and coated with mucus; or diarrhœa may occur. The dejecta may be very fœtid, and in some cases blood-stained. The anus is often dilated, and tenesmus may be present. The rectal mucous membrane is exposed and deeply congested.

Although diarrhœa is generally an unfavourable symptom, it occasionally, if not severe, may prove beneficial by relieving the constipation. More frequently it adds to the existing weakness, and hastens a fatal termination. Gastro-enteritis may occur, and is evidenced by continuous abdominal pain, a haggard expression of countenance, a weak, thready pulse, and total loss of appetite. Diarrhœa may occur at first, and be followed by obstinate constipation; the abdominal wall is retracted and painful on palpation. Gastro-intestinal complications are more especially met with in the type of influenza known as "pink-eye," but they may also occur in connection with the pneumonic form.

Nervous Complications.—In addition to the nervous phenomena which occur in all types of influenza, such as marked depression, muscular weakness, etc., serious symptoms attributable to lesions of the nervous system may present themselves. These may be manifested during the course of the disease or towards its termination. In some outbreaks cases showing nervous complications are of comparatively frequent occurrence, and we have met with a series of these in one stable.

Cerebro-spinal meningitis is, in our experience, the complication generally met with, and it is evidenced at first by a staggering gait. The animal soon assumes the recumbent posture, and struggles in a convulsive manner with the fore- and hind-limbs; is unable to rise; the eyes roll; the pupils are dilated; the sclerotic is deeply injected; micturition is frequent; the head is constantly being forced backwards; the convulsions are succeeded by short periods of calm, but soon become continuous. After a variable period the delirium is followed by a comatose condition and death; hyperpyrexia is usually present.

Cases are met with in which a comatose condition is in evidence, and is not preceded by convulsions. Other cases show spinal symptoms, such as paralysis of the hind-limbs. Paralysis of certain nerves may occur, resulting in loss of power in corresponding muscles and organs. Thus, paralysis of the rectum with dilatation of its walls, paralysis of the *sphincter ani* and dilatation of the anus, paralysis of the penis, and of some of the facial muscles, lips, etc., may be observed in some cases.

Ophthalmic Complications.—These have already been described on p. 852.

Hepatic Complications.—These give rise to an icteric tint of the visible mucous membranes of various degrees of intensity. The biliary intoxication increases the nervous depression and stupefaction that is already present. When organic changes in the liver are absent, the jaundice usually disappears as the patient becomes convalescent.

Renal Complications.—The urine often becomes of an oily consistence, due to excess of mucin; it is high-coloured, and shows an acid reaction and excess of hippuric acid. Albumin may be present. Frequent micturition may be observed, depending on either irritation or catarrh of the bladder, or on renal changes. When parenchymatous nephritis occurs the urine contains albumin, albuminous casts, blood-casts, etc.

The advent of nephritis is evidenced by dull colicky pains, the frequent passage of small amounts of urine, stiffness in the hind-limbs, a tendency to assume the recumbent position. In fatal cases the animal is unable to rise, and uræmic convulsions set in. An examination of the urine confirms the diagnosis (see Nephritis, Vol. II.). Suppression of urine is regarded by all clinicians as a very serious symptom in this affection, while polyuria may be a favourable critical sign. In one outbreak we observed that all the cases showed polyuria as an early symptom.

Cardiac Complications.—These are evidenced by tumultuous action of the heart and a small, irregular pulse. Special symptoms will accompany the prevailing lesions, and will be discovered by a physical examination of the heart (see Cardiac Diseases, Vol. II.). Cases of sudden death have been observed in the type of the disease known as "pink-eye," depending on the formation of cardiac thrombi. During convalescence from influenza it is not uncommon to meet with functional disorders of the heart, such as palpitation during exercise, an intermittent and sometimes an irregular pulse; anæmia may also be present. These tend to disappear with time and suitable treatment.

Complications in Connection with Joints and Synovial Sheaths.—Arthritis and synovitis may occur during the course of the disease, but more frequently during convalescence. They are more commonly observed in cases with pulmonary complications.

Acute lameness is manifested, with pain and swelling of the affected region, and in some cases the lesions may be of a metastatic character. The great sesamoid sheath in the fore-limb is a favourite seat for this complication; in cart-horses the tarsal sheath may be more often involved. In some cases the lameness does not appear until the animal has been put to work for the first time after an attack of influenza; it may develop suddenly while the horse is at work. Obscure cases of lameness are not uncommon after recovery from influenza. The older authors regarded these complications as being of a rheumatic character.

Influenza complicated by Strangles.—This is termed by Cadéac and others the "adeno-catarrhal" form of the disease. It is probably a secondary affection, and the symptoms resemble those met with in strangles, in addition to those of the primary affection. Some outbreaks are characterised by the large number of cases of the above type that occur. It is well known that horses suffering from influenza are very receptive to the infection of strangles.

Cutaneous Symptoms.—Gray describes an eruption of the skin as occurring in influenza, consisting of rough, elevated patches diffused over the body, of a gummy appearance when recent, and later on becoming scaly. In unclipped animals they cause an upstanding of the hair in patches. After the disappearance of the scales a hairless patch remains. Pruritus is absent. In some cases the epidermis and hair are removed in large flakes, and hairless patches of various sizes remain. Occasionally a pustular eruption is observed.* We have never observed these cutaneous symptoms in influenza.

It is not uncommon to find that during the course of the disease the hairs of the mane and tail are very easily pulled out, but this is a symptom that may occur in many debilitating affections. The cutaneous abscesses described by some authors as occurring in influenza are probably due to a secondary infection from strangles.

Laminitis may occur, especially in stallions and animals in high condition. It is generally of a subacute type, but it adds materially to the gravity of the case, and frequently proves fatal.

Purpura Hæmorrhagica may develop during the course of the disease, but more frequently it occurs during convalescence (see p. 870.)

Cadéac describes œdematous swellings as appearing on the dependent parts of the body, resembling those seen in purpura, but differentiated by the absence of petechiæ.

Abortion.—According to Cadéac, this has been observed in the pregnant mare when attacked by influenza, and the intestinal lesions of the disease have been discovered in the fœtus. It is believed to result from "the accumulation of toxic products in the blood and passive congestion and infiltration of the placenta." "Affected mares who carry to full term are unable to rear their foals; they have no milk, and after parturition they are often attacked by metroperitonitis."†

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* Paper on "The Influenzal Complaints of the Horse," by H. Gray, M.R.C.V S.

† Cadéac, "Pathologie Interne."

SEQUELÆ.

These include purpura hæmorrhagica, chronic cardiac affections, whistling, roaring, asthma, broken wind, etc. Some authorities regard the nervous affections known as "shivering" and "stringhalt" as occasional sequels to influenza.

COURSE.

The course varies according to the type of the disease, the severity of the symptoms, and the presence of complications. Mild uncomplicated cases may recover in from three to six days. A *peracute* form is sometimes encountered which is complicated with septicæmia; grave constitutional disturbance is present, a tumultuous action of the heart, feeble, irregular pulse, dyspnœa, high fever, blood-stained fœtid dejecta, etc. Such cases may succumb in two or three days. Ordinary cases with complications may continue for two or three weeks. Death may occur from many causes, depending on the nature and severity of the complications.

PROGNOSIS.

In mild uncomplicated cases, with good hygienic surroundings and early cessation from work, the prognosis is favourable. If, however, the affected animals are kept at work, and if the stables are overcrowded and ill-ventilated, complications are likely to ensue, the disease assumes a serious aspect and may have a high mortality. In debilitated, overworked horses, and also in those in fat condition and unfit for active work, the mortality, when complications are present, is usually high. Stallions kept for stud purposes are always bad subjects for the disease, especially those of the heavy breeds. Double pneumonia with pleurisy very often proves fatal, while brain lesions are invariably so. Abdominal complications must always be regarded as serious. In that type of the disease known as "pink-eye" the prognosis must always be guarded, in consequence of the danger of fatal cardiac or abdominal complications supervening, even in cases that appear to be progressing favourably.

The temperature cannot always be regarded as a reliable guide in influenza as regards prognosis. Even very high temperatures may return to normal in a short period of time, while very serious complications may exist with a moderate degree of fever. A sudden rise in temperature in a case that has run the usual course, and in which the fever has abated and the patient has been progressing favourably, is generally a serious symptom. When such occurs it usually indicates that a secondary infection has taken place, and that complications of various kinds may be expected. In cases of "pink-eye" we find that a gradual decline of temperature is a more favourable sign than a sudden abatement of the fever. A very high temperature that shows little tendency to abate, when present in connection with pulmonary complications, is always serious, and the prognosis is unfavourable. A subnormal temperature may be met with in some cases shortly before a fatal termination.

Cases complicated with strangles are always serious.

DIFFERENTIAL DIAGNOSIS.

The history of the case and the symptoms presented generally enable the diagnosis to be made with comparative facility. Influenza complicated with pneumonia may be confounded with epizoötic pneumonia. We must admit the similarity between the two affections, and also that in practice it may not be possible to differentiate them. Theoretically we may point out that in influenza the pulmonary affection does not occur at the commencement of the attack; it is a complication due to a secondary infection. In epizoötic pneumonia the pulmonary lesions occur early; they are the essential local manifestation of the disease. No doubt in a series of cases of uncomplicated influenza, or in that form known as "pink-eye," the diagnosis is sufficiently clear. A considerable clinical experience in both of these affections leads us to the belief that if they do not originate from a common cause, they are at any rate closely related.

The use of the clinical thermometer in all cases showing even slight evidences of illness will enable us to detect cases of influenza in the early stages, and thus secure isolation of the patient and cessation from work—most important details both from a prophylactic and a therapeutical point of view.

PROPHYLAXIS.

Every means should be adopted to check the spread of influenza. Apart from the risk of fatal cases occurring, the losses attendant on a number of horses becoming affected, and the long period of convalescence necessary before they are fit to resume work, must be considered. Again, the frequency with which sequelæ, such as whistling and roaring, occur after influenza renders the disease a serious one, more especially in the case of valuable hunters, as their value is diminished to a marked extent thereby.

Isolation of the affected animals is the first essential in prophylaxis. An isolation hospital for this purpose should be in existence where large studs of horses are kept. It is also desirable to separate those cases showing pulmonary complications and strangles from ordinary cases of the disease, as there is no doubt but that the latter may become infected from the former. Separate attendants, feeding utensils, clothing, etc., are also requisite for influenza cases, in order to prevent the spread of infection to healthy horses.

During an outbreak of the disease in a stable it is advisable to take the temperature of the apparently healthy animals twice daily, and all those showing a rise in temperature should be isolated at once. Whenever possible, newly-purchased horses, especially those coming from a district where the disease is prevalent, should be isolated from other horses in the stable for seven or fifteen days. As, however, the infection may exist in some horses for an unknown period without producing the disease in them, this precaution may not always be efficacious. Thorough disinfection of the stalls occupied by infected animals should be carried out, and contaminated fodder and manure should be properly disposed of. Special precautions should be taken with reference to stallions convalescent from influenza, as they are capable of transmitting the disease to mares by coition. Owners of stud horses often display great neglect in this respect, and the disease is spread to a serious extent thereby in breeding establishments.

In East Prussia notification of influenza and of contagious pneumonia has been compulsory since September, 1908. A new order came into force in Germany on October 1, 1908, compelling the declaration of these diseases.

Considering the neglect of even ordinary preventive measures that exists, it seems to us that some system of veterinary police regulations with reference to these diseases would prove of benefit in this country. These should apply more especially to livery stables, in which horses brought from outside may be exposed to infection, while horses from other districts are liable to introduce the infection. In both instances facilities for the spread of the disease are afforded.

TREATMENT.

In simple uncomplicated cases little medicinal treatment is necessary. Attention to hygienic and dietetic details is of importance, and early cessation from work is essential. Even in cases characterised by high fever antipyretics may not be necessary, as, in our experience, the temperature tends to fall spontaneously. Proper ventilation, suitable clothing, light, easily-digested food, such as bran mashes, scalded hay, boiled flax seed, etc., the judicious allowance of carrots, and of green food when it can be obtained, are essentials in treatment. A plentiful supply of pure cold water should be placed before the animal, and in this may be dissolved small doses of Epsom salt and nitrate of potassium.

When the case is seen at the commencement of an attack, and if rigors be present, a diffusible stimulant is indicated, provided there are no evidences of pharyngitis or laryngitis, which would render the administration of a drench dangerous. A suitable drench is prepared with 2 ounces of sweet spirit of nitre and 4 ounces of solution of acetate of ammonium. When pharyngitis and laryngitis are present, inhalations of steam medicated with terebene, oil of eucalyptus, etc., should be carefully carried out, so as not to annoy or distress the patient. An electuary containing belladonna, potassium chlorate, glycerine, and honey, should be given on the tongue three times daily, and a cantharides blister is to be applied to the region of the pharynx. This treatment relieves the cough and lessens the difficulty in swallowing. Milk, oatmeal gruel, linseed tea, hay tea, barley water, etc., should be allowed *ad lib.*

When the temperature is very high the indications for antipyretics must be considered. On no account should medicines in the form of drenches be given, as there is grave danger of inhalation pneumonia being induced. Quinine may be given in the form of electuary, the best basis for which is honey, as many horses take this without any trouble. In that type of the disease known as "pink-eye" we find that little medicinal treatment is required so long as complications are absent. When the pain and stiffness in the limbs is severe, salicylate of soda may be given in ½-ounce doses three times daily in the form of electuary until relief is obtained.

We cannot agree with those authors who advise the administration of a small dose of aloes as an aperient in the early stages of the affection. Not only is this drug unnecessary, but it is likely to prove positively dangerous, as the intestines are often in an irritable condition; besides, the nausea produced is distinctly harmful. If the bowels are torpid, enemas of warm water may be given, and small doses of Epsom salt prove serviceable in the food or drinking-water.

When debility and cardiac weakness are prominent symptoms, stimulants are necessary. As far as possible drenches should be avoided. We prefer the administration of carbonate of ammonia and camphor, in the form of bolus, repeated at intervals. If the temperature be high, quinine may be added.

When diarrhœa is present it should not be checked too quickly. If, however, it is continuous, it may be necessary to prescribe a few small doses of chlorodyne and prepared chalk, combined with an intestinal antiseptic such as cyllin.

The presence of slight colicky pains may be an indication of gastric or intestinal catarrh. Antispasmodics, such as a dose of spirits of chloroform in a pint of raw linseed oil, may be given. As a rule opiates should be avoided, as they check peristaltic action and favour excessive fermentation. When evidences of enteritis appear the case generally proves fatal in spite of treatment. All that can be done is to "smooth the path to death" by administering chloral hydrate combined with chlorodyne. Other complications must be treated as they arise.

Pneumonia and pleurisy are to be treated on similar lines to those advised for epizoötic pneumonia (see p. 841; and for Pneumonia, see Vol. II.). In cases where cardiac weakness and marked depression are present some practitioners have found cardiac tonics useful, such as tincture of strophanthus with tincture of nux vomica.

When prostration is extreme the subcutaneous injection of normal saline solution is indicated. Nuclein has been tried in the treatment of influenza, and so far with encouraging results; its method of employment is described on p. 841. Some practitioners find the hypodermic injection of strychnine useful in association with the nuclein where the prostration and debility are extreme.

In the nervous complications of influenza little can be done in the way of treatment so far as those cases showing evidences of cerebro-spinal meningitis are concerned. Nerve sedatives, such as chloral hydrate, may give temporary relief; but in our experience all these cases prove fatal, and destruction is the most humane course to adopt. In the case of paralysis of certain nerves local treatment is necessary, also the internal administration of potassium iodide and nux vomica.

In the so-called "bilious" type we find that no special treatment is necessary, as the symptoms of jaundice disappear as the case progresses favourably. The administration of sulphate of soda with bicarbonate of soda in the drinking-water may exert a favourable action. Some practitioners advise a few small doses of calomel, not sufficient to produce full purgation.

The ophthalmic complications are to be treated according to their nature. Early cleansing of the eyes with boric lotion is to be recommended, and the acute inflammatory conditions must receive suitable treatment.

During convalescence from influenza mild preparations of iron, such as the carbonate or the ammonio-citrate, with vegetable bitters, prove useful. The animals should not be put to work too soon, or exposed to chills, as relapses are likely to occur and complications to develop as a result.

Functional cardiac disorders, so frequently met with in cases recently recovered from influenza, are best treated with preparations of iron and nux vomica, and should not be put to work until the heart returns to a normal condition.

In the treatment of arthritic complications, and those occurring in connection with the large synovial sheaths, potassium iodide should be administered internally, and a cantharides blister applied locally.

Attention to the diet during convalescence is of importance. Large feeds and an excess of nitrogenous food should be avoided, otherwise digestive disturbances are likely to result; also azoturia may occur when the animal is too highly fed and unable to get a proper amount of exercise.

In a disease possessing so many phases as influenza it is difficult to lay down definite rules for treatment. The cases should be carefully watched and examined, so that the presence of complications may be detected early and suitable treatment adopted. The more clinical experience we gain in connection with the disease, the more convinced we become of the importance of care and nursing as regards successful results, and the less value we are inclined to place on drugs or special lines of treatment.

Serum Treatment.—Joliffe* records his experience of the employment of "influenza antitoxin" and of "anti-pneumonic serum" in cases of influenza and allied affections. Twenty-two cases were treated, the "influenza antitoxin" being used in five instances, and the "anti-pneumonic serum" in seventeen. The cases included those in which infection subsided without further complications, those in which pulmonary or pleural implications subsequently supervened, and those in which there was physical evidence of grave pulmonary lesions from the commencement of the treatment, and every indication for a very guarded or even unfavourable prognosis. Recovery occurred in every instance.

The "influenza antitoxin" employed was that prepared by Messrs. Parke, Davis and Co., the dose of which is 30 c.c., administered by intravenous injection.

The "anti-pneumonic serum" used was a Dutch product in two forms, known as "A" and "B" respectively. Both are obtained "from the blood of horses hyperimmunised against bacteria said to be associated with equine pneumonia, an ovoid organism being used in the case of the 'A' serum and a bacillus in the case of the 'B' serum.

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* *Veterinary Journal*, January, 1911.

The two sera are directed to be subcutaneously injected in 10 c.c. doses on alternate days, commencing with the ‘ A ’ serum, and continuing the alternate administration daily, according to the requirements of the case.”

Joliffe arrived at the following conclusion with reference to the two agents :

“ On the whole, judging from my own limited experience up to date, there seems good reason to suppose that both these sera, *if given early* and persevered with, and associated with favourable hygienic conditions, possess a distinct influence in increasing the patient’s resistance to the bacterial invasion of which he is the subject.”

For the clinical histories of the cases treated the reader is referred to the article from which the above quotations are taken.

IMMUNISATION.

According to Cadéac (1911), a successful method of preventive immunisation against influenza has not yet been discovered. “ The attempts at immunisation have been up to the present directed against the complications of the disease ; the results are not very encouraging. All the means adopted up to now are without practical value.”

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INFLUENZA IN THE DOG AND CAT.

By HENRY GRAY, M.R.C.V.S.,
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Does there exist in the dog and cat an acute infectious malady characterised by an elevation of temperature, a catarrhal condition of the various mucous membranes, and different types of nervous symptoms independent of distemper?

A few veterinary authorities, including Trasbot, Mégnin, and Woodroffe Hill, and amateur writers on diseases of the dog, such as the late Dr. Walsh ("Stonehenge") and Dr. Perry ("Ashmont"), consider there is such a malady; but from an analysis of the rather brief writings of the three latter authors, and the more pretentious statements of the two former eminent scientists, one gathers sufficient information to come to the conclusion that grave doubts exist as to there being such a catarrhal disease independent of distemper, both etiologically and clinically.

It is, however, quite true that some of the clinical types of distemper closely resemble the description given of human influenza, but nobody has yet satisfactorily demonstrated that, under the so-called generic term of "distemper" there are in reality two or more specific morbid entities confounded together. On the contrary, Carré, of Alfort, has shown that every type of canine distemper that he has investigated is due to an invisible virus. No authority as yet has elucidated the question of the duality or multiplicity of the specific etiological factors of what is known under the term "distemper." In man influenza is distinguished not only clinically, but also bacteriologically, from other human infections; it is due to a specific bacillus, the *Bacillus influenzæ*, isolated by Pfeiffer, which is also pathogenic for the monkey, and probably for the rabbit. It manifests itself in four main types—viz., the respiratory, the gastro-intestinal, the nervous, and the febrile. But these types vary not only during the period of its prevalence, but also in the same outbreak in a household.

Trasbot and Mégnin consider that the distinguishing character between canine *la grippe*, infectious pneumonia, or the distemper of the English, and the "true malady" or distemper, to be the presence of the varioloid eruption in the latter, and its absence in the two former diseases. Woodroffe Hill, Walsh, and Perry make no mention of this or of any other diagnostic sign, and therefore leave their readers in the dark with regard to the differentiation of one disease from the other. They do not go into much detail, and such evidence as they produce is more in favour of the disease being distemper than influenza. In fact, "influenza" is a term very frequently used by those who have not given a very serious attention to canine pathology.

Trasbot believes *la grippe,* or infectious pneumonia, is very frequently engrafted on true distemper, which he considers an eruptive or variolous disease. Lignières states that the distemper induced by his Pasteurella differs from that due to the invisible virus.

Some authorities describe a contagious bronchitis, not taking into consideration that distemper manifests itself in various types according to the season or epoch.

A few other authorities look upon canine and feline typhus, or "Stuttgart-epizoötic," to be the influenza of the dog and cat, quite distinct from distemper. The reason, purely clinical, given for this differentiation is that in typhus dogs and cats of all ages are affected; that catarrhal or febrile symptoms are rarely manifested; that vomiting and diarrhœa are very pronounced; that gangrenous stomatitis is frequently present; and that the mortality is, especially in old dogs, very high (*vide* Canine Typhus, p. 735).

On the contrary, distemper usually attacks young dogs; catarrhal and febrile symptoms are generally present; vomiting is comparatively rare; cutaneous, nervous, and pneumonic manifestations are common; the disease usually runs a protracted course; and the mortality is moderate compared to that of the former malady.

It may not seem out of place to mention here that those who have given most attention to canine medicine are unanimously silent on the dual or multiple etiology of distemper; or, rather, not one of the leaders of canine pathology has attempted to describe an influenza of the dog or cat independent of distemper.

Probably, on a further bacteriological investigation, combined with a careful clinical study of canine maladies, many affections generally included under the generic denomination of "distemper" will be isolated and defined. But until this is accomplished, the writer opines there is no justification for considering that the dog is affected by an influenza as well as a distemper, and that these two terms should still be regarded (as they have always been) as synonymous, or at least the question should be left *sub judice.*

PURPURA HÆMORRHAGICA

By E. WALLIS HOARE, F.R.C.V.S

Synonyms.—Petechial fever; Morbus maculosus; Horse typhus.

French: Anasarque; Maladie pétéchiale.
German: Hautwassersucht.

Definition.—A microbic disease generally occurring as a secondary condition to influenza, strangles, and other infective processes, and characterised by the presence of petechiæ on the mucous membranes, skin, and other structures, and extensive œdematous cutaneous swellings on various regions of the body.

GENERAL REMARKS.

In former times various views were held with reference to the nature of this disease. The older writers imagined that it possessed features in common with certain maladies in the human subject such as typhus fever, but the two affections are essentially different. The disease known as purpura hæmorrhagica in man bears little resemblance to the affection we are discussing.

The nomenclature of the disease is not satisfactory, as none of the terms employed express clearly the nature of the malady or its characteristic features. Various authors expressed the view that the disease depended on certain alterations in the blood and capillary vessels. Modern authorities, however, do not accept these statements as correct. According to Friedberger and Fröhner, "the blood shows no essential changes so long as the disease progresses without complications." Cadéac states that "the blood does not show any appreciable alterations at the commencement. It coagulates as quickly and as completely as in the normal condition; it is even richer in fibrin; the contraction of the clot is more intense; the serum separates from the clot with greater rapidity for twenty-four hours than in the normal state, but its proportion is diminished—*i.e.*, there is less serum in a given quantity of blood in this diseased condition. Later on the blood is found to contain a larger amount of serum than normal blood." *

Modern authors regard the affection as being a microbic one, although the specific micro-organism has not yet been determined, nor has it been demonstrated that the disease can be conveyed in the ordinary manner from one horse to another. It is not uncommon, however, to find that when one case of the disease occurs in a large stable others may follow. The œdematous swellings, the petechiæ, and the other phenomena found, are believed by some authorities to depend on a vaso-dilatory action of the toxins formed by the micro-organisms.

The disease is in the large majority of instances a secondary one, usually occurring as a sequel to strangles, influenza, and epizoötic pneumonia. Williams and Robertson described under the heading of "scarlatina" a special disease of the horse, but a careful consideration of the symptoms shows that in reality the affection is but a form of purpura hæmorrhagica. In any case, the term applied was most unfortunate, as the affection bears no resemblance whatever to scarlatina in man. According to Cadéac, purpura hæmorrhagica has never been observed in the mule or donkey. It may, however, occur in cattle (see p. 887).

ETIOLOGY.

As already mentioned, the affection is generally a secondary one. Occasionally cases are met with in which there is no evidence of a pre-existing disease. We observed one case which occurred in connection with a septic wound on the hock. The animal was kept in a very unsanitary stable, but previous to receiving the injury was in perfect health and condition. Cases are recorded by several authors in which the disease followed various conditions, such as injuries, surgical wounds, fissured heels, traumatic arthritis, abortion, internal abscesses, castration, docking, firing, etc.

The association between strangles and purpura is well known. The latter may appear at any stage of the former, but more frequently towards the termination of the attack. The severity of the primary disease does not appear to have any predisposing influence on the occurrence of purpura, as the latter may appear in connection with mild attacks of strangles. Judging by the large number of outbreaks of strangles that occur, and the comparatively few cases of purpura that result, we are led to the conclusion that the nature of the infection and the circumstances influencing its entrance and development in the body have yet to be discovered. We see many cases of strangles and of influenza kept under

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* "Pathologie Interne."

the worst conditions and surroundings, and yet purpura may not develop. On the other hand, purpura may be met with in connection with cases of the above affections in which the symptoms of the latter are not severe and the surroundings are good. In some outbreaks of strangles and influenza a number of cases of purpura appear, and we are unable to offer an explanation for this circumstance.

BACTERIOLOGY.

From time to time various micro-organisms have been discovered in the œdematous swellings and in the blood. Burke* found cocci, diplococci, and bacilli in the form of slender rods. " Cover-glass preparations of the blood and serum were allowed to stand over for several days, and then examined. The cocci assumed zooglœa masses, and the bacilli or rods appeared in colonies."

Limont† examined the exudate from the œdematous swellings in living animals, also the blood from areas free from swellings, also blood drawn from the jugular vein under aseptic conditions, and in every instance discovered micro-organisms, generally in the form of micrococci. After death, sections of the swollen eyelids and lips showed large cocci and bacilli of various lengths. The bacilli were found in the lumen of the capillary vessels and in their walls, also in the surrounding connective tissue.

Cadéac and Lucet found *Staphylococcus aureus* and *S. albus* in the serosity from the œdematous swellings. Lignières discovered various micro-organisms, and isolated streptococci by inoculating the muscles of a rabbit with impure cultures, blood, and the products of organs taken from horses dead from purpura. The streptococcus thus isolated had no specific action, and was a small variety of the streptococcus of strangles (Cadéac). Lignières also found the human type of erysipelas strepto coccus and the streptococcus of strangles.

Kolb discovered the *Bacillus hæmorrhagicus*, similar to the micro-organism met with in the disease known as *morbus maculosus* in man, and found in the blood three or four hours after death. Delamotte and Clarc have found *Streptococcus pyogenes ;* Coquot has found a Pasteurella. Further investigations are necessary in order to discover the specific micro-organism which is capable of producing the disease. The effects produced would suggest that the theory of vaso-dilatory toxins seems the most probable, but the source of these toxins has yet to be determined.

Cadéac expresses the view that when purpura occurs in connection with strangles, the toxins secreted by the streptococci and the staphylococci of suppuration are apt to produce purpura when they become absorbed. This absorption, he says, is more likely to occur in deep-seated abscesses which do not rupture than in superficial ones which soon arrive at maturity.

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* *Veterinarian*, 1887.

† *Veterinary Journal*, 1887.

The problem to be solved, however, if we accept this view, is, What are the conditions which induce the streptococci to secrete these toxins, or what are the influences which act on the animals' systems so as to render them susceptible to the action of these toxins? Purpura can occur in the absence of deep-seated abscesses, and, *per contra*, the latter condition is in only a small proportion of cases associated with the former. Possibly an ultravisible virus may yet be found to exist, which is capable of exerting these influences.

Dr. Hale White regards purpura hæmorrhagica in man as depending on endothelial toxins which act on the vessels, causing damage to their walls.*

According to Lignières, the serum obtained from the erysipelas type of human streptococci (*Streptococcus pyogenes* or *S. erysipelatus*) is very active against the streptococcus which is most often met with in purpura of the horse. This streptococcus has all the qualities of the streptococcus of human erysipelas. The human antistreptococcic serum (*i.e.*, erysipelas type), on the other hand, has no action against the streptococcus of strangles. "This speaks in favour of the identity of the streptococcus of erysipelas in man and that of purpura in the horse."†

Labat regards purpura as a complication or a secondary affection, which never occurs as a primary disease. It may appear during the course of, or as a sequel to, certain affections—viz., contagious pneumonia, glanders, strangles, pharyngitis, influenza, suppurations, wounds, etc. The proofs of infection in the disease have not been established, and attempts at inoculation and transfusion of blood have never succeeded in producing the affection in a healthy animal. Probably both the human streptococcus and the streptococcus of strangles may be regarded as the micro-organisms on which the disease depends. Lignières has demonstrated their frequent association in the same animal. The strangles streptococcus has been demonstrated in the serosity of the œdematous swellings, and, according to Drouin, accidental inoculation of strangles pus by way of the veins, during the opening of an abscess, has on two occasions produced a serious attack of purpura. Again, horses inoculated with gradually increasing doses of the cultures of the human streptococcus, during the process of obtaining Marmorek's serum, often show engorgements of the limbs and abdomen analogous to those seen in purpura. It sometimes happens during the course of this process of immunisation that the horse shows œdematous swellings and petechiæ similar to those occurring in typical cases of purpura. The various types of purpura met with depend on the micro-organisms which produce the disease. These micro-organisms may be of various kinds; they may act singly or be associated together.‡

With reference to the rôle of influenza in the production of purpura, the following is deserving of mention: Forty-six horses were inoculated with 2 c.c. of a culture of the equine Pasteurella for the purpose of immunisation. Fifteen of these developed purpura; one showed the typical symptoms of the disease, and died seven days after; one died on the eighteenth day, after showing typical symptoms; and thirteen showed more or less marked symptoms of purpura and recovered.

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* *Lancet*, February 17, 1912.

† Lignières, paper on "Pathology and Therapeutics of the Streptococci in the Domestic Animals," Hague Congress, 1909.

‡ Labat, paper on "Pathology and Therapeutics of the Streptoccoci in the Domestic Animals," Hague Congress, 1909.

ACCESSORY OR PREDISPOSING CAUSES.

Exposure to cold, chills, fatigue, etc., may be regarded as predisposing causes. Ill-ventilated, badly-drained, dark stables also act as predisposing causes by lowering the vitality of the animal and rendering him more susceptible to infection. Under surroundings of this kind the disease is more apt to assume a severe type and to develop complications.

IMMUNITY.

It is doubtful whether one attack of the disease confers any immunity. We have never met with a second attack in the same animal. No doubt the course of the disease is often prolonged, and during this period relapses are not uncommon. Recurrences have been met with, and three attacks observed in one year. Gray has seen two attacks in the same animal, with an interval of six months between them. The horse was at work during the interval.

MORBID ANATOMY.

The lesions are chiefly found in the skin, subcutaneous tissue, mucous membranes, muscles, and in severe or complicated cases they also appear in many of the internal organs. Petechiæ, ecchymoses, and extensive œdematous swellings constitute the striking phenomena in the disease; also the tendency to hæmorrhagic infiltrations and necrotic changes.

Skin.—When the skin is light-coloured, petechiæ may be discovered. Ecchymotic patches of various sizes due to hæmorrhagic infiltrations may be present. The œdematous swellings occur especially on the most dependent parts of the body. They depend on infiltration of the subcutaneous connective tissue with a blood-stained, imperfectly coagulated gelatinous material. In the region of the limbs deep fissures may be found, especially at the flexures of joints. Extensive sloughing of the skin may occur, as the result of which the underlying muscles and tendons may be exposed. Occasionally a dry, leathery appearance of the skin and subcutaneous tissue is met with.

The Muscles, especially those of the abdomen, quarters, thighs, chest, neck, and jaws, are studded with hæmorrhages, and the intermuscular and interfascicular connective tissue is infiltrated with serosity. The muscular fibres are of a pale colour, and show granular or fatty degeneration (Cadéac).

The Nasal Mucous Membrane is covered by petechiæ, which tend to become confluent and form large irregular patches of a dark purplish-red colour. Infiltration of the connective tissue and vascular distension may be so extensive that the lumen of the nasal cavities is rendered extremely narrow. Ulcers may form on the nasal mucosa, also areas of necrosis, depending on a secondary microbic infection. The *septum nasi* may be perforated by necrosis.

Larynx.—The laryngeal mucous membrane may show infiltration, hæmorrhages, and ulcers, and œdema of the glottis may be present.

Lungs.—A variety of lesions may be observed, such as pulmonary œdema, hæmorrhagic foci of various sizes, limited areas of necrosis, evidences of broncho-pneumonia, pulmonary gangrene, etc.

Pleural Cavity.—This may contain a sanguineous effusion, but evidences of pleuritis are usually absent, except when extension from the pulmonary affection occurs.

Mouth and Pharynx.—The mucous membrane of the lips, the gums, tongue, and pharynx may show petechiæ, hæmorrhages, and infiltration.

Stomach.—Petechiæ or diffuse ecchymoses may occur in the serous, muscular, and submucous layers; also on the gastric mucosa. Ulcers may be met with in some cases.

Intestines.—Similar changes to those in the stomach may be observed. Ulcers may occur chiefly in the duodenum, less often in the cæcum, colon, and rectum. Serous infiltration and hæmorrhages may be met with in the large intestine; the intestinal contents may be intermingled with blood and contain masses of exudate of various sizes. Intestinal paralysis is not uncommon, and is due to impaired circulation, such as engorgement or rupture of the capillary vessels of the viscus. Invagination of the intestine has been observed in some cases. Its mode of occurrence, as explained by Cadéac, is that a normal loop of intestine may be invaginated into a paralysed and dilated loop of bowel in the vicinity.

Liver.—In some cases little alteration is found in this organ; in others it may show passive congestion, and occasionally undergoes softening.

Spleen.—Occasionally extensive nodular hæmorrhages have been observed. Cases of rupture and internal hæmorrhage as a result are recorded.

Kidneys.—Interstitial infiltrations and ecchymoses may be present.

Bladder.—Petechiæ, and sometimes diffuse hæmorrhages, may be observed. Similar changes may occur in the vaginal mucous membrane in the mare.

Heart.—This organ may be pale in colour or show ecchymoses. The pericardium and endocardium may show petechiæ and hæmorrhages. Hydropericardium may be present.

Arteries.—According to Zschokke, pale thrombi have been found in the mesenteric arteries, also in a pulmonary bloodvessel. In some instances firm clots adherent to the walls of the vessel were observed.

Nervous System.—In those cases that develop severe nervous symptoms prior to death, the cerebro-spinal meninges show petechiæ and serous effusion, the latter causing pressure on the brain and cord. Williams described a specimen of the brain and spinal cord from a case of purpura in which the arachnoidean cavities, both cerebral and spinal, were filled with extravasation. The nerves are sometimes involved. Barreau observed at the autopsy of a horse that died from purpura, after showing symptoms of paralysis on the fifteenth day from the commencement of the attack, lesions in the femoro-popliteal and great sciatic nerves. The lesions consisted of "an extensive infiltration around the tissue of the nerve, and even in the substance of the nerve tissue itself" (Cadéac).

Other Lesions.—Pleuritic effusion, hydrothorax, and ascites may be met with in some cases. Lesions of the joints and synovial sheaths occasionally occur.

As already remarked (p. 869), the blood shows but slight alterations in the early stages of the disease. When complications occur and septic changes are induced, the blood becomes dark in colour, incapable of coagulation, and shows all the alterations characteristic of septicæmia. Various micro-organisms are present in it, such as those of septicæmia and putrefaction, associated with streptococci and staphylococci.

Abscesses in various parts of the body and purulent infiltrations may be met with in cases of purpura accompanying or succeeding to strangles.

SYMPTOMS.

In the early stages the symptoms may be neither regular nor constant. When the affection supervenes on an attack of strangles or influenza, the practitioner in attendance may observe slight swelling of the nostrils and lips, and this leads him to examine the nasal mucous membrane for the presence of petechiæ. Or the primary manifestation may be pain, lameness, and slight swelling in a hind-limb.

In some instances the characteristic symptoms—viz., the presence of petechiæ on the nasal mucous membrane and of the œdematous swellings on various parts of the body—may be rapidly developed, and quite apparent at the first examination of the case. Thus, as the disease may follow mild cases of strangles or of influenza, the occurrence of the œdematous swellings is observed by the owner, and leads him to call in the practitioner for the first time.

The presence of the primary disease is often overlooked, especially in the country, and this fact will account for many of the cases of purpura that are regarded as independent affections. In some cases the first symptom observed is the presence of a generalised eruption of rounded or oval elevations of the skin of various sizes, strongly resembling an attack of urticaria. Unless the nasal mucous membranes be examined, the real nature of the case may be overlooked. This eruption may occur very suddenly. In other instances the first symptom to attract attention is the trickling of a sanguineous fluid from the nostrils.

The Petechiæ occur on the nasal mucous membrane, the conjunctivæ, the mucous membrane of the lips, the gums, tongue, pharynx, larynx, and on the skin. They are most frequently met with on the nasal mucous membrane, and they generally precede the appearance of the œdematous swellings. The petechiæ on the skin are only recognised when this structure is of a white colour; they cannot be seen when it is pigmented. When the swellings appear to precede the petechiæ on the mucous membranes, it is probable that the latter first occur on the skin, but are not perceived.

The petechiæ commence as small red punctiform spots resembling flea-bites. They tend to coalesce and form patches of various sizes, with irregular outlines, and in some cases may ultimately cover the greater part of the nasal mucosa. They do not disappear on pressure, and hence are generally regarded as due to extravasations of blood. According to Cadéac, true hæmorrhage does not occur in the majority of the petechiæ. The blood-corpuscles accumulate at the seat of the lesions, the lumen of the capillary vessels is enormously increased, but the corpuscles do not generally leave the vessels. The colour of the petechiæ varies according to their age. At first red, they become of a purplish or mulberry colour or of a brownish tint. These alterations in colour depend on changes in the colouring matter of the blood at the level of the lesions.

The petechiæ are sudden in their appearance, and may disappear as rapidly. They tend to occur in successive eruptions throughout the course of the disease; hence various tints are shown according to their age. They may be followed by exudation, which induces an œdematous swelling of the nasal mucosa. Generally a sero-sanguineous fluid issues from the nostrils, but in some instances it is distinctly hæmorrhagic in nature.

Necrotic changes occur in the petechiæ that persist on the nasal mucosa and on the mucous membrane of the lips, but not in those occurring on the conjunctivæ; hence Cadéac regards these alterations as depending on secondary infection from the mouth and from the air. These necrotic changes are evidenced in the petechiæ by a greyish or leaden tint, and by a fœtid discharge containing shreds of necrotic tissue.

The Œdematous Swellings.—These form a prominent symptom in the disease. They appear at first in the form of rounded or oval elevations of the skin, from ¾ inch to 3 inches in diameter; in some cases, as already remarked, they resemble the eruption of urticaria. Their favourite seats are the limbs, lower portion of the face, nostrils, lips, chest, abdominal region, and sheath. They soon become confluent and increase in size, forming extensive œdematous swellings.

In the limbs the swellings may start at the fetlocks, and quickly extend towards the body. Usually they are sharply defined at a certain point, giving an appearance as if a ligature had been applied round the part. This peculiarity is also observed in other regions. The swelling in the limbs may be so extensive that the contour of the joints is concealed, and the œdematous parts may join those on the trunk and abdomen. In the dependent parts of the thorax and abdomen the swellings are very extensive, and usually continuous. They may spread from side to side and from the sternum to the pubic region.

On manipulation they do not show abnormal heat, are only slightly tender, and usually pit on pressure. In the head the swellings start at the nostrils, nose, and lips, and extend upwards to the border of the masseter muscles, and cease abruptly; or they may extend to the eyes and to the anterior border of the neck, causing the animal's head to resemble that of a hippopotamus. The swellings on the head and face are hard and resistant, thus differing from those in other regions; and on the nostrils, lips, eyelids, and sheath the skin is tense and of a shiny appearance. If a halter be worn, the noseband causes an indentation on the swollen nasal region. The swollen condition of the nostrils and of the nasal mucosa interferes with respiration, and when extensive, extreme dyspnœa is produced.

A peculiarity with reference to the œdematous swellings in purpura is their tendency to disappear from one region of the body and to reappear in another. Moreover, they may commence in the region of the nostrils and lips or in the limbs. Again, they may disappear for a day, and lead the practitioner to the belief that a rapid recovery is likely to occur, but without any warning fresh swellings manifest themselves. Or they may suddenly disappear, and evidences of serious pulmonary or intestinal complications may develop. From the surface of the swellings a small amount of sanious serum may exude, and the nutrition of the skin may be interfered with to such an extent that sloughing may occur at certain points.

As a rule the swelling remains in the limbs during the course of the disease, and in consequence of the extreme distension of the skin the movements of the limbs may induce deep fissures at the level of the flexures of joints, such as the pastern, knee, and hock, followed by secondary infection and serious complications.

A Vesicular Eruption, occurring especially on the hind-limbs, is a common symptom. It tends to appear in successive crops, and may also be observed inside the limbs, where the skin is fine, and in the hollows of the pasterns. The vesicles rupture and discharge a yellow viscid fluid, which trickles down the skin, and has an irritant effect on the latter, producing excoriations. In the hollows of the pasterns the vesicles may become confluent and form deep fissures.

Constitutional Symptoms—*Fever.*—When purpura accompanies the primary disease, its advent generally produces an increase in the fever already existing. At times the rise in temperature is but slight, unless complications occur. In some cases, however, hyperpyrexia may be observed; in others the fever is lessened on the appearance of the œdematous swellings. It is characterised by a tendency to oscillations during the course of the disease.

The Pulse.—In cases where the weakness and debility of the primary disease are in existence, the pulse is soft, and tends to become feeble. At times it assumes a dicrotonous character. The heart-beats may show irregularity, and later on the cardiac impulse becomes feeble. In cases where the primary disease has been of a mild type, the pulse in the early stages of purpura may only show slight alterations, such as increased frequency.

Marked Depression is generally present, but in some instances the patient may become brighter in appearance on the advent of the swellings. This, however, is only temporary.

In consequence of the swollen condition of the limbs and the stiffness of the joints, progression is painful, and the animal can only be moved with difficulty. In severe cases he stands persistently in the same spot, and does not attempt to lie down. Occasionally rigors may be observed prior to the advent of the swellings.

The Appetite is not lessened in the early stages, but later on, in consequence of the swollen condition of the lips, prehension of food becomes difficult, and the swelling of the head interferes with the movements of the tongue and jaws, thus preventing mastication, while the pharyngeal lesions render deglutition painful.

Respiratory Phenomena.—*Dyspnœa* may result from the swollen condition of the nostrils, from the tumefaction of the nasal mucosa, and from infiltration and œdema of the larynx. When these conditions are severe, symptoms of asphyxia occur. The respiration becomes of a "snuffling" character, owing to the swollen condition of the nasal mucosa and to the nasal discharge mixed with necrotic débris.

The Nasal Discharge varies in appearance. It may be of a sero-sanguineous character, or it may contain débris of food, saliva, etc., resulting from pharyngeal lesions, and be fœtid, or pure blood may trickle from the nostrils, arising from the nasal passages, pharynx, or even from the bronchi and lungs.

Pulmonary Œdema may appear suddenly, and give rise to marked symptoms of asphyxia. This may supervene in cases where the external swellings suddenly disappear. Inhalation pneumonia may occur from portions of food, fluids, saliva, etc., entering the trachea and bronchi, owing to the pharyngeal lesions present. As a result, pulmonary gangrene ensues.

Phenomena in Connection with the Digestive Organs.—Occasionally the tongue may be swollen and covered with dark-coloured vesicles. Evidences of pharyngitis are often observed, and paralysis of the pharyngeal muscles, owing to excessive œdema, contributes to the difficulty in swallowing and to the character of the nasal discharge already mentioned.

Various intestinal complications may occur during the course of the disease. Thus, slight diarrhœa depending on intestinal exudation may be observed. Colicky pains of varying degrees of intensity may occur, accompanied by a profuse sanguineous, fœtid diarrhœa. Acute enteritis of a hæmorrhagic character may occur, or obstinate constipation depending on intestinal paralysis. Occasionally invagination of the intestine results (see p. 874). These abdominal phenomena tend to occur when the external swellings disappear in a sudden manner, and a fatal termination always results in such instances.

Nervous Phenomena.—Paraplegia may be observed in some cases, occurring early in the disease. Instances are recorded in which the leading symptoms presented were convulsive fits suggestive of meningitis, without the usual evidences of the purpura. Williams described such cases, and found that the only symptom of the disease present was a mulberry, purple or claret colour of the tongue. He also recorded instances in which complete loss of appetite, marked dulness, no pain, the surface of the body cold, the pulse imperceptible, were the only symptoms present, death occurring within a few hours. The autopsies showed petechiæ in several of the internal organs, and grave alterations in the character of the blood. Enteritis was present in some of the cases.

In the present day it would be reasonable to suggest that some at least of these cases were due to anthrax, as no bacteriological examination was carried out with a view to a differential diagnosis.

Renal Phenomena.—The urine may be diminished in amount after the swellings appear. Albumin has been found in the urine by some authorities, and is believed to depend on alterations in the blood, and not on renal lesions. It is not persistent (Cadéac). Hæmaturia is not uncommon during the course of the disease. Cadéac has discovered diplococci and streptococci in urine withdrawn with aseptic precautions from cases of purpura. Micturition is often difficult or impossible in cases where extensive swelling of the sheath is present.

Ocular Phenomena.—The conjunctivæ are swollen and studded with petechiæ. The eyeballs may be completely covered by the upper eyelids, and lachrymation occurs. Various complications may result, such as keratitis, and lesions of the iris, the choroid, and the retina. Amaurosis has also been observed. In rare cases extravasation may take place within the periorbital sheath, and protrusion of the eyeball results (exophthalmos). Or the extravasation may be intraocular, so that destruction of the eye may result in some instances.

COMPLICATIONS AND SEQUELÆ.

In protracted cases septicæmia or pyæmia may appear as secondary conditions. Cicatricial contractions of the skin over the flexor surfaces of joints, due to ulcerating cutaneous lesions, may interfere with the free movements of the limb. Paraphimosis and paralysis of the penis have been observed. Extensive sloughing of the skin and connective tissue may occur in the region of the swellings, so that the underlying structures are exposed.

Occasionally degenerative changes or necrosis of the flexor tendons have been observed, resulting in detachment of their insertion and descent of the fetlock. Separation of the coronary band from the horn of the wall occasionally occurs, and as a result the foot may be shed. Robertson met with instances in which cardiac affections occurred as sequels to purpura, and rendered the animals permanently useless.

COURSE.

The course of the disease is generally irregular, and relapses are likely to occur. In mild cases the symptoms may continue from eight to fourteen days before improvement commences; in some instances convalescence may occur in even less time. In severe cases the symptoms may continue for a period of four to six weeks, and may be of a remittent character. Sometimes the disease runs a chronic course, and may continue for upwards of two months.

The rapid disappearance of the swellings is likely to be followed by pulmonary œdema or by intestinal complications. In rare cases petechiæ may occur without any local swellings, and the disease appears to be abortive; or the swellings may be present in the head only, or in the limbs only, or petechiæ may be absent from the nasal mucosa. As already mentioned, recurrences of the disease within a variable period may be met with.

Death may occur rather suddenly in some cases, with evidences of pulmonary, nervous, or enteric complications. In some instances the disease runs a peracute course, showing hyperpyrexia, extensive petechiæ, and very large swellings.

PROGNOSIS.

A guarded opinion should always be given in cases of purpura, in consequence of the remittent character of the disease and the liability to complications. Some cases appear to progress in a favourable manner until complications develop without any warning. The mortality varies to a marked extent. The affection may prove very serious even in cases where the primary disease was of a mild character.

Amongst the favourable signs are an absence of high fever and of dyspnœa, a fair appetite, moderate swellings, especially in the region of the head, and no evidences of complications; also the gradual disappearance of the swellings.

Unfavourable signs are complete loss of appetite and inability to feed, excessive swelling of the nostrils and nasal mucosa, evidences of necrotic changes in the latter, fœtid breath, marked dyspnœa, high fever, feeble pulse, the presence of pulmonary, nervous, or enteric complications, hæmaturia, sloughing of the skin over the swellings, and secondary infection therefrom. Sudden disappearance of the swellings should always be regarded as an unfavourable sign. Instances of recovery in severe cases, in which the result could not be attributed to treatment, have been recorded.

DIFFERENTIAL DIAGNOSIS.

In cases presenting typical symptoms no difficulty will arise in connection with diagnosis. Atypical cases, or those in which one or other of the classical symptoms are absent in the early stages, may lead to an error in diagnosis. Petechiæ may be present in other diseases than purpura, such as acute glanders, anthrax, horse-pox, septicæmia, and rarely in urticaria. The presence of petechiæ, however, during the course of strangles, influenza, etc., or during convalescence from these affections, is highly suggestive of the commencement of an attack of purpura. Hence the history of the case, the presence of petechiæ, and the character of the swellings, are the salient points on which a diagnosis is to be based.

The following diseases may require to be differentiated from purpura:

Glanders.—When a horse suffering from glanders becomes affected with purpura, the diagnosis presents considerable difficulty. Mallein cannot be employed in consequence of the existing fever, and from the fact that this agent is found to cause an extensive swelling in cases of purpura. Inoculation of the nasal discharge or of the serosity of the swellings into susceptible animals is the only means of arriving at a definite diagnosis in an expeditious manner (Cadéac). For further information on the differential diagnosis of glanders and purpura, see Glanders (p. 88).

Anthrax.—This disease possesses certain analogies with those cases of purpura which succumb rapidly without external evidences of the nature of the affection. A bacteriological examination is the only means of arriving at a definite diagnosis. As already remarked, it is probable that at least some of those recorded cases of purpura of the above type were in reality anthrax, as in the present day it has been demonstrated that the latter disease is of far more frequent occurrence in the horse than was formerly imagined, and the history and the lesions described were certainly suggestive of anthrax.

Erysipelas.—In this affection the swelling is generally gradual in its development, and confined to one limb. It is very painful, and not always remittent in character. If petechiæ are present, they are not so well marked as in purpura. The head is not involved in erysipelas unless in cases resulting from wound infection in this region, when a swelling very similar to that of purpura may be met with affecting the nostrils, face, and eyelids (see Erysipelas, p. 252).

Urticaria.—At the outset of both diseases the skin eruption may be similar. Extensive swelling of the head and face may also occur in some cases of urticaria. Petechiæ, however, are rare in urticaria, and the eruption and swelling are of short duration. In some cases of purpura the initial symptom is an urticarial eruption, which may disappear, to be followed by the characteristic swellings and other symptoms.

Variola Equina, or Horse-Pox.—Before the appearance of the characteristic eruption of this disease, the swelling of the limbs, the fever, and the petechiæ might be mistaken for symptoms of purpura. When the pustules appear, the differential diagnosis presents no difficulties (see Variola Equina, p. 580).

TREATMENT.

Attention to hygienic and dietetic details is of first importance in treatment. Proper ventilation and drainage of the stable is essential, and good nursing is of quite as much importance as medicinal treatment. Nourishing food should be supplied, such as oatmeal gruel, boiled flaxseed, scalded hay, barley-water, etc., and milk should be allowed *ad lib.*

Various drugs have been advised for the treatment of this disease, and up to the present it cannot be said that a rational or successful method of treatment has been discovered. In some cases the time-honoured method of treatment with oil of turpentine gives good results, but it has the disadvantage that it must be given in the form of a drench, and repeated. Not only have we to consider the risks of drenching, but also the fact that in severe cases of the disease, with extensive swelling of the head and the presence of pharyngeal lesions, this method of administration cannot be carried out. The oil of turpentine is given in doses of from 6 to 10 drachms, well shaken up with milk and eggs, two or three times daily, until improvement in the symptoms is apparent. The risks of the drug inducing renal irritation must be borne in mind, and the doses must be reduced after a few days. Potassium chlorate may be given in addition to the above, and was highly recommended by Williams. Tincture of perchloride of iron is combined with the oil of turpentine by some practitioners. In ordinary cases of the disease we have seen good results from the above treatment. In addition, inhalations of steam medicated with antiseptics prove of value.

In severe cases, where drenching is impossible, various methods of treatment are advised. The intratracheal injection of Lugol's solution of iodine was suggested and advised by Dieckerhoff. Opinions as to the value of this method are very variable. Some authorities condemn its employment, and state that serious sequelæ have resulted, such as necrotic tracheitis, bronchitis, and pulmonary gangrene.

W. H. Bloye, F.R.C.V.S., of Plymouth, has found this treatment most successful, and has kindly favoured us with the following description of the technique he employs and the results obtained :

" In this country, as far as I am aware, practical attention was first directed to Dieckerhoff's method of treatment of purpura hæmorrhagica in an article by J. A. W. Dollar, which appeared in the *Veterinary Record* some time in 1896. It contained an epitomised translation of a report of some cases by Schaumkell, who had sucessfully applied the treatment. To that article I am entirely indebted.

" Let me say at once that I have found this method of treatment almost invariably successful ; that its beneficial effects are seen within a comparatively short time—certainly well within twenty-four hours.

" I have never known a case of skin sloughing to occur since its adoption, and, further, as clearly showing the extraordinary efficiency of the method, the prominent symptoms—swellings and petechiæ—can almost be experimented upon by lessening or withholding the dose, and again increasing it.

" And it is this question of dosage which has, I believe, been responsible for the disfavour with which the treatment has been received in some quarters.

" It has been stated that Dieckerhoff originally recommended a dose of from 3 to 9 drachms of the iodine solution. Schaumkell frequently gave as much as 18 drachms, morning and evening, and, following his lead, I never, in bad cases, give less than this amount for the first two or three doses, varying afterwards according to the severity or otherwise of the symptoms, and gradually lessening until I cease the administration altogether, and put the patient on to iron, or iodide of iron, given in the form of bolus. (Parenthetically, it may be mentioned that pure iodine, even in quite large doses, is wholly inefficient when given internally during the active stages of the disease.)

" Of course, recurrences of the symptoms do take place, after the manner of the disease, but not so frequently or so severely as under the old treatment. When they do occur, however, and when possibly the dosage has been lessened too quickly, they can readily be combated by again increasing it.

" The injections have rarely to be continued over more than four or five days, and frequently during the latter of these a single daily dose is sufficient.

" The solution employed is composed of—

Iodine	1 part
Potassium iodide	5 parts
Boiled water	100 parts

Of this solution I rarely, under any circumstances, give less than 1½ ounces as a dose, and in the case of a big horse, when occasion demands it, I do not mind giving as much as 2½ ounces. Either an ordinary hypodermic syringe or one of the special intratracheal syringes may be used. The latter is larger, and obviates frequent refilling, and, further, its cannula is curved and directed downwards. The chief points to be observed in the operation are: First, rigorous asepsis, both of the skin and of the needle or trocar and cannula. Neglect of this precaution will produce much swelling of the neck, and consequent serious interference with subsequent injections. In the next place, care must be exercised during the injection. This latter, it must be admitted, is a tedious and patience-trying performance; perhaps nowhere is *festina lente* more thoroughly applicable than here. The instrument is inserted between any two rings fairly high up in the trachea. My method is to have a groom to support the horse's head on his shoulder, while the fluid is being *very* slowly injected.

" The utmost care is necessary to prevent the horse from allowing his larynx to sink below the level of the instrument in the trachea; for, so far from resenting the injection, he usually stands perfectly quiet, and appears to fall asleep under it. As he gets drowsy, the neck becomes more extended, and, the entire weight of the head being on the man's shoulder, the neck soon approaches the horizontal; then it is quite an easy matter for the larynx to sink below the operation-point. Immediately this is allowed to take place, some portion of the fluid, aided by the expired air, gravitates towards the larynx, and a violent fit of coughing occurs. The same thing happens if the injection is carried out too rapidly, and in either case, of course, impedes the operation very considerably, not to mention the possibility of actual injury to the animal.

" Attention to hygiene, nursing, and feeding, is equally as essential in this as in any other form of treatment.

" In conclusion, I would add that I have never known pulmonary gangrene or tracheitis, as mentioned by Friedberger and Fröhner, or other serious ill-effects, to follow this treatment."

The late F. W. Wragg had great success with a combination of iodine and iodide of potassium, 10 grains of the former and 50 grains of the latter being given in the form of bolus twice daily.

W. Robb, F.R.C.V.S., Glasgow, has observed good results from the intravenous injection of a solution of formalin—1 drachm of formalin solution (40 per cent.) to 10 ounces of boiled water. D. Imrie, M.R.C.V.S. Glasgow, has also obtained excellent results from the employment of formalin, given in a similar manner to that adopted in the treatment of pneumonia (see p. 841). He usually finds that one dose is sufficient. Cadéac advises the administration of ergotin with arseniate of strychnine.

Adrenalin has been found useful by some practitioners; it may be administered by intravenous injection, and should be repeated at proper intervals, as its action is transient. We have found excellent results in one instance from this agent, given by hypodermic injection in doses of 2 drachms once daily.

In cases where marked dyspnœa was present Gray has observed that the intravenous injection of a solution of iodide of potassium exerted a beneficial influence, the respirations were reduced to normal in a few minutes after the injection, and remained so for four or five days, or longer if the iodide was repeated.

Imrie finds that a warm solution of iodine and potassium iodide administered intravenously proves as efficient as the formalin solution.

Lignières found that antistreptococcic serum (of the human erysipelas type) has an active influence in cases of purpura, but as in the latter the streptoccocus of strangles is often associated with the *Streptococcus pyogenes*, a polyvalent serum gives the best results. This polyvalent serum can be obtained by mixing antistreptococcic serum and serum prepared from the streptococcus of strangles.

He advises large doses of this serum—viz., 40 to 60 c.c., repeated after twenty to twenty-four hours. It can be given intravenously or by subcutaneous injection.

Marmorek's polyvalent antistreptococcic serum has also been employed by many authorities with good success. According to Cadéac, "it acts on the nervous system and on the phagocytes, overcomes the vaso-dilatory action of the toxins, increases the defensive power of the phagocytes, and assists the system to eliminate the toxins."

The intravenous or subcutaneous injection of normal saline solution is advised by some authorities when evidences of cardiac weakness are present.

In cases where extreme dyspnœa is present, depending on occlusion of the nostrils and nasal passages, or on œdema of the glottis, tracheotomy may become necessary in order to prevent the occurrence of asphyxia. Whenever possible it should be avoided, as the operation wound is likely to become septic, and a further complication is added to the disease. Rigid antiseptic precautions should be employed during the operation, and also in connection with the resulting wound.

In cases where the dyspnœa depends on extensive swelling of the nostrils alone, some authorities advise that a suture be inserted through the alæ, and brought across the middle line of the nose. Jöhne advised the introduction of a light metal tube into each nostril in such cases.

In the local treatment of the cutaneous swellings, some authorities advise the application of astringent lotions, such as solutions of alum, acetate of lead, or acetate of aluminium. Gray has found the application of a liniment composed of 1 part of oil of turpentine to 3 parts of raw linseed oil useful. Others advise inunction of paraffin ointment to lessen tension in the swollen parts.

Scarification of the swellings must be condemned, as it leads to secondary infection and gangrene. When, however, there is extensive œdema of the sheath, which interferes with urination, or in cases of paraphimosis, scarification carried out under strictly aseptic precautions is indicated. When sloughing of the skin and deep fissures are present, the parts should be dressed with antiseptic agents, such as solutions of lysol or cyllin, or solutions of iodine. Solutions of peroxide of hydrogen are very useful in such cases. When discharge ceases, the parts should be dusted over with a combination of iodoform, boric acid, and zinc oxide.

Halters and surcingles should not be worn by animals suffering from purpura, as the slightest pressure on the skin is liable to produce sloughing. During convalescence careful dieting is necessary, and the animal should not be put to work too soon, otherwise a relapse may occur. A course of tonics is generally advisable.

Horses suffering from purpura should be isolated, as, although we have no evidence of direct infection occurring, the microbic nature of the disease renders such a course advisable; besides, it is sometimes found that one case of purpura in a stable may be followed by further cases.

PURPURA HÆMORRHAGICA IN CATTLE.

Purpura in cattle is described by some British authors, but is not mentioned by others. Steel, Gresswell, Armitage, and Woodroffe Hill, describe it as occurring chiefly in calves. We find no mention of it in Williams's "Veterinary Medicine," nor in Moussu and Dollar's "Diseases of Cattle and Sheep." Henry Thompson, M.R.C.V.S., of Aspatria, who has one of the largest cattle practices in England, informs us that he has never seen a case of this disease in cattle.

The disease is described as occurring on the Continent by Friedberger and Fröhner, also by Cadéac and many others. According to Friedberger and Fröhner, it appears to be more rapid in its course and more unfavourable than in horses. Cadéac gives a full description of the disease in cattle, and states that it is chiefly found in working oxen.

In former times it was described under a variety of terms, such as "elephantiasis," "de rouge," "fièvre érythémateuse," or "fièvre angioténique." In Germany it is described under the name of "morbus maculosus," and was regarded by Dieckerhoff as a similar affection to purpura hæmorrhagica in man.

It often assumes an enzoötic character. The symptoms generally resemble those met with in the horse. Lameness is a frequent symptom, and paralysis of either the fore or hind limbs, depending on hæmorrhages affecting the nervous system, is often observed. Skin lesions and loss of hair appear to be specially frequent in the purpura of cattle, and the course of the disease is very prolonged.

The mortality is said by Cadéac to be low, but the long period of convalescence and the tendency to incomplete recoveries render it a serious affection in cattle. In the benign form of the disease the swellings are insignificant.

In the treatment of the affection, bleeding is advised in the early stages only, before the swellings appear; this is believed to act beneficially by enabling the paralysed vessels to recover their contractile power, and thus to prevent effusion (Cadéac).

A purgative dose of sulphate of soda can be substituted for the venesection. In other respects treatment is similar to that advised for the disease in the horse.

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EPIZOÖTIC CEREBRO-SPINAL MENINGITIS IN THE HORSE

BY E. WALLIS HOARE, F.R.C.V.S.

Synonym.—Cerebro-spinal fever (Robertson).

GENERAL REMARKS.

Under the above heading an affection of the nervous system of horses is considered at length by Continental authors.

The disease occurs in the form of an epizoötic or as an enzoötic, and most authorities agree that it is also met with as a sporadic affection. Judging by the literature of the subject, the disease appears to be a well-known and important one in many parts of the Continent and in America, while in Great Britain, although sporadic cases are not uncommon, there are but few records of outbreaks of the epizoötic form of the affection. In fact, at the present day, if outbreaks of the disease do occur in this country, they are not reported; and on inquiry amongst practitioners of wide experience we find that such outbreaks have not come under their observation.

Some years ago we met with a series of cases in the one locality, all presenting symptoms of cerebro-spinal meningitis, and showing after death the lesions described as being present in this affection. Attempts at elucidating the etiology and nature of these cases proved unsuccessful. So far as we could judge, the symptoms and lesions were exactly similar to those occurring in sporadic cases of meningitis which we meet with in ordinary practice.

It seems rational to conclude that at the present time the epizoötic form of the disease occurs very rarely, if at all, in this country.

On consulting veterinary literature, we find that the disease received attention from the following British veterinary authorities :

Gibson,* in 1743, observed a disease occurring in young horses which, from his description, resembled epizoötic cerebro-spinal meningitis.

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* "Treatise on the Diseases of Horses," 1751.

Axe * described an outbreak amongst a stud of cart-horses in Essex in February, 1881, and also referred to an outbreak amongst the horses of the London Street Tramway Company in the same month and year.

Daniels † described the latter outbreak, in which twenty-six horses were affected and five died. Some of the convalescent horses were transferred to another stable, and caused a fresh outbreak within five days.

The disease also occurred in Norfolk in the epizoötic form at the same period. Ellis (Maldon) bled several horses suffering from the disease, and the blood was thrown into the yard adjoining the stables. Several sparrows partook freely of the blood-clot, and after a short period became paralysed in their wings and unable to fly. Next day a sow and a dog consumed the blood; they became paralysed, and had to be destroyed. Several young pigs that were being suckled by the sow in question were seized with convulsive twitching of the voluntary muscles, accompanied by paralysis.

Large ‡ described the disease as it occurred in America; and Liautard studied it in 1869. It has been recognised in America since 1850. Amongst earlier British writers who described the affection, we may mention Litt, § Small of Dundalk, and Storrar of Turriff, who also met with epidemics of spinal paralysis. Percivall also mentions an epidemic form of paralysis.

Robertson, ‖ under the heading of "Cerebro-spinal Fever," gave a full description of the disease, which corresponded in many respects with the clinical histories given by other observers. He regarded it as "partaking of the character of an acute specific disease," and pointed out that there was "an absence of relation between the severity of the symptoms exhibited and the extent of textural change observable after death;" also that the cause of the affection was not understood.

Various Continental authorities have recorded outbreaks of the disease, including Meyer, Aubry, Thiernesse, Mouchot, Genée, and in later years Haase, Jöhne, Siedamgrotzky, and Schlegel.

According to Apostolides, an epidemic of the disease occurred all over Egypt in 1876, resulting in the death of 5,000 horses, 700 mules, and 200 asses in Cairo and its vicinity alone.

During October, 1911, an outbreak of the malady occurred in the region of Gray, the Commune of Var Haute-Sâone, Côte d'Or, Dampierre et Flée, and Montigny-sur-Vingeaune, in France, where it was hitherto

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* *Veterinarian*, 1883.

† *Ibid.*, 1881.

‡ *Ibid.*, 1867-1883.

§ *Ibid.*, 1858.

‖ "Textbook of Equine Medicine," 1890.

unknown. A bacteriological study of the affection revealed the presence of the bacillus of cerebro-spinal meningitis.*

According to Friedberger and Fröhner, the disease known as "Borna sickness" in horses should be differentiated from cerebro-spinal meningitis, as in the former affection there is an absence of lesions in the brain and spinal cord. Jöhne and Ostertag hold a similar opinion. Cadéac, however, states that the practitioners in the cantons of Borna, Merseburg, etc., have established the identity of the two affections. The clinical history of both appears identical, and as lesions of the nervous system may be absent in some cases of cerebro-spinal meningitis—or, at least, have not been discovered—and nothing definite has been ascertained as regards the bacteriology of either affection, it does not seem desirable to consider each disease separately. Cadéac does not differentiate these affections. The disease appears to assume a very virulent form in some years. Thus, in the kingdom of Saxony, in 1896, a total of 1.198 horses were attacked, and only 76 recovered. In Merseburg, in 1896, 60 out of 69 horses died (Friedberger and Fröhner).

Jöst† investigated "Borna disease" (Borna sickness) during an outbreak that occurred in Saxony in 1907, and found that, although macroscopic lesions were absent, a histological examination of the brain and spinal cord revealed typical morbid changes. He termed the disease "an infectious inflammation of the brain and spinal cord of the horse."

As regards the connection between epizoötic cerebro-spinal meningitis of the horse and the disease known as cerebro-spinal fever in man, although there are certain points of resemblance in the symptoms and lesions, most authorities agree that the affections are distinct. In cerebro-spinal fever of man, the specific micro-organism has been discovered—viz., the *Diplococcus intracellularis*—while in the equine affection the bacteriology is as yet undetermined. Again, cutaneous symptoms such as herpes, and a petechial rash, which are so commonly associated with human cerebro-spinal fever, are absent in the equine affection. Fever of an irregular and variable type is generally present in the former disease, while in the latter affection it may be absent in many cases.

Although Laveran observed, during an epidemic of human cerebro-spinal fever at Naples, that several horses were attacked by a similar disease, this has not been the usual experience of those who have studied the subject. According to Cadéac, the micro-organism of human cerebro-spinal fever is not pathogenic to the horse.

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* *Revue Vétérinaire*, No. 11, November, 1911, p. 704.

† Jöst and Dengen, ex. *Berl. Tier. Wochens.*, vol. xxvii.; translation in the *Journal of Comparative Pathology and Therapeutics*, December, 1911.

Heubner induced cerebro-spinal meningitis in two goats by the intradural injection of bouillon cultures of the *Diplococcus intracellularis* (Friedberger and Fröhner).

OCCURRENCE.

Horses are more commonly attacked, but outbreaks are also recorded in cattle, sheep, and goats. The affection is very rarely met with in dogs, only 110 cases being reported from the Berlin Veterinary College Dog Hospital in nine years (Friedberger and Fröhner). Stent recorded an outbreak of the disease in dogs (see p. 898). Young and vigorous horses are especially attacked. Most of the outbreaks occur during the months of June, July, and August; it diminishes in autumn, and disappears in winter (Cadéac).

ETIOLOGY.

Nothing is definitely known with reference to the etiology of the disease.

In common with other affections of the nervous system, various theories have been advanced to account for the phenomena that are observed, but further investigation has demonstrated that causes such as exposure to chill and cold, musty food, polluted water, etc., possess but an imaginary influence in this direction. Some American writers attributed the disease to dietetic causes, such as foods undergoing fermentation, the presence of toxic fungi, hay made from lucerne, worm-eaten corn, parasitic fungi on plants, grain, etc.; but positive evidence is required before we can accept these as etiological factors.

Many authorities believe that the disease is an infectious one, but are unable to determine the nature of the infecting agent.

In "Borna sickness," which some authorities claim to be a distinct disease, Ostertag believes that the infective agent is a special streptococcus (the *Borna streptococcus*). Pure cultures of this micro-organism injected under the dura mater, or repeatedly injected into the blood-stream, are said to induce the disease. If any other means of introducing the agent are attempted, they prove negative. The symptoms are believed to be due to the action on the nerve centres of toxins derived from the micro-organisms. Ostertag found the streptococcus in well-water that was contaminated with drainage from stables in which infected animals were housed.

According to Jöst,* "the brain becomes infected by way of the lymph-stream, and not by the blood. The infection travels from the mucous membrane of the nasal cavity by the lymph vessels accompanying the olfactory nerves, these forming an unobstructed path from the nasal mucous membrane to the pia mater of the olfactory lobes. The virus reaches the pia mater in this way, and causes inflammation, and from this the olfactory lobe becomes affected. The inflammatory processes in the brain tissue extend . . . and sometimes involve the spinal cord, probably by continuous extension through the nervous tissue."

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* Jöst and Dengen, ex. *Berl. Tier. Wochens.*, vol. xxvii.; translation in the *Journal of Comparative Pathology and Therapeutics*, December, 1911.

Siedamgrotzky and Schlegel (1896) discovered in the cerebro-spinal fluid of affected animals micro-organisms in the form of cocci or diplococci, which, on inoculation, proved pathogenic to horses.

Jöhne (1896) isolated from the fluid in the subdural spaces and cerebral ventricles a micro-organism which he named the *Diplococcus intracellularis equi ;* this showed a marked resemblance to the *Diplococcus intracellularis* of human cerebro-spinal fever. Intradural inoculation induced symptoms of cerebro-spinal meningitis in horses.

Jöhne believed that the disease resulted from the action of toxins derived from the diplococci, but he was unable to produce experimentally any inflammatory lesions of the nervous system, the fluid present in the subdural spaces and ventricles being due to passive transudation. According to Cadéac,* "it is still an open question as regards the specific character of the micro-organisms studied in this disease. It is probable that the disease is not due to a specific microbe, but simply to agents whose virulence is increased, so that the epizoötic form can originate from the sporadic, just as contagious pneumonia can originate from the sporadic type of pneumonia. The contagium is spread by infected litter, forage, and water."

MORBID ANATOMY.

In many instances the macroscopic lesions are neither well marked nor constant. The absence of lesions of the nervous system is said by some authors to be characteristic of "Borna sickness," but, as already remarked, Jöst has discovered histological changes in the brain and spinal cord, to which we shall presently draw attention.

Robertson observed that in the cases he met with, the structural changes were not always in direct relation to the duration of the disease or its severity. This is also our experience with reference to sporadic cases of cerebro-spinal meningitis. Marked differences of opinion exist in regard to the lesions that are most commonly present. According to Jöhne and Ostertag, the only lesions observed in "Borna disease" were catarrh of the stomach and of the small intestine, but no evidences of inflammation of the cerebro-spinal meninges were discovered.

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* "Pathologie Interne."

In the enzoötics of the disease that occurred in England, Axe observed a deformity of the normal contour of the brain, a tendency to divergence or spreading of the cerebral hemispheres, subarachnoid effusion, and marked congestion of the pia mater. The effusion in the spinal cord was most marked in the cervical region. In some cases the gastric mucosa showed patches of congestion, and occasionally ecchymoses were observed.

Robertson found congestion of the pia mater and of the entire vessels of the cranium and spinal canal; an abnormal amount of fluid of a turbid character was present in the subarachnoid space; occasionally hæmorrhagic spots were found in the dura mater, and a gelatiniform and feebly organised material between the arachnoid and pia mater. The lesions occurred chiefly in the brain and in the cervical and lumbar regions of the spinal cord. According to Cadéac, the effusion may be of a serous character, or purulent, sero-purulent, fibrino-purulent, or hæmorrhagic; sometimes it is clear and of a yellowish tint. Other authorities state that the lesions are those of a serous cerebro-spinal meningitis. Lesions may be found in other organs, such as ecchymoses on the mucous membranes of the stomach and intestines; pulmonary congestion and broncho-pneumonia have also been observed. Sometimes the blood is found dark-coloured, and various alterations occur in the heart, liver, kidneys, etc.

Jöst,* as the result of his investigations into "Borna sickness," regarded the disease as "an acute, diffuse, infiltrative, non-purulent meningo-encephalitis and myelitis of the lymphocyte type, and chiefly mesodermal (vascular) in character."

The principal seat of the lesion is the brain, acute encephalitis being present; slight meningitis is also found, which is probably a secondary condition. The lesions in the spinal cord are less extensive and not so well marked. The vessels of the brain, and to a less extent those of the spinal cord, usually show a marked inflammatory infiltration of their external coats; the perivascular lymph spaces are partly involved. The substance of the brain and spinal cord show similar infiltrations widely diffused. The infiltrating cells are chiefly lymphocytes—*i.e.*, they are for the most part derived from the vessels.

Jöst described special bodies termed "nuclear inclusions," which can as a rule be demonstrated "in the olfactory convolution and in the large polymorphic ganglion cells of Ammon's horn." These bodies are not found in any other disease, and are said, when present, to be diagnostic of "Borna sickness." But "in individual cases they bear no relationship, either as to size or number or as to localisation, to the inflammatory infiltrations."

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* Jöst and Dengen, ex. *Berl. Tier. Wochens.*, vol. xxvii.; translation in the *Journal of Comparative Pathology and Therapeutics*, December, 1911.

The inflammatory process is most marked in the olfactory bulb and olfactory convolution, and next to these in the caudate nucleus and "Ammon's horn." It is believed that it commences in the olfactory lobe, and then extends to other parts, also that the meningitis occurs subsequently to the encephalitis.

SYMPTOMS.

The symptoms resemble those met with in sporadic cerebro-spinal meningitis. They may appear suddenly, or may be preceded by a staggering gait and inco-ordination of movements. In the former instance the horse falls to the ground and is unable to rise; he struggles in a convulsive manner; the clonic spasms may chiefly affect the anterior regions of the body; the head is constantly being forced backwards; the conjunctiva and sclerotic are deeply injected; dilatation of the pupils, nictitation, and nystagmus, are present; short periods of ease are observed, but without any warning violent convulsive fits occur, so that great care is essential in approaching or handling the animal. Marked hyperæsthesia of the skin is also present, and sweating occurs in patches over the body. Paraplegia results, and is followed by complete paralysis and coma.

In some cases symptoms of delirium and nervous excitement predominate; in others depression and stupor, with coma, are chiefly in evidence. Occasionally the primary symptoms manifested are a staggering gait, soon followed by acute delirium and convulsive fits, during which the horse will rush about the stall, with head extended and membrana nictitans protruded, as in tetanus. After a time he goes down, and struggles violently at intervals. Fever is a variable symptom; it may be absent, or hyperpyrexia may be observed, and some very high temperatures have been recorded. In many instances the fever may be of an intermittent character. Some cases present symptoms of spasmodic colic at the commencement, depending on gastro-intestinal catarrh. In the majority of cases of sporadic cerebro-spinal meningitis on which we have held autopsies, we have found evidences of gastric catarrh or of gastritis; in such instances the cerebral symptoms have masked the evidences of pain which would otherwise have been present.

Local paralysis has been observed in some cases, such as paralysis of the nerves supplying the muscles of the tongue and pharynx, resulting in salivation and inability to swallow ; or paralysis of the nerves supplying the muscles of the lips, eyelids, etc. ; or the pneumogastric nerve may be involved, and roaring is induced.

As sequels to the disease, amaurosis, paralysis of the seventh nerve, and " shivering " have been observed. Pulmonary complications are not infrequent, due in many instances to paralysis of the pharynx, so that saliva and particles of food gain entrance to the bronchi.

Cases of a milder type are met with, in which, after a long period of convalescence, recovery may occur ; these are more often seen at the termination of an outbreak.

COURSE AND PROGNOSIS.

According to Robertson, the percentage of fatal cases was large, but rather irregular, ranging from 10 to 80 per cent. Cadéac gives the mortality as from 76 to 80 per cent., and even up to 90 per cent.

Even amongst those that survive, a large percentage are permanently affected by various sequels. Some cases run a peracute course, death resulting in from ten to twelve hours ; some last for seven to eight days, occasionally from ten to fifteen days. Death results from coma, septicæmia, or from broncho-pneumonia.

DIFFERENTIAL DIAGNOSIS.

The disease can only be differentiated from sporadic cases of cerebrospinal meningitis by its epizoötic character.

It must be remembered that influenza and epizoötic pneumonia may be complicated by meningitis, and a series of cases in which nervous symptoms appear early may occur in the same stable. The history of the outbreak must be taken into consideration in arriving at a differential diagnosis.

PROPHYLAXIS.

This consists in isolation of the affected animals, the proper disposal of contaminated litter, food, etc., and thorough disinfection of the buildings. The water-supply should receive attention, in view of contamination from infected stables.

TREATMENT.

When delirium and violent convulsions are present, treatment is of no avail, and the most economical and humane course is to destroy the animal.

In milder cases a purgative consisting of aloes and calomel should be administered. In order to allay nervous excitement, the best agent is chloral hydrate.

Local applications to the region of the head and spine may be tried, such as ice-bags. Some authorities advise stimulating liniments, or even blisters. During convalescence a course of potassium iodide is indicated.

In the French outbreak of 1911 Lesage adopted sero-therapy, with the object of overcoming or limiting the malady, but up to the present the results are not known.

I. EPIZOÖTIC CEREBRO-SPINAL MENINGITIS IN CATTLE AND SHEEP.

The symptoms resemble those met with in the horse, but the stage of excitement is much shorter, and paralysis sets in early.

In 1910 we observed a number of cases in young cattle that occurred in one district. The symptoms present were a staggering gait, stiffness of the neck and fore-limbs, defective vision, dilated pupils, and prominence of the eyeballs. Later on paralysis set in, in some cases preceded by convulsive movements of the fore- and hind-limbs, and total blindness. Nictitation and nystagmus were also observed. In some instances delirium was present, and the animals ran against surrounding objects and bellowed loudly, but paralysis soon supervened.

In sheep, marked opisthotonos may be observed, also grinding of the teeth and convulsions of the muscles of the extremities. The disease almost invariably proves fatal, and treatment is useless.

Nothing is known with reference to the etiology of the affection.

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JÖST AND DENGEN, ex. Berl. Tier. Wochens., vol. xxvii., No. 25, June 22, 1911, pp. 453, 454; translation in Journal of Comparative Pathology and Therapeutics, December, 1911.

II. EPIZOÖTIC CEREBRO-SPINAL MENINGITIS IN THE DOG.

Stent* described an outbreak of the above disease. Four bitches in the same kennel were attacked, and the symptoms appeared in each in about a week after whelping. At the time of attendance (December, 1907) the malady had been in existence for six weeks in one case, for five weeks in another, and for three weeks in a third case, while the fourth case was in the initial stage, and had whelped two weeks previously.

The symptoms observed were as follows: Stiffness in gait; grunting on being handled; appetite normal; later on the animal was unable to rise, and rapid abdominal breathing was present. In one case the lower half of the ribs appeared in a "twisted" condition; a grunt was emitted with each respiratory movement; the animal could stand if placed on its feet, but seemed afraid to move; when started, it would walk quickly, with very short steps. If the head was turned to one side, the tail would curl to the opposite side. On raising the head suddenly the animal would collapse. Slight congestion of the conjunctivæ was present, but the pupils were normal; the respirations were very rapid and of the "abdominal" type; the lower half of the ribs on each side was drawn in to such an extent that they almost touched each other.

One animal was destroyed, and the autopsy revealed intense inflammation of the cerebral and spinal meninges. The ribs and the spinal column showed a remarkable softened condition of the bony structure. A prominent "knuckle" was found on the lower third of all the ribs. Under treatment with calcium phosphate, followed up with Easton's syrup, also uterine injections and the application of an iodine preparation to the neck, the other cases recovered in about three weeks; but the "knuckle" in the ribs remained.

The kennels were disinfected, and in May, 1908, two bitches whelped therein and remained healthy. But in the following August two bitches whelped in the same kennels and developed similar symptoms to those mentioned. The condition of the ribs, however, was not so well marked. One of these animals had suffered from a previous attack.

The etiology of the disease was not discovered.

Sporadic cases of cerebro-spinal meningitis occur in the dog. In most instances the symptoms develop rather suddenly, and consist of restlessness, excitement, evidences of pain, inability to lie down for any length of time, panting, anxious expression, etc. Later on the head is drawn backwards, the superior vertebral muscles are contracted, and feel hard on palpation ; the back is arched, and when the animal sits on his haunches the hind-limbs are rigidly but passively maintained under the abdomen. When the animal attempts to put weight on the hind-limbs, the feet double backwards from loss of power.

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* Report of Meeting of Lancashire Veterinary Association, *Veterinary Record*, October 3, 1908.

Cerebro-spinal meningitis also occurs as a complication of distemper, and many dogs in the same kennels may manifest similar symptoms (see Canine Distemper, p. 685).

INFECTIOUS PARAPLEGIA.

Under this heading an enzoötic disease of the horse and ass is described by Continental authors. So far as we are aware, the affection, has not been observed in the British Isles. It is characterised by lesions in the urino-genital mucous membranes, by partial or complete paralysis of the hind-limbs, and in some cases by lesions in the spinal cord and spinal meninges.

According to Cadéac, the disease has been observed and studied by Comény in 1888, in an outbreak amongst the horses of a cavalry regiment.

Runcoule in 1892, Blin in 1897, and others, have also recorded observations on the affection. Friedberger and Fröhner also draw attention to the disease, but omit any mention of urino-genital lesions. These authors quote Duhlström, who stated that "infectious paraplegia of horses has been known in Denmark for over fifty years by the name of 'typhus of the spinal cord,' and is there almost universally regarded as identical with hæmoglobinuria (azoturia)." The symptoms mentioned by the German observers certainly resemble those occurring in azoturia, with the exception that the history of the cases was different, no alterations in the appearance of the urine were recorded, and also the disease was of an enzoötic character. Cadéac is of opinion that the urino-genital mucosa is the primary seat of the disease, and the lesions of the spinal cord and its meninges are secondary.

Various micro-organisms have been isolated by different investigators from the spinal cord, kidneys, and urine of affected subjects, but none have proved to be specific. Zschokke (1889) discovered in the intestinal contents, kidneys, lung, liver, and spleen, a cocco-bacillus, resembling in its characters the *Bacillus coli communis;* this micro-organism, when inoculated under the skin of a goat, produced paresis.

Lignières* discovered a special streptococcus in a state of purity and in great abundance in the subarachnoid fluid. He has included this in his group of strangles streptococci.

Up to the present time nothing is definitely known with reference to the nature of the infecting agent.

Infection is believed to occur by means of contaminated litter, urine from affected animals, and grooming utensils. When large numbers of horses are kept together, the disease may spread from one to the other, or the cases may appear in an irregular manner.

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* Lignières, "Contribution à l'Étude de la Paraplégie du Cheval"; *Bulletin de la Société Centrale,* November, 1898; also *Recueil de Médecine Vétérinaire,* 15 August, 1899.

Mares are more commonly attacked than horses, the reason suggested for this being that the urino-genital mucous membrane is more exposed to infection. The lesions are found in the vaginal mucous membrane, in the bladder, the ureters, the kidneys, the spinal cord, and its meninges.

The vulva is swollen, and shows ecchymoses on its mucous membrane, and an œdematous swelling occurs on the perineum, internal surface of the thighs, and on the udder; the vaginal mucous membrane is thickened, infiltrated, and covered with a mucous exudate containing clots of blood.

The vesical mucosa is deeply congested and covered with a thick mucous coating, studded with ecchymoses, and all the coats of the organ are thickened and show ecchymoses.

The spinal cord shows inflammatory lesions of the meninges in the lumbar region; in some cases the entire cord is thus affected, and also the cerebrum (Cadéac).

The symptoms presented are inco-ordination of the movements of the hind-limbs. When the horse moves, the legs cross each other; in severe cases he goes down, and cannot rise without assistance; and paraplegia occurs.

In the horse the sheath is swollen and the penis hangs in a flaccid condition; in the mare the lips of the vulva are swollen and micturition is frequent, small amounts of viscid urine being passed at a time; the temperature is subnormal. Recovery may occur after a long period of convalescence, or complete paralysis may result. In the 108 cases recorded by Comény, 37 deaths occurred. The differential diagnosis from dourine is based on the absence of the special trypanosome of this latter disease.

Treatment does not appear to be successful in severe cases. The use of slings is advised when the animal can place some weight on the hind-limbs, also irrigation of the bladder and vagina with solutions of boric acid.

Prophylactic measures consist in isolation of affected animals, disinfection of buildings, proper disposal of contaminated litter, disinfection of grooming utensils, clothing, etc.

MEDITERRANEAN FEVER

By T. CHIVERS GRAVES, M.B., B.S.Lond., B.Sc.Lond., M.R.C.S.Eng., L.R.C.P.Lond., M.R C.V.S.

Synonyms.—In human medicine the disease has a varied nomenclature. Thus, indicating its one-time geographical recognition, it has been variously named Malta fever, Danubian fever, Neapolitan fever, and Rock fever; whilst, indicating its clinical course in man, it has been described as Typho-malarial fever, Intermittent typhoid fever, Bilious remittent fever, and Undulant fever.

French : Fièvre de Malte.

Definition.—A disease due to infection by the *Micrococcus melitensis* (Bruce), the constitutional reaction of the host depending on the susceptibility of the species attacked.

SHORT HISTORY.

The disease has for a long time been recognised as a distinct entity in the human subject, in whom it produces a continued undulatory pyrexia, with arthritic pains, enlargement and tenderness of the spleen, profuse diaphoresis, and small mortality, but has been confounded with enteric fever, acute rheumatism, and malaria.

In 1887 Colonel Bruce demonstrated the causal factor to be a specific organism, which he named the *Micrococcus melitensis*.

In 1904, at the request of the Colonial Office, Admiralty, and War Office, the Royal Society appointed a Committee to investigate the disease as it was met with in Malta; and it is principally from the researches of these investigators that our knowledge of the conditions under which the disease is propagated in both animals and man is chiefly drawn.

That the lower animals react to the organism experimentally was shown. In 1905 Zammit and Horrocks found that the sera obtained from apparently healthy Maltese goats agglutinated emulsions of the micrococcus, and that the specific organism could be recovered from the milk and urine of these reacting animals.

Dubois investigated the disease in France, and recorded his observations in the *Revue Vétérinaire*, August and September, 1910, and in March, April, and May, 1911; a translation of his last article on the subject appeared in the *Journal of Comparative Pathology and Therapeutics*, vol. xxiv., 1911.

GEOGRAPHICAL DISTRIBUTION.

The disease has a world-wide distribution. First definitely recognised on the shores of the Mediterranean and Red Sea, Gibraltar, Malta, Sicily, Naples, on the banks of the Danube, and in France, it has since been reported as occurring in the Soudan, round the shores of Lake Albert Edward, in the Orange River Colony, North-West India, China, Fiji Islands, North America, West Indies, and South America.

In France an epizoötic of the disease occurred amongst goats in the suburbs of Paris in 1908, and at St. Martial in 1909, and was investigated by several authorities. Researches carried out in 1910 showed that the disease was prevalent in the Department of Gard, fowl, ducks, sheep, dogs, and horses being attacked, while over 400 cases were observed in man in less than a year (Dubois).

ANIMALS AFFECTED.

Naturally.

GOATS.—Amongst the lower animals the goat is chiefly attacked, and seems to act as a natural reservoir of infection.

COWS.—Shaw, examining cows in Malta, found that out of thirty-three cows, ten were infected, and living micrococci were recovered from the milk of two of these.

DOGS.—According to Kennedy, the dog may become infected with Mediterranean fever. Out of 114 dogs examined in Malta, nine showed unmistakable signs of infection; whilst from one dog, whose serum reaction was very definite, the organism was recovered from the mesenteric glands on post-mortem examination. Dubois records cases occurring in cats.

MULES.—Kennedy also found that out of eighty-seven mules examined by him in Malta, about 45 per cent. gave a serum reaction to the micrococcus. He therefore deduces that "mules are exposed to, and suffer in a mild way from, infection by the *Micrococcus melitensis*." According to Dubois, horses may be attacked occasionally.

SHEEP, RABBITS, AND FOWLS.—According to Dubois, sheep are as susceptible as goats to the disease in France. Aubert, Cantaloupe, and Thibault, investigating an outbreak of Mediterranean fever in the department of Gard, in the Cevennes, found amongst the domestic animals giving positive serum-agglutinating reactions that, in addition to goats, sheep and rabbits seemed capable of acquiring the disease by natural infection.

According to Dubois,* fowls can be attacked by the disease, and he describes an epizoötic that occurred in the suburbs of Nîmes. Diagnosis was based on the agglutination test, which gave a positive reaction in 60 per cent. of the cases. Ducks are less susceptible.

Experimentally.

Larger Animals.—The goat and monkey have been shown to be easily infected.

Smaller Animals.—Rabbits and guinea-pigs are less susceptible, but by a series of intracerebral inoculations the virulence of the organism can be exalted for both these animals.

ETIOLOGY.

Causal Factor.—The *M. melitensis* (Bruce).

Channels of Infection.

Subcutaneous inoculation of the organism produces a rapid effect. *Ingestion* of infected material is also a sure mode of infection. The passage of the organism through an unbroken mucous membrane is comparatively slow, and under these conditions the wave of fever appears to be prolonged, but when absorbed through an abraded mucosa or skin the reaction is very definite.

That the organism can invade the body from the alimentary tract and upper air-passages by the ingestion of infected material, whether as food, or dried dust inhaled into the nose, has been definitely established.

By means of infected milk the disease can be transmitted from goat to kid; but whether the infection so acquired will persist until adult age is not yet determined.

Horrocks and Kennedy found that agglutinins are transferred *in utero* from mother to kid, as the blood reaction of the kid corresponds exactly with that of the mother on the day the kid is born, but apparently the organism does not enter the fœtal circulation.

The question as to whether Mediterranean fever can be disseminated by the bites of insects, such as mosquitoes, etc., is still in some doubt. It is probable that man may become infected by that means. Zammit states that he infected a monkey by allowing mosquitoes (*Stegomyia*

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* *Revue Vétérinaire*, August, 1910.

fasciata) that had previously fed on a Mediterranean fever patient to bite it.

In experiments with monkeys it has been shown that healthy monkeys may become infected by urine secreted by monkeys suffering from Mediterranean fever. It seems probable that healthy monkeys walking in the infected secretion convey the specific organism into the mouth by means of the paws.

The manner in which animals naturally become infected is a matter of considerable importance to the hygienist.

In 1905 Staff-Surgeon E. A. Shaw wrote : " Goats may be seen any day in the streets of the chief city of the island of Malta feeding on filth and rubbish of every possible variety, some of it visibly saturated with urine, animal and human. Among the lower-class Maltese, workmen have been found who void living *M. melitensis* in the urine, as do a certain number of the infected goats. Thus the path of this manner of infection becomes clear.

" Having satisfied their hunger in the above manner, the goats lie down in the streets to digest their meal, with their teats and udders often in contact with the ordure of the gutters and roads, till they are kicked up by the goatherd to be milked into the vessels brought to the doors of the adjacent houses by their occupants.

" With regard to cows the evidence is not quite so clear. Kept shut up in ' shippens,' and seldom allowed outside, they have their food brought to them ; but as this food is composed of vegetable and other refuse collected from every possible source and situation, it is easy to understand that they can hardly escape from receiving infected food from time to time."

According to Dubois, infection from mother to offspring, by the ingestion of contaminated milk is rarely observed in animals. Kids born of infected mothers do not harbour the *M. melitensis*, and do not usually contract the disease from drinking the mother's milk. On the other hand, kids born of healthy mothers can become infected by drinking the milk of infected goats, and can in their turn carry the infection to healthy animals by sucking them. Infection between goats is chiefly brought about by contact with infected urine or infected milk.

The milker may convey the micrococci from the udder of an infected goat to the surface of the udder and teats of healthy animals. Contamination by means of urine seems to be the most frequent mode of infection. The litter is contaminated by the urine of infected animals, and also by the urine of infected human beings ; the latter, even being apparently cured of the disease, may still eliminate the micrococci in their urine.

Contaminated litter spreads the infection by contact with the skin of a healthy animal, by animals ingesting it, or by infection through the genital passage when the animals are lying down.

Infection by copulation is very frequent, and the male goat constitutes an important agent of transmission. Dubois found that 65 per cent. of male goats were affected, while only 16 per cent. of she-goats gave evidences of the disease by the agglutination test. Once the male goat becomes infected, he contaminates all the she-goats that he serves.

BACTERIOLOGY.

Morphological Characters.

The organism is rounded or slightly oval in shape, about 0·3 μ in diameter. Occurs singly or in diplococcal grouping.

In broth cultures, after about ten days' incubation, the organism may be found arranged in short chains composed of from four to fourteen elements; but these chains are easily broken up, so that it is somewhat difficult to make permanent specimens of this grouping. The fact that the individual cocci are so very loosely bound together facilitates emulsification for agglutination purposes.

In hanging-drop cultures the organism shows decided movement, generally accepted as being only very active Brownian movement, for no translation of an individual coccus from portion to portion of a field occurs. Nevertheless, it has been held that the organism possesses a true motility—in fact, Gordon claims to have demonstrated the existence of flagella, but this statement has failed to receive support at the hands of other observers.

A bacillary form in pure cultures of *M. melitensis* has been noticed by some workers, but, according to Shaw, this variety is less virulent than the coccal form.

The organism, easily stained by the ordinary aniline dyes, is Gram negative.

Cultural Characters.

Cultures are best obtained under aërobic conditions; nevertheless, some growth can be obtained in an anaërobic atmosphere.

The optimum temperature is between 37° to 38° C., but an extremely slow growth occurs at 22° C. Growth is inhibited by exposure to 42° C.

It has been found that the quickest and most copious growth is obtained with media of alkalinity +10 (Eyre's scale).

Sugars are not fermented, indol is not produced, nor is nitrate reduced by this organism.

Whereas in glucose agar media (tinted with litmus) the Gram-positive streptococci produce colonies varying in tint from a rose-red to bright red, the *M. melitensis* colonies are always blue, and after a few days' incubation the colour deepens in tint. This cultural character is useful in separating organisms which occur in urine and milk.

The addition of malachite green, carbolic acid, or sodium taurocholate, to culture media used for growing the *M. melitensis* has an inhibitory effect on the growth of this, as well as other organisms which may be associated with it, in urine, milk, etc.

In broth the organism causes the formation of a diffuse cloudiness without pellicle formation; after a few days a whitish deposit is obtained at the sides and bottom of the tube, leaving the rest of the medium somewhat cleared.

On agar slope, growth appears on the fourth to fifth day, composed of minute circular colonies. With continued incubation the growth assumes a transparent amber colour; later still the colonies, increasing slowly in size, develop an opacity in from four to five days after their first appearance. No liquefaction occurs.

In agar stab a diffuse growth is obtained.

Gelatine stab and slope: Growth is very slow, but no liquefaction of the medium is produced.

On potato a moist transparent film is obtained.

Litmus milk becomes alkaline without clotting at the end of three weeks.

Biological Characters.

Vitality.—Thermal death-point: The upper limit is about 55° C. and the lower about 15° C.

In pure culture the organism maintains its vitality for over three months; in fact, Shaw found that after nine months a dried-up agar culture was still alive and capable of multiplication, and after five months in a broth culture.

In sterilised earth Horrocks found the organism still living at the end of sixty-nine days, and after eighty days on dry fabrics.

Its vitality is, however, lessened by exposure to water and moist earth; nevertheless, the organism has been recovered alive after nine days' exposure to diluted healthy urine, and after seventy-nine days' exposure to diluted Mediterranean fever urine.

Agglutination.—For the diagnosis of the disease, whether in animals or man, the agglutination reaction is most valuable.

The phenomenon may be demonstrated by either the microscopic or the sedimentation methods.

The agglutinin, formed by the host as a reaction to the presence of the organism, has been demonstrated to exist in the blood-serum, milk, and urine of animals infected by the micrococcus.

The fact that the milk of infected goats causes agglutination of the *M. melitensis* was first shown by Zammit, and the reaction is called "Zammit's test."

The following description of the reaction with milk is taken from Dr. Zammit's report:

"The test can be applied on a slide in the ordinary way or in capillary pipettes, as in the method of precipitation. In time, however, the precipitation method was adopted, as being more conclusive and easier to work, especially when a great number of samples had to be dealt with. The only precaution to be taken in applying the test is the addition of an antiseptic, strong enough to prevent the clotting of the milk, but without affecting in any way the agglutinins.

"For the examination of a large number of samples the following method was found to answer best:

"A strong emulsion of the *M. melitensis* is prepared in normal saline solution in a watch-glass. To this a small quantity of formaldehyde solution is added (one small loopful of a 1 per cent. solution), the whole being drawn into a pipette.

"One drop of the emulsion is placed on a glass slide, and a loopful of milk is mixed thoroughly into it.

"This mixture is then drawn up into a fine capillary pipette, left in an upright position for twelve hours, and the reaction noted at the end of that time.

"The reaction is often seen after a few minutes. The cream collects at the surface, and does not interfere with the reaction.

"Between July 10 and September 22, 1905, 710 samples of milk were examined, and a positive reaction was obtained 133 times.

"With a view to checking the value of this method, all the milks that showed a positive reaction were tested a second time. Further, where possible, blood was obtained from the animal for the serum test, and the suspected milk was plated out.

"The serum test constantly confirmed the milk test, and when a strong reaction was obtained, the specific organism was always recovered from the milk.

"In conclusion, in my opinion, the milk test is a safe one, and quite as reliable as the serum test. For sanitary purposes, more especially where a great number of goats have to be examined, the milk test is at once convenient and efficient."

Horrocks and Kennedy, however, working with Zammit's test, found the sedimentation in tubes method unreliable, as the sediment often consisted of fat and débris, and always required to be submitted to microscopical examination. They accordingly adopted the agglutination under the microscope method, which they describe as follows:

"Equal parts of milk and emulsion were placed on a slide and allowed to stand for twelve hours in a moist chamber. At the end of this time the fatty part of the milk had collected in the centre and the surface of the drop, leaving the edges and the bottom clear.

"The clear part was then examined under a ⅛-inch lens, for clumping of *M. melitensis.* It should be noted that the milk was prevented from turning sour by adding 1 drop of 40 per cent. formalin to 10 c.c. of milk."

With regard to urine, Shaw has shown that out of thirty samples of urine obtained from Mediterranean fever (human) cases, twenty-three effected agglutination of the micrococcus.

By successive subcutaneous inoculation of the micrococcus into goats, during a period extending over a few months, the intensity of the agglutinins present in the blood-serum can be raised so that the reaction can be obtained with dilutions as high as 1 in 5,000.

When employing the test by the microscopic method for naturally occurring cases, a forty-eight hours' broth culture is used. Dilutions varying from 1 to 30 to 1 in 100 are employed, as well as controls with normal serum, because old laboratory strains of the organism may sometimes agglutinate with normal serum in dilutions of 1 in 20 or 30.

Relation of the Agglutination Reactions to Each Other and to the Excretion of the Organism.—Goats may have a marked blood reaction (1 in 100) and yet never excrete the *M. melitensis* in the milk; but if neither the blood-serum nor the milk agglutinate the micrococcus, the specific organism is not present in the milk.

There is no constant relation between the amount of agglutinins in the milk or blood and the excretion of the micrococcus in the milk, but the higher the dilution of the serum which agglutinates the organism, the greater is the probability of the milk being infected.

A blood reaction may exist for some weeks before the micrococcus is excreted in the milk.

The milk agglutination test is a surer indication of the micrococcus being excreted in the milk than is the serum reaction.

MORBID ANATOMY.

Goats.—In a typical case of naturally acquired infection no lesions whatever may be found on post-mortem examination.

Many goats which during life have given definite agglutination reactions have been passed as carcasses fit for human consumption by competent inspectors at the abattoirs.

Further, animals in perfect condition, very fat, and with all the organs perfectly healthy, can, at post-mortem examination, be demonstrated to have the organism present in many organs of the body.

Dubois has observed in some cases that have succumbed from the disease, congestion and degeneration of the liver, hypertrophy of the spleen, and swelling of the abdominal lymphatic glands, without any alterations in the blood. He, however, states that at autopsies on goats destroyed during the course of the disease no lesions were discovered, although the *M. melitensis* was isolated from the spleen. Moreover, these animals during life appeared to be in perfect health.

Monkeys.—As a result of experimental inoculation of monkeys with the *M. melitensis*, a well-marked febrile reaction occurs, yet monkeys so infected may, on post-mortem examination, be found to have no abnormalities discernible in any of their organs, although on taking cultures from the spleen, kidneys, lymphatic glands, etc., of such cases well-marked growths of the organism can be obtained. On the other hand, the body may be well nourished, but some of the lymphatic glands may be enlarged, the spleen enlarged and softened, and the liver and kidneys may be congested. Similarly, from these organs the micrococcus may be recovered.

DISTRIBUTION OF THE ORGANISM IN THE BODY AND CHANNELS OF EXCRETION.

This disease is of the septicæmic type, the causal organism invading and multiplying in the blood-stream.

In certain phases of the disease the specific organism circulates freely in the blood, and it can then be easily recovered therefrom. According to Dubois, the micrococci are found in the blood and the spleen during the first stages of the disease, then they become localised in the spleen, lymphatic glands, and kidneys. Their persistence in the spleen explains the possibility of their appearance in the blood from time to time. They disappear after an indefinite period, and only remain in the udder.

Shaw demonstrated that living micrococci may persist for seven months in the blood of a goat experimentally infected. It by no means follows, however, that in any given naturally occurring case the organism can be demonstrated by a blood culture.

Owing to the fact that the coccus is Gram negative, it is not possible to satisfactorily demonstrate its existence in sections of infected tissue. Recourse must therefore be had to culture methods to demonstrate its presence in infected organs. By this method the organism may be demonstrable in the spleen, kidneys, and lymphatic glands.

Dr. Zammit states : " Our goats having been bought already infected, we could not ascertain how long they had been in that condition, but it is a fact that after fifteen months the specific organism was still living in the lymphatic glands." Dubois isolated it from the spleen twelve to fifteen months after the commencement of the disease.

When monkeys become infected by feeding with milk, the lymphatic glands always contain far more colonies of the *M. melitensis* than the spleen. This fact suggests that the specific micrococci contained in the food are carried to the lymphatic glands, and there undergo considerable multiplication. It has not yet been proved that the mesenteric glands are always infected at an earlier date than the femoral and axillary glands, but this may be the case at times.

Milk.—This is one of the principal modes by means of which the organism leaves the body, the importance of which may be gathered from the fact that the deposit from 1 c.c. of infected milk, when plated out, may yield, after four days' incubation, colonies so numerous that an accurate count is rendered impossible.

The excretion of the micrococcus may be intermittent, appearing for a few days, and then disappearing for a week or more.

That the organism so excreted is a virulent form is shown experimentally by the fact that monkeys and goats can be infected by feeding with cultures of the micrococcus isolated from milk, and also by feeding with infected milk itself.

Urine.—As regards excretion by the urine, Horrocks, in 1905, wrote: "The *M. melitensis* is also excreted in the urine of goats suffering from Mediterranean fever, but up to the present it has only been found when the disease has existed for some time and physical changes have occurred in the milk."

According to Zammit, the micrococcus in infected goats tends to disappear from the system after a time, but the process is slow.

Dubois states that elimination of the micrococci by the urine is not a permanent phenomenon. They appear at more or less frequent intervals, varying according to the cases examined. In some instances they may be absent from the urine, but present in the milk.

PERIOD OF INCUBATION.

In feeding experiments the incubation period appears to vary between three and four weeks.

Following subcutaneous inoculation the temperature rises during the next day.

SYMPTOMS.

Goats.—As already indicated, a goat infected with the micrococcus may show no clinical signs which would lead to any definite diagnosis.

In this connection the observations of Horrocks and Kennedy may be quoted:

"It was expected that those goats whose blood reacted would have some symptoms of illness, but this was not apparent, except in a few instances.

"A few goats were noticed to have an unusual degree of lassitude and to be off their food. In the later stages of the infection, when the milk was beginning to dry up, a short hacking cough was noticed, and the goats appeared to steadily lose flesh; the coat also became thin.

"The quantity and quality of the milk seemed in most cases to be unaffected—indeed, it was remarked how often the best milkers in the herd were picked out as a result of the blood examination.

"The excretion of the specific organism may continue steadily for three months without any change occurring in the physical character or chemical composition of the milk, and without the animal exhibiting any signs of ill-health.

"A good average milk production is from 4 to 5 pints a day. When the blood reaction is very marked, there may be a diminution in the quantity, although the physical characters and chemical composition of the milk remain unchanged."

Horrocks examined a goat with a serum reaction of 1 in 100, and found that the deposit from 1 c.c. of its milk yielded an immense number of colonies on plating; yet this animal was valued at £5, whereas the ordinary price for a goat in milk varies from £3 to £4.

A goat similarly affected was in good health and its udder full of milk; this secretion yielded, on chemical analysis, the following results:

Specific gravity at 15° C.	1031
Total solids	14·0 per cent.
Fat	3·6 „
Ash	0·73 „

Later on in the affection, when the physical characters of the milk undergo alteration, the secretion may become thin and serous, or in some cases practically cease, and be replaced by a thick, ropy, brownish or gelatinous material; under these circumstances the organism may still be present in enormous numbers. The disease may, of course, be complicated by other localised affections, such as a suppurative mastitis producing pus in the milk, and on bacteriological examination rendering the detection of the *M. melitensis* difficult on account of the immense number of staphylococci present.

A febrile condition in the natural cases has not been observed. In the experimental cases the subcutaneous injection of the organism causes a pyrexia, varying from 2° to 5° F., persisting for a few days; subsequently the temperature remains normal. The serum, however, will agglutinate in varying high dilutions. The temporary rise of temperature suggests a sort of racial tolerance of this infection on the part of the Maltese goat.

Pregnancy goes on uninterruptedly in infected goats, according to some observers.

According to Dubois, however, the most striking symptom in the disease is the occurrence of abortion in the pregnant she-goats; when the affection occurs for the first time in a flock, 50 to 90 per cent. of the animals may abort. On again becoming pregnant, abortion is still observed but to a less degree, and it may even cease altogether in an infected flock. The specific micro-organism has been isolated from the milk of a goat which had recently given birth to kids, and the same animal was known to have aborted eight months previously. Abortion may occur in infected females of all ages and at all periods of gestation, but the usual period is at the fourth month. When it occurred at a short period prior to full term, the fœtus was generally born dead, or succumbed shortly after birth. . Sterility is rarely observed.

In the cases that abort certain sequels have been observed, such as lameness, mastitis, and ocular symptoms.

The lameness may affect a fore-limb or a hind-limb, or two limbs at the same time. In some cases arthritis and synovitis have been observed, but generally the seat of the lameness is difficult to discover, and no apparent lesions are present.

The mastitis is characterised by the formation of nodules the size of a nut in the mammary tissue, and by alterations in the character of the milk. The attack is generally a mild one, and terminates by resolution without treatment; in some cases the supply of milk is diminished.

In one case ocular troubles, consisting of conjunctivitis and intense keratitis affecting both eyes, followed an abortion complicated with genital infection, but only lasted for a short time.

In the male goat lameness, with similar characters to those mentioned above, is observed, and in rare cases orchitis.

In sheep the symptoms are similar to those observed in goats, abortion being also of frequent occurrence in infected animals of this species.

In other species no special symptoms are observed.*

MONKEYS.—Following experimental infection by feeding, monkeys sometimes suffer from a typical wave of fever and lose flesh; at other times they show no obvious signs of ill-health, and may even gain in weight.

Dubois points out that, although the mortality in goats and sheep is scarcely 3 to 4 per cent., still, from an economical point of view, the occurrence of abortion causes serious losses to owners of these animals.

DIFFERENTIAL DIAGNOSIS.

By the use of simple clinical methods it is manifestly difficult to arrive at a diagnosis of naturally occurring Mediterranean fever in the goat.

The occurrence of widespread abortion and of lameness may suggest the existence of the affection, but they are not diagnostic symptoms.

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* Dubois, "La Fièvre de Malte chez les Animaux Domestiques," *Revue Vétérinaire*, March, 1911.

PROGNOSIS.

The prognosis, as regards life, of a naturally infected goat need not be considered, because by the time that a given goat is implicated as being affected by the disease any acute manifestations resulting from the infection will have subsided, and the animal will, in all probability, be in apparent good health.

The principal point at issue, therefore, in regard to prognosis is the determination of the period during which there is the possibility of the affected animal continuing to harbour and excrete the causal organism.

It has been shown by Horrocks and Kennedy that the excretion in the milk is generally a late phenomenon, only appearing about seventy-four days after the blood reaction has developed.

It is definitely established that the excretion by means of the milk can continue for at least three months without any physical change occurring in the milk; and, as already indicated, the organism may still be present in abundance when the physical characters have altered. Recourse must therefore be had to the bacteriological examination of blood, milk, and urine from suspected animals, together with the employment of the agglutination reaction with the blood-serum or milk. The presence of the organism in the milk is an important point to establish, but one examination of the milk is not sufficient, because the excretion of the micrococcus by this channel may be intermittent. Hence it is better, in examining a herd, to investigate first by means of the blood or milk agglutination tests. Reactors to these tests can then be further examined bacteriologically.

As illustrative of the methods employed in establishing the presence of the disease in animals the following recent investigations may be quoted:

Bruce has recently shown that "muhinyo," the native name for a disease prevailing in parts of Uganda, is nothing more nor less than Malta fever—testing the agglutination reactions of fifty cases in man, and obtaining positive reactions, together with the recovery of the micrococcus from their bodies. Goats were subsequently tried, with positive results, the organism also being recovered where the agglutination reactions were successful.

Aubert, Cantaloupe, and Thibault, investigating an epidemic in man in the Cevennes, found that it was preceded by an epizoötic amongst goats, also directly associated with *M. melitensis*. The inhabitants of the affected area consumed a large quantity of goat's milk, and hence the question of conveyance arose. Investigations amongst the domestic animals in the affected area, by the use of the serum agglutination reaction, yielded the following results:

213 goats (67 infected)	31·9 per cent.
74 sheep (7 infected)	1·9 ,,
96 rabbits (20 infected)	20·8 ,,

With regard to sheep, they state that ewe's milk is hardly ever consumed now as human food, but when not used for feeding young lambs, it is turned into cheese. Whether the *M. melitensis* may be present and retain its vitality in such cheese is a point which is of some importance.

They think that the frequency of infection amongst the rabbits investigated is a matter of some significance; if these animals excrete the *M. melitensis* in their urine, they may prove to be a dangerous means of spreading the disease.

They appear to have no doubt as to the reliability of the serum test (1 in 20 dilution) on rabbits, since they investigated a large number of control rabbits at Marseilles, and found that none of them gave a positive result, even with a dilution of 1 in 10.*

TREATMENT.

No treatment up to the present has proved successful, so that our attention must be directed to prophylaxis so far as the domestic animals are concerned.

Measures in this direction must follow ordinary hygienic lines, indicated by the fact that animals may become infected by food and litter contaminated by excreta from affected animals, by dust, and possibly also by mosquito transmission. Attempts at immunisation have given negative results.

Perambulation of healthy animals in an infected district should be prohibited. Attention to cases of abortion occurring in infected animals is also necessary.

As there is a strong probability that infection is also carried from infected human beings to goats by mosquitoes, the keeping of goats in houses, and in small yards attached to the houses, should be forbidden. Hence goats should be kept in pens as far away from human habitations as circumstances will allow.

Animals shown to be excreting the micrococcus must be regarded as dangerous to the community, and should be slaughtered, and their carcasses condemned as unfit for human consumption. Similar remarks apply to sheep suffering from the disease. As cows may possibly act as carriers of infection, they should also receive attention.

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* *British Medical Journal*, 1910, p. 1545.

Attention should be directed to male goats kept for stud purposes, and frequent examinations should be made and the agglutination test applied.

TRANSMISSION TO MAN.

The part that animals, and especially the goat, play in the transmission of Mediterranean fever to man is that of a "carrier." Acting as a natural reservoir of infection, goats can undoubtedly be the means of disseminating the organism in a virulent form by means of their infected milk and urine. Infected milk drunk raw is a certain means of acquiring the disease. Dust contaminated with infected urine may play some part in the transmission of the disease to man.

Culex pipiens and *Stegomyia fasciata* act as carriers of the micrococcus, and it is extremely probable that human beings are infected by bites of infected mosquitoes.

An interesting fact pointed out by Horrocks is that the disease has practically disappeared from Gibraltar since the practice of importing goats from Malta has been stopped.

Eyre* also pointed out that, as a result of the adoption by the Maltese naval and military authorities, of the recommendation of the Mediterranean Fever Commission—viz., that the use of milk from Maltese goats should be prohibited—there has been a striking diminution in the case incidence of the disease amongst the naval and military forces stationed in Malta. This diminution during the years 1907-1910 is in strong contrast to the incidence of the disease amongst the civil population, to whom the prohibition order did not apply.

It is possible that the organism may be conveyed by means of cheese made from infected milk.

Pasteurisation (68° C. for ten minutes) destroys the *M. melitensis* in infected goat's milk.

Dubois† draws attention to a series of cases where human beings have contracted the disease by contact with infected goats and sheep, and also from the ingestion of infected sheep's milk and of fresh cheese made from this milk.

Man can also become infected by milking animals suffering from the disease, from giving assistance at cases of difficult parturition, and from opening and handling affected carcasses and the organs thereof.

As possible causes, contact with liquid manure and fæces of infected animals, and shearing of affected sheep, may be mentioned.

Bacteriologists and their assistants have become infected while carrying out laboratory experiments with the *M. melitensis*, and some fatal results are recorded.

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* *Lancet*, January 13, 1912.

† *Revue Vétérinaire*, September, 1910.

RINDERPEST

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Synonyms.—Cattle plague.

French : La peste bovine; Typhus du gros bétail; Typhus contagieux.
German : Viehseuche; Viehpesthe.
Italian : Tifo Bovino.
Polish : Dzuma.
Russian : Tchouma reina.
Chinese : Lowpeng.
Punjaubi : Murri.
Hindustani : Mata.

Definition.—An acute contagious disease of cattle, sheep, goats, camels, and the wild ruminants, and also at times attacking swine. It is characterised by its short period of incubation, sudden appearance of high temperature, and the presence of lesions throughout the alimentary canal, and by the high rate of mortality.

HISTORY AND GEOGRAPHICAL DISTRIBUTION.

Rinderpest was brought from China by the Huns to the basin of the Caspian Sea; it was carried by the Huns and others to Western Europe. In the ninth century rinderpest spread to several European countries which were the scene of the wars during the reign of Charlemagne. Similarly, in the thirteenth century it was spread throughout Europe by the Mongols. In the eighteenth century it caused enormous losses throughout the whole of Europe, and in 1744 is said to have been first introduced into England by the importation of animals from Europe.

France suffered enormous losses during the latter part of the eighteenth and the beginning of the nineteenth century, and about this time every country of Europe, with, it is said, the exception of Sweden and Spain (Friedberger and Fröhner), suffered from the ravages of rinderpest. In 1841 it was introduced into Egypt by the importation of animals from

Austria, and in the year 1879 great loss occurred in Germany and Russia. In 1872 an outbreak occurred, but was quickly stamped out, after causing great loss.

It was imported into Abyssinia in 1890 by cattle supplied for the Italian Army, and in 1898-99 it was carried to the Philippines by the cattle sent from Asia for the supply of the American Army (Law). The disease appeared in South Africa in 1881, and again in 1896, 1897, and 1901, where large numbers of cattle were lost.

In India the disease is endemic, and many thousands of cattle die annually. It has never appeared in America, Australia, or New Zealand.

In 1865 an extensive outbreak of the disease occurred in Great Britain, which originated in a cargo of cattle sent from Revel to Hull. It was stamped out after eighteen months, but caused enormous losses. An outbreak occurred in 1872, due to cattle imported from Russia, but it was quickly stamped out. The last outbreak of the disease in Great Britian occurred in 1877.

SUSCEPTIBILITY.

The cattle of the steppes of Southern Russia, Hungary, and Roumania take rinderpest in a mild form, and often recover in about eight to ten days. They are believed to possess to some extent hereditary immunity. Cattle imported from other countries contract a virulent form of the disease, and usually succumb.

Sheep, goats, buffaloes, deer, camels, and swine, are susceptible. A large outbreak occurred amongst the wild pigs in Ceylon in 1909, and may have been the means of spreading the disease.* Nencki states that buffaloes are less susceptible than cattle. Tartakowsky states that camels are only very slightly susceptible and very seldom die of the disease. English, Australian, American, Himalayan, and South African cattle are highly susceptible. Man, cats, dogs, rats, chickens, birds, and horses, are immune.

ETIOLOGY.

Although many workers have investigated the cause of rinderpest, they have not succeeded in isolating any organism. Semmer mentions spore-like bodies in the cell nuclei. Metschnikoff obtained a short bacillus from the blood of rinderpest animals and from the ulcers in the alimentary tract, and other investigators have described various bacilli; but up to the present time no organism has been recognised as the causal agent of rinderpest. The disease is caused by an ultravisible organism which, as the result of filtration experiments, has been shown to pass through the pores of the finer porcelain filters. The contagium is contained in the excretions and secretions of the body, in the red corpuscles of the blood, in the peritoneal fluid, but not in the bile or blood serum; but blood-stained bile or serum containing the slightest trace of red colour is virulent. The disease can be spread by direct and indirect contact (manure, attendants, utensils). The length of time that infective matter remains virulent is not clear, different workers stating various lengths of time, from three days to six months. Semmer states that pieces of spleen kept in ice are virulent after six months. Blood kept in gelatine at room temperature remains virulent after a fortnight, and, according to Nicolle and Adil-Rey, blood can be preserved in gelatine at a low temperature for two months. Nasal mucus in hermetically sealed tubes remains virulent after six weeks (Friedberger and Fröhner). Nocard maintains that manure remains virulent for several weeks.

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* Report of the Ceylon Government Veterinary Surgeon, 1909.

According to Krajewsky, skins are rendered harmless by corrosive sublimate, 0·1 per cent., in twenty-four hours; and by carbolic acid, 2·5 per cent., in twelve hours. The virus is destroyed by heating to 50° C. for half an hour, also by putrefaction, and by all ordinary disinfectants. Galtier states that sterilisation can be retarded when the air is confined and not renewed, when it is wet and fresh, when the light does not reach the virulent matter, and when desiccation or putrefaction is retarded. Sterilisation is hastened by putrefaction, by desiccation, by renewed air, by aëration, by ventilation, by heightened temperature, by great cold, and by light.

PERIOD OF INCUBATION.

This varies in different countries. From observations made during the outbreak in England it was estimated as from four to five days, whereas Roloff has observed a rise in temperature to occur in thirty-six to forty-eight hours after experimental inoculation. In the Himalayan hill cattle the period of incubation is about four days, whereas in the cattle of the plains of India it is very much longer.

IMMUNITY.

If an animal recovers from an outbreak of the disease, active immunity follows. Calves born from cows passing through the disease during the later stages of gestation are said to be immune. Regarding immunity produced by protective inoculation, see Protective Inoculation (p. 922).

MORBID ANATOMY.

The morbid lesions may be divided into external and internal.

The External Lesions consist of extreme emaciation; a muco-purulent discharge from the mouth, eyes, and vagina; epithelial eruptions on the region of the head, inside the thighs, and on the udder. These eruptions do not occur in the Himalayan hill cattle nor in the Philippine cattle.

The Internal Lesions.—The chief seat of the lesions is the mucous membrane of the alimentary tract, the abomasum and the small intestines being chiefly affected. The buccal mucous membrane is congested, and shows desquamation of the epithelium and erosions on the inside of the upper and lower lips; on the dental pad, the hard palate, and at the root of the tongue, there may be large areas showing abrasions of the mucous membrane; in the pharynx and œsophagus desquamation of the epithelium occurs. In the *rumen, reticulum,* and *omasum* areas of congestion, softening, and detachment of mucous membrane may be observed. In the *abomasum,* which is the seat of the most marked lesions, the mucous membrane is greatly congested, and ulcers are generally seen. Woolley states "that in the earlier stages of the disease the ulcers in the fourth stomach are preceded by minute red hæmorrhagic points, which resemble those seen in the early stages of intestinal amœbiasis in human beings. At the side of these points necrosis takes place, with coincident thrombosis of the bloodvessels supplying the affected area. Gradually this process extends, until finally an ulcer results, which has a punched-out appearance, with its necrotic base lying upon the muscular layer or even deeper tissues; the sides and bases of these ulcers consist in many instances of an almost homogeneous material, which is partly granular, staining profusely, and devoid of nuclei."

The mucous membrane of the *small intestine* is greatly congested, and ulcers are often present. Peyer's patches are also congested, and necrosis is often visible.

The *large intestine* is not the seat of marked lesions, though the mucous membrane is often congested. The mucous membrane of the rectum is highly congested, and presents a characteristic zebra-like marking.

Spleen and Liver are usually normal.

Gall-Bladder.—The lining membrane is congested, and sometimes presents erosions.

Heart and Kidney usually show petechiæ.

The mucous membrane of the *bladder, uterus,* and *vagina* is usually congested, especially that of the vagina. The *lungs* show a very characteristic interlobular emphysema, which gives a gross appearance of marbling, as in lung plague, only in this case the inflated interlobular tissue is dark-coloured instead of white, as in lung plague, and it collapses at once when incised (Law). The cerebrum and central nervous system appear hyperæmic.

Blood.—The blood shows marked increase in the leucocytes, alteration in the shape of the red blood-corpuscles (poikilocytosis), and does not coagulate readily.

Baldrey, who made a large number of observations on normal and rinderpest blood, states that in rinderpest there is a very marked leucocytosis, which may be divided into two periods, the primary and the secondary. The extraordinary feature of this leucocytosis is its suddenness. Twenty-four hours after inoculation with rinderpest virus there is an immediate rise to as much as three times the normal; the maximum may not be obtained until the second or third day after inoculation, or concurrent with the first rise of temperature. There is then a fall which is not quite so sudden, attaining its minimum about the fourth or fifth day.

SYMPTOMS.

The earliest symptoms are the sudden rise of temperature (104° to 105° F.). The animal is off its feed, has a staring coat, shows congestion of the visible mucous membranes, and there is cessation of rumination and lactation. When the fever is at its highest, small vesicles, the size of pinheads, are visible on the lower surface of the tongue, lips, and dental pad; these, as the disease progresses, develop into ulcers. The animal lies down with its head turned towards the flank; there is great tenderness of the loins. Diarrhœa of a fœtid character sets in on the fifth or sixth day, and towards the end the dejecta contain blood. There is salivation and a discharge from the eyes. The animal loses weight quickly, and becomes greatly debilitated. In some countries we observe that during expiration the affected animals make a peculiar clicking sound, but this does not occur among the Himalayan hill cattle. Skin lesions are also sometimes observed, especially in the more chronic cases; they consist of epidermic eruptions of the skin, in the region of the face, thighs, and udder. After the fever reaches its height, on the fifth day, it subsides somewhat rapidly, and the animal dies from the seventh to the tenth day, the temperature falling rapidly before death, and the animal usually dying in a comatose state. In some cases the animal dies on the day after the appearance of the high temperature, the fever suddenly abating.

COURSE AND PROGNOSIS.

The prognosis is very unfavourable, death usually occurring from the seventh to tenth day. In the indigenous breeds of Eastern Europe and Asia the disease is milder, and a large proportion of cases recover. In those cases that survive recovery is slow and convalescence prolonged. The progress of the disease in a herd is, as a rule, first slow, but afterwards becomes greatly accelerated. The disease spreads more widely in wet weather than in dry. The mortality in ordinary susceptible cattle is from 90 to 95 per cent.; in the cattle of the Russian steppes, already alluded to, the mortality is from 30 to 35 per cent.; in the Himalayan hill cattle it is 99 per cent. (Lingard).

DIFFERENTIAL DIAGNOSIS.

In the diagnosis of rinderpest the following points should be borne in mind:

1. The rapid spread of the disease.
2. The high mortality.
3. Sudden rise of temperature.
4. Vesicles or ulcers in the mouth.
5. The rapid wasting.
6. Tenderness in the region of the loins.
7. Groaning during respiration (not always observed).

Rinderpest can be differentiated from—

Malignant Catarrh by the presence of the vesicles or ulcers, by its rapid spread, by the respiratory system in malignant catarrh being alone affected, and by the course of the disease being much slower than in rinderpest.

Foot and Mouth Disease.—Although the eruptions of foot and mouth disease somewhat resemble rinderpest, it is easily differentiated by the appearance of the erosions on the mouth and upper surface of the tongue and the lesions of the feet, by the excessive salivation and champing of the jaws; there is not the same high temperature or disorder of the alimentary tract, and the constitutional symptoms are not nearly so severe.

Stomatitis.—In this disease principally young animals are affected, and although the eruption is somewhat similar to rinderpest, there is no rise of temperature and constitutional symptoms are absent.

Anthrax.—The characteristic lesions of the mouth found in rinderpest are absent; the enlargement of the spleen and the presence of the anthrax bacillus in the blood serve to differentiate this disease from rinderpest.

Hæmorrhagic Septicæmia.—The absence of any lesions in the mouth, the presence of the small bipolar organisms in the blood, the swelling of the throat, and the rapidity with which death takes place after the first symptoms are noticed, enables hæmorrhagic septicæmia to be differentiated from rinderpest.

Dysentery.—In dysentery no vesicles or ulcers are present in the mouth, and on post-mortem examination none of the characteristic lesions of rinderpest are seen.

PROPHYLAXIS.

This includes measures with reference to—(*a*) exclusion ; (*b*) extinction.

Exclusion.—The most important detail in connection with this consists in prohibiting the importation of ruminants and pigs from countries where the disease is known to exist into a country that is free from the disease ; also cattle and pigs that may have passed through infected routes should be prohibited. Hides, bones, wool, hair, etc., unless properly disinfected, should also be prohibited.

Extinction.—In any country where the disease is not endemic, rigid measures are necessary, and all affected animals should be traced and destroyed ; also those which have been in contact. Thorough disinfection of all buildings, boats, railway-trucks, and all litter, fodder, manure, feeding-troughs, attendants, etc., should be enforced. Clothing should be burnt, and the district placed under quarantine. Rinderpest is scheduled under the Diseases of Animals Act, and for further details with reference to prophylaxis the reader is referred to this Act.

TREATMENT.

Treatment in countries where the disease is not endemic is prohibited by law, owing to all therapeutic measures being practically inefficacious, and also to the serious danger of extensive outbreaks occurring. In countries where treatment is not prohibited by law all affected animals should be given large quantities of anti-rinderpest serum intravenously, and stimulants *per os ;* all animals in contact should be injected subcutaneously with serum, and allowed to mix with the diseased animals, so that they may contract the disease in a mild form, and thus obtain an active immunity.

PROTECTIVE INOCULATION.

The following are the main methods of protective inoculation :

1. Koch's fresh bile method.
2. Eddington's glycerinated bile method.
3. Simultaneous method (serum and virulent blood).
4. Serum alone method.

The Preparation of Anti-rinderpest Serum.

The method of making anti-rinderpest serum varies in different countries. The method for the preparation of serum used in India is as follows (Lingard's method) ;

Two classes of animals are employed—(1) cattle from the plains, averaging about 500 pounds in weight; (2) animals from the hills, averaging about 230 pounds in weight.

Plains Cattle Serum.—The animal is first immunised according to the simultaneous method, which consists of the injection of virulent blood (1 to 3 c.c.) on one side, and a dose of anti-rinderpest serum on the other. The result of this is that the animal gets a mild attack of the disease. There is a marked thermal reaction; the animal may go off his feed, and have slight diarrhœa; but after fifteen to sixteen days he regains his normal health. Three or four days later he receives a subcutaneous injection of virulent blood, varying from 4,000 to 6,000 c.c., the result of this being a very high thermal reaction, which gradually subsides, and after ten to twelve days his temperature becomes normal. About ten days later, if the animal is feeding well and is in perfect health, he is bled 2,200 c.c. twelve times at an interval of one week; he is then re-injected with 5,000 to 6,000 c.c. of virulent blood, and when this reaction is over, he is again bled twenty-one days or so from the date of the second injection six times (2,200 c.c. at intervals of a week). A week later he is again injected with 5,000 to 6,000 c.c. of virulent blood, and twenty days later he is again bled three times at intervals of a week: 2,200 c.c. at the first two bleedings, and at the final bleeding he is bled as much as he can stand.

Hill Cattle Serum.—The animal is first immunised by the simultaneous method, as already described in the case of the plains cattle. After the immunising reaction is over, he is injected with 2,000 c.c. of virulent blood, the result being a thermal reaction. Fourteen to sixteen days later, if the animal is in health, and has completely absorbed the blood injected, he is again injected with 2,500 c.c. of virulent blood. Again a reaction takes place. Twenty days from the date of the last injection the animal is bled 6 c.c. per pound body-weight, and forty-eight hours afterwards bled to death.

If the hill serum is mixed with plains serum in the proportion of 1 of the former to 3 of the latter, a protective serum is obtained, the protective dose of which is usually 108 c.c. per 600 pounds body-weight for hill cattle.

In order to hyper-immunise animals, it is evident that large quantities of virulent blood are necessary. This entails the slaughter of a large number of animals, and great expense.

Ruediger in Manila injected 5,000 c.c. of a 0·5 per cent. solution of potassium citrate into the peritoneal cavity of animals that were to be bled for virulent blood. An hour after the injection of the potassium citrate they were bled to death and the peritoneal fluid collected. This peritoneal fluid he used for hyper-immunising his serum-making animals. He found that the serum obtained had a greater protective value than the serum obtained from bulls that had been hyper-immunised with virulent blood. By using this peritoneal fluid a great saving of animals (for obtaining virulent blood) was gained.

This method was tried at the Muktesar Laboratory, but the serum obtained had not as great a protective value as that derived from bulls hyper-immunised with virulent blood.

Baldrey,* as the result of investigations into the value of this method, arrived at the following conclusions—viz.:

"1. That the method of employing peritoneal washings to augment the amount of inoculable virulent material is a good one.

"2. It produces an anti-rinderpest serum of a high value, but less potent than that produced by blood inoculation.

"3. In comparison with serum from blood inoculations, it is of greater value in the less susceptible plains animals than in the highly susceptible hill cattle.

"4. That its reaction is produced principally by a toxin which is rapidly formed under the vital influence of the peritoneal cavity.

"5. The inoculation of very large doses is not advisable in Indian cattle on account of the danger of death from toxæmia, the inability of the animals to absorb it subcutaneously, and the extreme caustic action it has upon the tissues.

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* "The Preparation of Anti-Rinderpest Serum by Means other than the Injection of Virulent Blood," by Major F. S. H. Baldrey, F.R.C.V.S., D.V.H., I.C.V.D., the *Journal of Tropical Veterinary Science*, vol. vi., No. 1, 1911.

"6. The method in suitable doses, which are small in comparison to the massive blood injections, is valuable in that a good serum is produced.

"7. Further experiments in diluting the fluid or mixing with blood may demonstrate improvements on the system as at present practised."

Method of Standardising Anti-rinderpest Serum.

Before serum can be issued for field work it is necessary to determine the dose of serum necessary to protect against the disease. If the average weight of cattle is taken at 600 pounds, it is necessary to find out what dose of serum will be required to be injected in order to protect this animal against rinderpest. The test is always done at the Muktesar Laboratory, which supplies the serum for field work in India on hill animals—*i.e.*, animals very susceptible to rinderpest. Two hill animals are injected at the rate of 36 c.c. of serum per 100 pounds on one side, and 0·5 c.c. of virulent blood on the other.

Two hill animals are injected at the rate of 72 c.c. of serum per 600 pounds on one side, and 0·5 c.c. of virulent blood on the other.

Two hill bulls are injected at the rate of 108 c.c. of serum per 600 pounds on one side, and 0·5 c.c. of virulent blood on the other.

The bulls are carefully weighed, and let us suppose that—

Bull No.	1	weighs	220	pounds.
,,	2	,,	230	,,
,,	3	,,	225	,,
,,	4	,,	240	,,
,,	5	,,	260	,,
,,	6	,,	250	,,

Bulls Nos. 1 and 2 are put on at the rate of 36 c.c. per 600 pounds
,, 3 and 4 ,, ,, 72 ,, ,,
,, 5 and 6 ,, ,, 108 ,, ,,

	Actual Weight.	Actual Dose of Serum given.
At 36 c.c. per 600 pounds—		
Bull No. 1	220 pounds.	13·2 c.c.
,, 2	230 ,,	13·8 ,,
At 72 c.c. per 600 pounds—		
Bull No. 3	225 pounds.	27·00 c.c.
,, 4	240 ,,	28·80 ,,
At 108 c.c. per 600 pounds—		
Bull No. 5	260 ,,	46·80 ,,
,, 6	250 ,,	45·00 ,,

The actual weight of bull No. 1 is 220 pounds; therefore the actual dose of serum to be injected is 13·2 c.c., this being at the rate of 36 c.c. per 600 pounds.

The actual weight of bull No. 2 is 230 pounds; therefore the actual quantity of serum to be injected is 13·8 c.c., this being at the rate of 36 c.c. per 600 pounds.

The actual weight of bull No. 3 is 225 pounds; therefore the actual quantity to be injected is 27·0 c.c., this being at the rate of 72 c.c. per 600 pounds.

The actual weight of bull No. 4 is 240 pounds; therefore the actual quantity of serum to be injected is 28·8 c.c., this being at the rate of 72 c.c. per 600 pounds.

The actual weight of bull No. 5 is 260 pounds; therefore the quantity of serum to be injected is 46·8 c.c., this being at the rate of 108 c.c. per 600 pounds.

The actual weight of bull No. 6 is 250 pounds; therefore the actual quantity of serum to be injected is 45·0 c.c., this being at the rate of 108 c.c. per 600 pounds.

If bulls Nos. 1 and 2 die and Nos. 3 and 4 show a large reaction, but live, and Nos. 5 and 6 show only a very slight thermal reaction, it is evident that 45 c.c. of serum has sufficient protective value to protect a hill bull weighing 250 pounds—

i.e., at the rate of 108 c.c. per 600 pounds—against rinderpest. As the average weight of these hill bulls is under 300 pounds, the dose that the serum is issued at is 50 c.c. per animal.

Koch's Bile Method.—This consists in taking bile from the affected animal from the sixth to the eighth day of the disease, and injecting non-immune animals with 10 c.c. of this bile. The bile thus taken must be absolutely free from blood.

As the result of this injection of bile, an active immunity is produced after ten days. Koch had good results by this method, but it has several drawbacks, and in the hands of others the results were not so successful (Lingard, Rogers, and Hutcheon). The great disadvantages of this method are: Firstly, immunity does not set in for ten days, and, according to Haedicke, only lasts four months. Secondly, there have been a large number of fatalities from rinderpest after its use. Thirdly, a sufficient quantity of bile cannot be obtained for the inoculation of large herds. This method has been modified by other workers. Kohlstock, working in German West Africa, gave two inoculations of bile, and about fourteen days after injected small doses of virulent blood. This method, which entails three inoculations, is inconvenient from a practical point of view. Eddington's modification consisted of adding an equal quantity of glycerine to the bile and keeping it for eight days. Eddington injected 20 to 50 c.c. of bile, and a few days later injected small doses of virulent blood. Immunity set in after fourteen to sixteen days, lasting a long time. With this method it was found that in many cases the bile did not protect the animal when inoculated with the virulent blood, and death resulted; and here, again, it has the disadvantage that there is great difficulty in obtaining a sufficient quantity of bile for large herds of cattle. Eddington states that glycerinated bile can be kept for a year without losing its protective properties.

Glycerinated bile alone, produces a passive immunity lasting only for a very short time.

Serum Simultaneous Method.—This method consists of injecting the animal with anti-rinderpest serum on one side and virulent blood on the other. The animal gets a mild form of the disease, and a lasting immunity is established after fourteen to sixteen days. It has the disadvantage that a small percentage of deaths occur after inoculation, and that at times it is difficult to obtain the virulent blood from a sick animal.

Serum Alone Method.—This method consists in simply injecting a protective dose of anti-rinderpest serum. The animal injected with serum should be allowed to mix with the diseased animals, whereby they contract a mild form of the disease, and thus obtain an active immunity. If they are not allowed to mix with the diseased animals, a passive immunity only is produced, which soon passes off. With regard to the immunity produced by the serum alone method, Holmes, in the Report of the Research Work of the Imperial Bacteriological Laboratory, Muktesar, 1908-09, has gone fully into the question, and the following summary indicates the results obtained:

Summary.

The general conclusions arrived at from the results of these experiments are that—

1. A single dose of anti-rinderpest serum confers immunity against the inoculated virus for about two weeks only.

A double dose protects for about three weeks, a treble dose for five weeks, and four times the single dose for about six weeks.

2. The serum protects against natural infection for the same period as against the inoculated disease.

3. The immunity following a simultaneous injection of serum and virus, even when no reaction occurs, is of an active nature and lasts for

several months, and that to produce this result it is not necessary to determine a balance between serum and virus, nor to give rise to any clinical symptoms of the disease.

4. When animals are injected with serum and immediately exposed to infection, they acquire an active immunity of several months' duration, without evincing any clinical symptoms of the disease.

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EQUINE PERNICIOUS ANÆMIA

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Equine infectious anæmia.

French : Anémie infectieuse; Anémie épizootique; Anémie pernicieuse progressive; Typho-anémie infectieuse.

American : Swamp fever; Equine malaria; Spanish fever.

Definition.—A contagious inoculable disease, caused by an ultravisible virus, affecting the horse and mule, and characterised by extensive alterations in the blood, with usually profound and rapid emaciation and marked debility. Three types of the affection are recognised—viz., the acute, the subacute, and the chronic.

HISTORY.

The disease was first described in veterinary literature by Lignée in 1843, and in the same year by Denoc and by Charlier. It was observed in France, chiefly in the Valley of the Meuse, also in the Haute-Marne and other districts. Delafond investigated it in 1851, but did not recognise its contagious nature. In 1859 Anginiard held that it was contagious, and in 1861 Ledru advanced a similar view. From this period the disease appears to have attracted very little attention, until Carré and Vallée published the results of their investigations concerning it in 1904 and 1905, also in 1906 and 1907,* and demonstrated the inoculability of the malady and the ultravisible character of the virus on which it depends.

At the present day the disease occurs in France in the district of the Marne, in the Côte-d'Or, in the cavalry regiments " de l'Est." Zschokke has met with it in Switzerland, Ostertag in Germany, and Hutyra and Marek in Hungary (Cadéac). A disease possessing similar characters, and known as " swamp fever," prevails in various parts of the Western United States and in some of the North-West Provinces of Canada, and has been studied by Van Es, Harris and Schalk, at the North Dakota

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* *Revue Générale de Médecine Vétérinaire.*

Agricultural Experiment Station; by Torrance, in Manitoba; by Peters in Nebraska; and by many others, including Mohler. According to Todd and Wolbach,[*] swamp fever seems to have been first recognised as a distinct disease in or about 1880, and since then it has been widely identified. The cases are usually occasional and few in number; sometimes, however, the disease occurs repeatedly on the same farm, or it may become almost an epizoötic, and carry off at once the majority of the horses from a farm, or from the stud of a contractor engaged in railroad construction work. The first definite mention of swamp fever in the literature of the subject was in 1884, when Rutherford recorded cases occurring at Portage-la-Prairie, N.W.T. Since then the disease has occurred as far north as Dauphin and Yorkton, and as far west as Edmonton. In the United States it was first met with in Oklahoma (North-Eastern Indian Territory), and during the past eight years has extended north and east into Kansas and Missouri.[†] It is not quite certain that the American disease is identical with the equine pernicious anæmia of the Continent. Up to the present time the disease has not been identified in Great Britain.

ANIMALS AFFECTED.

In France the disease is found to attack horses, and neither age nor breed seems to have any effect on its incidence. According to Carré and Vallée, it can be communicated to the ass. Cadéac states that it has not been observed in the mule, and that bovines, sheep, goats, dogs, mice, and white rats, are refractory to infection. In America mules are affected as well as horses; the disease is said to be to a slight extent more prevalent in the latter than in the former.

ETIOLOGY.

Equine pernicious anæmia is caused by an ultravisible virus, and can be communicated experimentally to healthy horses by intravenous or subcutaneous injection of blood obtained from affected animals, and naturally by ingestion of forage or litter contaminated by the virus. The experiments conducted by Carré and Vallée demonstrated the above facts. These authorities also showed that the disease can be transmitted to a series of horses by inoculation, and that successive passages induce an exaltation of the virus. Thus, in an experiment carried out in which inoculations were made from horse to horse, the animal from which the infective blood was taken in the first instance died in five months; the first subject inoculated succumbed in sixty-nine days; the second in forty-two days; the third in twenty-seven days; and the fourth in twenty-six days. Inoculations may produce either the acute, sub-acute, or chronic type of the disease, thus showing that all depend on a similar virus. The amount of blood inoculated has no influence on the severity or course of the disease produced, 10 c.c. producing similar effects to 300 c.c. The animal inoculated with 100 c.c. of blood-serum from an infected case, or 500 c.c. of filtrate of the diluted blood, shows, after six days of incubation, the usual symptoms of the disease, and death occurs in forty-six days, typical lesions being found at the autopsy.

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[*] Dr. I. L. Todd, Special Report on Swamp Fever to Canadian Department of Agriculture.

[†] Kinsley, *American Journal of Veterinary Medicine*, January, 1911.

The virus is present in the blood during life, and experiments show that immediately after death the blood is also virulent. Some authorities state that the blood contains the virus in greatest abundance.

The urine is also virulent during the entire course of the malady. The fæces are also infective, especially when dysentery accompanies the disease. The virulent blood is unaffected by exposure to diffuse light for a month, but exposure under similar conditions for three months renders it non-infective. The virulence is unaffected by desiccation in a vacuum at room temperature for ten days, but after seven months' desiccation it is rendered ineffective. When heated for one hour at 58° C. the virulence is destroyed. Advanced putrefaction has no effect on the vitality of the virus. A flask containing 20 c.c. of virulent serum and 1 litre of virulent urine was buried in a manure-heap for three months; then 200 c.c. of the putrid mixture administered to a horse *per os* produced a typical attack of pernicious anæmia (Carré and Vallée).

In swamp fever the virus was found to be resistant to severe cold. According to Todd and Wolbach, it is caused by an infecting agent which can be transmitted from horse to horse by the inoculation of blood taken from an infected animal. It can be conveyed experimentally to horses, mules, and donkeys, but attempts to inoculate cattle, dogs, cats, rabbits, and guinea-pigs failed. The causal agent was not discovered.

MODES OF INFECTION.

The ingestion of forage, litter, and water contaminated by the urine or fæces of infected animals, is recognised as the common method by which the disease is spread. The forage and litter become contaminated by contact with the floor of the stables in which infected animals are kept. Experiments show that considerable amounts of the virus must be ingested before infection can occur in this manner, but the continuous ingestion of contaminated fodder, such as occurs when a horse is kept in a stable with infected animals, can bring about transmission of the disease.

In infected districts it is well known that stables occupied by horses suffering from the disease prove infective for fresh horses introduced therein, unless thorough disinfection be carried out.

Apparent recoveries from the disease are not uncommon, and such animals act as "carriers" of infection, as they are capable of contaminating the forage and litter by means of their urine, which contains the virus.

These carriers, when introduced into a stable of healthy horses, transmit the disease to the latter. Carré and Vallée draw attention to certain resemblances between pernicious anæmia and piroplasmosis; thus, in both affections the blood remains infective in cases that appear to have completely recovered, and in such instances relapses are frequently met with. After an infected animal has been introduced into a stable, cases of the disease appear in from ten to twenty-five days. According to Mutulet, pastures may also become contaminated by the virus.

American observers point out that the disease is more prevalent during July, August, and September, than during the remainder of the year, and more cases are met with in wet seasons; also that foals may become affected while suckling a healthy mare; again, in other instances foals may suckle affected mares with impunity.

Rutherford states that the disease is most prevalent in a low-lying swampy country, and is more frequently met with in the late summer and early autumn, but that a few fresh cases come under observation between December and the latter part of July.

According to Ostertag, instances are known in which horses were kept in the same stable as an infected animal, and ingested the litter, but remained free from the disease; this apparent immunity in some horses has also been observed by American practitioners.

MORBID ANATOMY.

The most important lesions are found in the circulatory system. The morbid alterations in the blood will be found described under the heading of Symptoms (see p. 933).

Heart and Bloodvessels.—In the acute form the heart and large vessels are usually full of dark, imperfectly coagulated blood. The heart shows multiple subserous, subpericardial, and subendocardial ecchymoses. On section, the myocardium is studded with numerous hæmorrhagic centres. Valvular œdema is also observed, and frequently the ventricular endocardium shows a greyish-yellow, parchment-like, horny appearance, as if it had been subjected to the action of a mild caustic.

In the chronic form the myocardium is discoloured and spotted with rose-coloured or greyish patches, which are the remains of former hæmorrhages. In some cases a slight valvular œdema, and also atheromatous lesions of the aorta, are observed (Carré and Vallée).

According to Cadéac, the heart is flabby; its cavities and those of the large vessels only contain a slight amount of blood, which forms small clots, partly white and partly black in colour.

Liver.—This organ is generally enlarged and pale yellow in colour; in some cases it is studded with small white spots, and may present the appearance of a " cardiac " liver.

The liver substance is very friable, and rupture of the organ has been observed as the result of the animal lying down abruptly (Carré and Vallée).

Intestines.—In acute cases numerous subserous hæmorrhages are found. The mucous and muscular coats present more or less intense congestion, most marked in the large intestine, in which the mucous membrane may show widespread hæmorrhages. Ulcerations may sometimes be observed in the chronic form.

In cases of the disease that develop slowly the intestines may be normal in appearance.

Spleen.—In the majority of cases the spleen is double or treble its normal size. When the attack is not acute, the organ is thickened, shows rounded projections towards its centre, and its consistency is firmer than normal. On section, the splenic substance is pale, and the Malpighian corpuscles are hypertrophied, granular, and greyish in colour. When the attack is acute, the organ shows softening, and its capsule is studded with hæmorrhages. Some American observers state that in acute cases the spleen shows no enlargement, although it is darker in colour than normal and softer in consistency. Others describe this organ as being generally much enlarged. According to Mohler, it is at times found enlarged and covered by petechiæ. Hutyra and Marek found that the slower the course of the disease the slighter were the changes in the spleen, and in very protracted cases the enlargement was often completely absent.

Kidneys.—In the acute form these organs are congested, hæmorrhagic, and very friable. The capsule is not adherent; in some cases small circumscribed nodules containing pus, and of the dimensions of a pin's head, are found in the cortical portion, and project above the surface of the capsule. In the chronic form the kidneys are pale in appearance.

Lungs.—In the acute form subserous ecchymoses may be observed in some cases.

Lymphatic Glands.—In the acute and subacute forms, and in cases that die from an acute attack while suffering from the chronic form, the lymphatic glands—especially those of the mesentery and spleen—show congestion or even hæmorrhages. In the chronic form the lymphatic glands of several regions are found pale, swollen, and œdematous, and are often surrounded by a yellowish effusion.

Bones.—Lesions of the bone-marrow are generally present. They are especially well marked in the femur; the bone-marrow to a great extent is altered to a sanguineous pulp, of a brick-red or blackish colour, and resembles in its consistence and histological characters fœtal bone-marrow.

Amongst other lesions met with in the disease is the presence of extensive infiltrations of the subcutaneous cellular tissue in the dependent regions of the body. Intramuscular and subserous infiltrations may also be observed. There is an absence of fat all over the body, and marked emaciation is present; the muscles are atrophied, especially those of the hind-quarters and thighs, and sometimes all the tissues show a slight icteric tint.

SYMPTOMS.

Clinically, three types of the disease are recognised—viz., *acute, sub-acute,* and *chronic.* These appear to merge into one another, and all of them may be represented in the same outbreak.

Acute Type.—In this the evidences of the disease appear rather suddenly. The animal has a dejected appearance, is unfit for exertion, and if forced to work, he shows weakness of the hind-limbs and accelerated respirations; loss of appetite is also observed, and food may be kept in the mouth without any attempt at mastication being made. The conjunctiva is injected, œdematous, and of a reddish-yellow colour. The hæmoglobin transudes with the serum into the conjunctivæ, and infiltration of this structure occurs. This infiltration is frequently associated with the presence of petechiæ of a red, brownish, or purple colour, which disappear in twenty-four to forty-eight hours, but reappear in a short time. Swelling of the eyelids and photophobia are generally absent, but lachrymation is frequently observed. The temperature gradually rises to 104° or 105° F., and in some cases to 107° F., and generally remains high until the approach of death; the maximum is not reached until after two or three days. Remissions are sometimes observed for a period of twenty-four to forty-eight hours.

The pulse is soft, small, and frequent; the cardiac sounds are loud, vibrating, and metallic in character; and the slightest movement of the animal induces irregularity and intermittency of the heart's action (Carré and Vallée). The dependent parts of the body show slight œdematous swellings. While the animal is at rest nothing abnormal may be observed in connection with the respiratory system.

In some cases the disease has an insidious onset, and well-marked symptoms may not be observed for two or three days.

The loss of muscular power is so extreme that the hind-limbs are trailed along the ground while walking; and if forced to take exercise, or if turned round quickly, the animal may fall. The attitude assumed by the patient in the stable may resemble that observed in cases of laminitis. Or the head may be held immovable and extended, and the nostrils are dilated, so that the appearance may resemble that of tetanus. Again, the head may rest on the manger, and the lower lip may be pendulous. Atrophy of the muscles is especially well marked in those of the hind-quarters, and rapid emaciation is one of the leading features of the disease.

As the fatal termination is approached, paralysis of the hind-quarters supervenes; incontinence of urine is also observed, and relaxation of the *sphincter ani.*

The urine is increased in amount, is generally high-coloured, and almost always contains albumin. The amount of albumin present varies; in some instances Carré and Vallée have found up to 14 grammes to the litre. Van Es and his colleagues found that in swamp fever the occurrence of albumin in the urine was transitory, and frequently synchronous with the febrile exaçerbations. Epithelial and granular casts are present, but hæmoglobin and red corpuscles are generally absent.

Evidences of enteritis, with diarrhœa, may be observed. The fæces often present a reddish tinge, and in some cases contain streaks of blood; colicky symptoms may also be present.

The usual termination of the acute type is death, which generally occurs in eight days; but in some cases the patient may last for fifteen days.

The alterations in the blood constitute the most striking feature in connection with the disease. Carré and Vallée point out that in cases of the acute type evidences of the altered condition of the blood and of anæmia may not be apparent in the patient, the clinical picture resembling that observed in influenza. The various alterations in the blood are described by the above-named authorities as follows:

"The blood coagulates slowly; the red blood-corpuscles separate rapidly from the plasma; they are of a brownish-red tint, and become agglutinated, forming small masses which float in the plasma. The plasma is only slightly coagulable; it is of a deep yellow or greenish tint, and very often dichromatic, notably in the acute type, and during the course of the subacute or acute attacks which supervene in the chronic type of the disease. The red blood-corpuscles in the acute type are very friable, and agglutinate easily and rapidly. They are irregular in form, very variable in diameter, poor in hæmoglobin, and are fragile. They show no powers of resistance to the normal serum of the rabbit and the horse, saline solutions, etc. (agents which exert no destructive action on healthy blood-corpuscles). A large number of punctated corpuscles are observed; these, after treatment with basic aniline colours, reveal a corpuscle which in form, dimensions, and colour resembles a piroplasm."

"The number of red corpuscles is diminished to a marked extent. The number of leucocytes is slightly lessened, the alteration oscillating between 7,000 and 7,500, instead of 9,000, which is the normal count; the minimum observed in the cases investigated was 4,560. In the acute type the polynuclears are always increased to a marked extent; they represent from 75 to 90 per cent. of the total number of leucocytes."*

According to Cadéac, the acute type of the disease is to be regarded as a general intoxication of the system, of a typhus character, which determines general depression and alterations of the blood, and especially loss of the oxygenating functions of the blood.

Van Es found that "many cases of swamp fever terminated fatally without a marked reduction in the red blood-cells—a fact denying the popular conception of the disease being primarily an anæmia."

Subacute Type.—This commences with all the characters of the acute type, but they are more or less modified. Well-marked remissions occur, during which, when evidences of anæmia are not apparent, one might imagine that recovery had taken place. During these remissions the appetite returns, the ocular symptoms disappear, the temperature is lowered, and the œdematous swellings become absorbed. In a variable period—from a few weeks to several months—a fresh attack appears, and the symptoms return.

According to Cadéac, the return of the animal to work acts as an exciting cause for the development of the disease that is present in a latent form in the system. Although the animal may appear to be in good condition, an examination of the urine shows the presence of albumin, and careful observation reveals the altered condition of the conjunctiva; if blood be drawn from the jugular vein and examined, evidences of anæmia will be found. The horse is unfit for exertion, and if forced to work, an acute attack may develop which may prove fatal in the course of a few hours or days.

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* *Revue Générale de Médecine Vétérinaire*, 1906.

The duration of the disease is very variable in the subacute form; death may supervene in a few weeks or not for several months, either during an acute attack or from progressive exhaustion.

Chronic Type.—The affection commences in an insidious manner, without any premonitory symptoms being observed. The animal loses condition, has a "staring" coat, and the hairs are readily removed. The buccal mucous membrane is pale and of a yellowish tint; the conjunctiva is infiltrated and of a yellowish hue, and the eye has an "oily" appearance; the appetite is capricious, and the animal is easily distressed, and sweats profusely when at work. The temperature is generally normal, but intermittent febrile attacks are observed. Circulatory disturbances are common, tachycardia, cardiac intermittency, and a venous pulse being present. The pulse is soft, frequent, and irregular, and the artery is compressible. A cardiac murmur may be observed in some cases. The chronic type of the disease is thus characterised by well-marked evidences of anæmia. The urine is increased in amount, is generally albuminous, and micturition is frequent.

Intermittent attacks of diarrhœa may sometimes be observed. An examination of blood drawn from the patient shows that it is lighter in colour than normal blood; the clot formed is lacking in consistence, and resembles jelly in appearance; the red corpuscles show a remarkable diminution in number, and on the approach of death the blood-count may not show more than one million of red corpuscles to the cubic millimetre. The mononuclears and polynuclears are about equal in amount. Under careful dieting and cessation from work, an animal suffering from the chronic type may temporarily improve, but death ultimately results, either from the occurrence of an acute attack or from exhaustion depending on an advanced stage of anæmia. When accidental hæmorrhage occurs, such as from wounds, there is marked difficulty in arresting it, and it is found that even slight wounds tend to heal very slowly. During the later stages of the disease the animal shows marked weakness, and moves with difficulty, staggers in his gait, and may be unable to rise without assistance. The duration of the disease varies from three to four months, and is sometimes longer. Recovery is rare, and exacerbations are commonly met with. When new horses are introduced into an infected stable, they generally develop the acute or subacute type of the disease; these are also the types generally observed when the affection first appears in a district.

According to Todd and Wolbach, swamp fever is usually a chronic disease, characterized by emaciation, weakness, irregular temperature, and anæmia.

DIFFERENTIAL DIAGNOSIS.

The acute type of the disease may be confounded with influenza, and it would appear that this error is not uncommonly committed. According to Cadéac, the diagnosis is to be based on the history of the case, and on the prevalence of the disease in a district. Influenza occurs in the form of an epizoötic, and either brings about the death of the patient in a comparatively short time, or terminates in complete recovery. On the other hand, in infectious anæmia neither swelling of the eyelids nor photophobia occurs.

Still, it must be remembered that in the acute type of the latter disease death may occur within eight days, and that ocular symptoms do not occur in all cases or outbreaks of influenza, so that considerable difficulty may be experienced in the differential diagnosis of cases encountered for the first time.

In the chronic type the prolonged course of the disease, the evidences of anæmia presented, and the fact that recoveries are rare, should enable a differential diagnosis to be made.

Examination of the blood is regarded by some observers as an auxiliary in the diagnosis of the disease. According to Zschokke, if 10 c.c. of blood drawn from an affected animal be placed in a graduated glass, and the latter be placed in water at a temperature of 10° to 12° C., it will be found that after coagulation has occurred the layer of red corpuscles does not rise to the same height as in the case of normal blood; in the latter the height is equal to two-thirds of the total column of blood. Kinsley* states that the hæmoglobin test is of considerable value, but that blood inoculation is the only positive method of diagnosis.

Van Es† states: "At present there is no positive means of diagnosing the disease except by the inoculation of a susceptible animal." He also observes that "the appearance of cases of the disease on a farm from time to time indicates that there is a 'carrier' on the farm; to find this 'carrier' is often a most difficult task."

PROGNOSIS.

This must be regarded as very grave in all forms of the disease. The losses in infected districts in France are very serious.

In stables containing from ten to twenty horses, all the animals may succumb in a few months, and those purchased to replace them meet with a similar fate; in other instances from one-quarter to one-third of the affected animals perish.

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* *American Journal of Veterinary Medicine*, January, 1911.

† *Ibid.*, February, 1911.

PROPHYLAXIS.

All doubtful cases should be isolated. With reference to obviously infected cases, isolation is also advised, but Carré and Vallée and others consider that the most economical course is to order their destruction.

Horses apparently cured are the most fruitful source of infection, and when discovered they should be isolated or, more preferably, destroyed. A careful examination of the urine should be made in suspected cases, as the presence of albumin therein is one of the most persistent symptoms in cases that, although apparently recovered, act as "carriers" of the disease. Horses should not be purchased from infected districts, and owners should not be permitted to sell horses that are known to be "carriers" of the disease. The fæces and urine of affected animals must be carefully disposed of, so as not to come into contact with healthy horses. Infected stables should be thoroughly disinfected. Attention should be directed so as to prevent forage and water from becoming contaminated by infected animals.

According to Rutherford, "the reports from the western provinces indicate that, as a result of draining and cultivation in the low-lying districts where swamp fever was formerly exceedingly prevalent, its appearance is now comparatively rare."

TREATMENT.

Up to the present time no medicinal agent has been discovered that exerts any beneficial effect on the disease. Atoxyl and allied substances, also trypan-blue, have been tried, but with negative results.

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MILK SICKNESS

By E. WALLIS HOARE, F.R.C.V.S.

Synonym.—The Trembles.

In certain districts of the United States, to the west of the Alleghany Mountains, a peculiar enzoötic disease termed " milk sickness " occurs in cattle, sheep, pigs, and dogs. In the days of the earlier settlers it was far more prevalent than at present, and timbered, uncultivated lands seem especially favourable to its occurrence. The disease is communicated to man by eating the flesh or drinking the milk of affected animals, also from ingesting butter or cheese made from infected milk.

Etiology.—Nothing definite is known with reference to the causal agent, and so far as adult cattle and sheep are concerned the mode of infection is not determined. Calves fed on milk from an affected cow contract the disease; pigs which consume the flesh of calves that have succumbed to the malady become infected; dogs suffer from the disease as the result of ingesting the meat and milk of affected animals. In experiments it was found that 1 ounce of butter or cheese made from the milk of an affected cow, or 4 ounces of raw or boiled beef from an animal that had suffered from the disease, given three times a day to dogs, proved fatal within six days.

In districts where the disease was prevalent it was found that by cutting down the timber, and thus exposing the land to sunlight, no fresh cases occurred. Ploughing and cultivating the land had a similar effect, but was not found essential in all instances. The malady is said to be unknown in the open prairies of the Western States, where animals were not allowed to remain overnight in the timber belts. Dry seasons are found to be favourable to the occurrence of the disease, and animals left out during the night, when fogs and dews are prevalent, seem specially liable to become infected. The majority of cases occur during late summer and autumn.

Milch cows are very susceptible to the affection, and a peculiar point observed is that, although showing no apparent symptoms, such an animal is capable of infecting others. But when a milch cow becomes dry, she shows similar symptoms to those observed in other animals.

Bullocks, bulls, and heifers, are very susceptible, and are attacked in a severe manner.

Exertion or fatigue acts as a predisposing cause of the malady. In cows suspected of being infected it is found that by subjecting them to exertion the symptoms develop.

Period of Incubation.—This is found to vary from eight to twelve days, but in some instances it is as short as two days.

Symptoms.—In *Cattle* the first symptoms observed are a dull, listless appearance, drooping head and ears, loss of appetite, cessation of rumination, thirst, and obstinate constipation. The animal is generally found lying down, and is made to rise with difficulty. If forced to walk, it does so in a stiff manner. A characteristic symptom of the disease is the occurrence of trembling when the animal stands still. This trembling involves the entire muscular system. In some cases death may occur suddenly, or may be preceded by convulsions.

As the disease advances, muscular debility is well marked; the animal lies down with head stretched on the ground, and is unable to rise. The eyes are injected and become fixed and glazed, the corneal reflex is abolished, the pulse becomes infrequent, the respirations are slow, and the temperature is often found to be subnormal. Coma next appears, and death generally occurs on the eighth or tenth day. When affected animals are subjected to severe exertion, death is always hastened. The mortality is high, and in cases that recover convalescence is prolonged.

Mild types of the disease are met with, in which no symptoms are observed while the animals are at rest; but if subjected to the slightest exertion, the characteristic trembling of the muscles appears, accompanied by evidences of debility and disinclination for movement. Calves are observed to tremble while sucking, and may vomit, death usually occurring in a sudden manner.

In *Sheep* marked prostration is a prominent symptom, and the animals are generally unable to rise.

In *Pigs* vomiting occurs, constipation is present, and depression is well marked.

In *Dogs* dulness, vomiting, stiffness in progression, and disinclination for movement, are the symptoms observed.

Lesions.—"The gastro-intestinal mucosa may be congested, and a dark, firm condition of the membrane is usually present. In some cases capillary stasis and sloughing are observed."

"The contents of the stomach (paunch in cattle) and intestines form hard balls like cemented sawdust, firmly adherent to the dry mucosa" (Law).

Prophylaxis.—This consists in clearing the timber and brush land, and cultivating the ground; also keeping animals away from infected woods in late summer, and excluding them from such pastures from before nightfall until after the dews have evaporated on the following morning (Law).

Calves, pigs, and dogs, should not be fed on the milk from infected animals.

Treatment.—Various forms of treatment have been suggested for cattle suffering from the disease, such as eserine to act on the bowels, internal antiseptics, nerve stimulants, etc., but without any beneficial results.

MILK SICKNESS IN MAN.

According to Osler, the disease is rare in the present day; but it prevailed extensively in the early settlements in certain of the Western States of America, and proved very fatal. It still prevails in parts of North Carolina. The symptoms are those of a more or less acute intoxication. After a few days of uneasiness and distress, the patient is seized with pains in the stomach, nausea, vomiting, fever, and intense thirst. Obstinate constipation is usually present, the tongue is swollen and tremulous, the breath is foul, cerebral symptoms are sometimes marked (such as restlessness, irritability, convulsions, and coma), and a typhoid state may gradually be produced, in which the patient dies. The duration of the disease varies. Death may occur in from two to three days in the most acute form; other cases may last for ten days, or even for three or four weeks. No definite pathological lesions are known, and the causal agent is undiscovered.

Man contracts the disease only by eating the flesh of affected animals; also from the use of infected milk, butter, and cheese.

Prophylaxis.—Milk from infected districts should be pasteurised or sterilised before use, and also before being employed for the production of butter and cheese. It is advised that animals from these districts should be tested by long or vigorous driving before they are slaughtered for food. Milch cows may be a special source of infection, as they may show no symptoms and yet yield infective milk. The test of subjecting them to exertion in order to develop latent symptoms is employed in the vicinity of infected localities, and appears a rational course to adopt.

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DENGUE

By E. WALLIS HOARE, F.R.C.V.S.

Synonyms.—Stiff sickness ; Three-day sickness.

This is a peculiar epizoötic disease affecting cattle in Egypt, the Transvaal, Natal, and Rhodesia. It has also been recognised in India. It is characterised by the presence of fever, muscular stiffness, and lameness, and in uncomplicated cases it runs its course in three or four days.

The disease was described in Egypt by Piot-Bey,* and in some of the outbreaks large numbers of cattle were attacked, especially those of the male sex.

Bevan † wrote a preliminary report on the affection under the heading of "Stiff-Sickness or Three-Day Sickness of Cattle in Rhodesia." According to him, the disease first made its appearance among cattle in North-Western Rhodesia, and was investigated by Edmonds during the latter part of November, 1906. In January, 1907, an outbreak occurred at Mtobo, a few miles south of Bulawayo, and soon afterwards another was notified 100 miles north-east of Mtobo. After this, outbreaks occurred in various districts. Cases were observed at Umtali in March, and during the last week of that month outbreaks of the disease were officially notified from the Transvaal and Natal.

Cattle of all ages and breeds, of both sexes, and in high or low condition, were susceptible to the disease. Working cattle were affected, as well as those at pasture, and the nature of the soil does not appear to have had any effect on the incidence of the affection. Cases are recorded of the disease occurring naturally in sheep. As regards immunity, according to Piot-Bey and Bevan there is no record of an animal having suffered from a second attack of the malady.

Etiology.—The causal agent has not up to the present been discovered. The mode of transmission is also unknown. Bevan points out that although "the disease has made its appearance along a definite route, intervals of many miles have occurred between the various infected areas. Moreover, in the infected areas outbreaks have occurred upon different farms far removed from one another. It is unlikely that the disease is transmitted from area to area or from farm to farm by the movement of cattle, since under the regulations enforced to eradicate African coast fever the movement of cattle is prohibited except by permit issued by the Veterinary Department, and herds are regularly inspected and their numbers checked by a staff of cattle inspectors. If the disease can be transmitted from a sick to a healthy animal, the foregoing circumstances would point to an intermediary bearer capable of travelling long distances."

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* *Bulletin de l'Institut Égyptien*, 1897 and 1909.

† *Journal of Comparative Pathology and Therapeutics*, vol. xx.

Microscopical examination of the blood has given negative results. The same investigator injected quantities of citrated blood from an affected animal into a donkey, a rabbit, a chicken, and a sheep ; but only the last of these yielded a reaction. He ascertained that if the blood of an ox suffering from the disease be inoculated into a sheep, a typical febrile reaction is produced between the sixth and tenth days, but this reaction is not accompanied by stiffness or lameness. A similar reaction was obtained by inoculating a sheep subcutaneously with an emulsion of spinal cord taken from an ox that had died of the disease, thus showing that the infective material is also present in the spinal cord. Sheep which reacted to the above inoculations showed no reaction to a second injection of virulent blood. Robertson injected virulent blood into the jugular vein of an ox, and obtained a temperature reaction and also distinct stiffness of the limbs.

Morbid Anatomy.—As the mortality from the disease is very low, opportunities for observing the lesions have been limited. The chief pathological changes encountered by Bevan were " an altered condition of the blood, which clots very readily, and which allows a ready effusion of red-coloured material into the surrounding tissues ; also a quantity of blood-stained fluid in the pericardial sac, and a violent inflammation of the lining of the abomasum."

The lesion in the abomasum was characterised by an intense magenta discoloration of the mucous membrane, resembling the condition of the stomach occurring in South African horse sickness. In one case the lesions were confined to the brain and spinal cord, both of which showed congestion. An elevation of the covering membrane between the convolutions of the cerebrum, due to the presence of a watery gaseous fluid, was also observed. Other nervous lesions recorded were a pink staining of the cerebrum, caused by effusion from the bloodvessels, and an abnormal amount of reddish cerebro-spinal fluid.

Bevan thought that the lung lesions which were met with may have been due to the administration of medicines which, owing to the difficulty in swallowing, entered the trachea and bronchi.

In some cases, with the exception of cerebral congestion and pericardial effusion, no lesions were found on post-mortem examination, although typical symptoms were present during life.

Symptoms.—Although stiffness and lameness constitute the prominent features of the disease, there were many instances in which the earliest clinical symptoms presented were a swelling around the eyes, injected conjunctivæ, well-marked lachrymation, and a glaring appearance of the eyes.

Either one or more of the limbs may be affected with stiffness and lameness, both fore or both hind legs may be involved, or one fore or one hind. In other instances all four limbs are attacked simultaneously. The lameness may be of a metastatic character, rapidly passing from one limb to another. The animal is disinclined for movement, and when forced to walk the gait resembles that observed in a horse suffering from laminitis.

The muscles of the neck or of the back may be affected. In the former case the neck is held in a stiff manner and the head is protruded. Occasionally the lips are drawn to one side.

If the animal be examined while at rest, the muscles of the affected limbs are found in a rigid condition. They may feel hard and tense to the touch, and often a quivering movement is detected in them. The pharyngeal muscles may become involved, and deglutition is rendered difficult, or the animal may be unable to swallow either solids or fluids. Depression is well marked, frequent grinding of the teeth and groaning is observed, and when the animal lies down the head may fall to one side, the attitude resembling that seen in cases of milk fever in cows.

In some cases the animal can only be made to rise with difficulty, or he may be able to raise the hind-quarters, but rests on his knees, and fails to stand on the fore-feet. High fever is present at the onset of the disease. The temperature may reach as high as 106·5° or 107° F., but it declines spontaneously, and on the second day usually falls to 104° F.

Difficult breathing may be observed in some cases, but is not a constant symptom. Loss of appetite and cessation of rumination occur on the first day of illness, but these functions are generally resumed on the second day. In many instances a well-marked jugular pulse is observed, also weakness of the cardiac beats.

Constipation is not an invariable symptom, but for the first two days defæcation may not take place, and in most instances the fæces are dry, lumpy, and covered with strings of mucus. Diarrhœa is not uncommon, while dysentery has been recorded in some cases, also tympanites.

Nervous phenomena have been observed in some cases, such as evidences of vertigo, turning in circles for hours, and finally falling down from exhaustion.

Cases were also met with in which the only symptoms present were those of high fever; stiffness and lameness being absent. In one instance a cow showed, in addition to the high temperature, swollen eyes, a well-marked jugular pulse, and cessation of the secretion of milk.

Bevan observed two sheep apparently affected with a severe cerebral form of the disease.

Course.—In typical cases the disease runs its course in three or four days, but when complications arise the illness may continue for three weeks or longer. According to Cadéac, in rare cases the duration may be reduced to twenty-four hours.

Recovery generally ensues when the appetite returns and the functions of the stomach and intestines are restored, a favourable sign being the passage of fæces of normal consistence.

Mortality.—This is estimated at 2 to 3 per cent., but in some instances it may be as high as 5 per cent. Animals in very high condition, and especially those affected with marked stiffness of the neck, show the greatest mortality; but, according to Bevan, fatal results in such cases are probably due to the careless administration of drenches.

Treatment.—Although a saline purgative followed by febrifuges would seem to be indicated, the dangers of administering fluids, in consequence of the difficulty of swallowing which is often present, must be considered.

Bevan found that animals recovered quite as satisfactorily when no medicines were prescribed; hence attention to the general comfort of the patients and to diet is all that is necessary.

DENGUE IN MAN.

Although there are certain points of resemblance between this disease in animals and in man, there are also important distinctions.

In man it is known as "break-bone" fever, in consequence of the excruciating character of the pain; also as "dandy" fever, from the stiff, dandified gait. The word "dengue" is supposed to be derived from a Spanish, or possibly Hindostani equivalent of the term "dandy."*

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* Osler's "Principles and Practice of Medicine."

The disease in man often assumes a pandemic character, and its diffusion is very rapid. Epidemics have occurred in America along the Gulf States, in the Philippine Islands, and in tropical countries. The causal agent has not been discovered. The points of resemblance between the malady in man and animals are the febrile period, lasting three or four days, the muscular stiffness, and the non-fatal character.

But in man an eruption of a polymorphous character occurs towards the terminal stages in a large number of cases, and may persist for a month after other symptoms have disappeared. There is also an initial erythematous condition of the skin, the fever recurs in paroxysms, and the pains return. Enlargement of the lymph glands is not uncommon, and may persist for weeks after the disappearance of the fever. Convalescence is often protracted, and the pains in the muscles or joints may persist for weeks. The disease is regarded by some authorities as belonging to the group of exanthematous fevers.

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BACTERIAL DISEASES OF FISH

By H. GRAY, M.R.C.V.S.

I. EPIZOÖTIC DISEASE OF FRESH-WATER FISH.

Synonyms.—Salmon disease; Salmon plague; Epizoötic salmon disease; Mycosis, or Integumentary mycosis of fresh-water fishes; Saprolegniosis; Fungous disease.

French: Peste du Saumon.
German: Lachspest; Pestis salmonis.

Definition.—An epizoötic contagious disease of fresh-water fishes, more notably the salmon tribe, attributed to a special cocco-bacillus—*Bacillus salmonis pestis*—which is said to gain an entrance to the system of the fish by abrasions or wounds, and thereby sets up invading necrotic ulcerative lesions of the skin and of the oral mucous membrane, the superficial necrotic parts thus forming a suitable soil for the development of a phycomycete fungus of the family Saprolegniaceæ and of the genus Saprolegnia—*Saprolegnia ferax* or *ferox*.

Species Affected.—It occurs in the Salmonidæ, including, among others, the true or Atlantic salmon (*Salmo salar*), von Behr, brown or brook trout (*S. fario*), Loch Leven trout (*S. levenensis*), Great Lake trout (*S. ferox*), river trout (*S. trutta*), sea trout (*S. eriox*), rainbow trout (*S. irideus*), landlocked salmon (*S. sebago*); in the genus Salvelinus (Char), including, among others, the red-spotted North American brook or fresh-water trout or char (*Salvelinus* [*Salmo*] *fontinalis*); in the genus Coregonus (whitefish or salmonoid fishes), including the vendace of Lochmaben (*Coregonus vandesius*), the pollan (*C. pollan*), the powan or fresh-water herring (*C. clupeoides*); in the Cyprinidæ (carp family), including the common carp (*Cyprinus carpio*), the Crucian carp (*Carassius carassius*, or *vulgaris*), gold or red fish (*C. auratus*), the barbel (*Barbus barbus*, or *vulgaris*), the dace (*Leuciscus leuciscus*), the roach (*Rutilus rutilus*), the perch (*Perca fluviatilus*); the black bass (*Micropterus salmoides* and *M. dolomieu* of the family Centrarchidæ); the tench (*Tinca tinca*, or *vulgaris*), the minnow (*Phoxinus phoxinus*, or *aphya*); the gudgeon (*Gobio gobio*, or *fluviatilis*); the eel (*Anguilla anguilla*, or *vulgaris*); the pike (*Esox lucius*); the grayling (*Thymallus thymallus*); the rudd (*Scardinius erythrophthalmus*), etc.

History and Geographical Distribution.—It has been asserted that it is only during the last hundred years that this disease has attracted attention, but it seems very probable that other pathological entities have been confounded with it until quite recently, when the diseases of fishes have been submitted to the modern methods of investigation.

More than 200 years ago Isaac Walton mentioned that it was not unusual to find dead and dying salmon and sea trout in rivers every season after they had spawned. But there is no evidence to indicate that they were affected by the disease under consideration. The same may be said of the disease that occurred only among haddocks in the Firth of Forth, as described in an *Annual Register* dating back 130 years. Valentin, in 1841, wrote that in fishes, "as the *Cyprinus nasus*, when kept in narrow vessels, and the water not quite sweet, he observed the same fungus [*Saprolegnia ferax* ?] on all the parts which might be abraded, as, for instance, the head and tail." In 1844 Unger observed a destructive disease of carp confined in the tanks of the Botanic Gardens at Gratz. He described and figured the fungus [*S. ferax* ?] to which he attributed the malady. In 1851 Davaine brought before the Société de Biologie a carp suffering from a similar disease. In 1850, 1874, and again in 1879, an enzoötic fungous disease appeared in the roach, pike, perch, dace, and gudgeon in the ponds at Ightham House, Ightham, Kent. It did not seem to affect large pike or perch, and the eel was spared. The fish when in the water had the appearance as if covered with a halo, remaining at the surface nearly motionless, frequently putting their mouths out of the water, and turning belly uppermost immediately before death. The fungus was found thickly matted on the shoulders behind the head, clogging up the gills, also on the pectoral fins and tail portion of the body. The ulcerations were covered with the fungus. Death was caused by suffocation in every instance. In 1852 Crosbie studied a fungous disease affecting salmon in the Tweed. In 1861 Byers saw a similar disease in the Harrison River, one of the tributaries of the Fraser, in British Columbia, where several thousands of salmon were destroyed. John Keast Lord, in his book entitled "The Naturalist in British Columbia," published in 1866, described it as follows: "You will often find salmon with their noses quite worn off, their heads bruised and battered, fins and tail ragged, bodies emaciated, thin and flabby, the bright silvery tints dull and leaden in hue." It was observed in British Columbia again in 1867 and 1868 by those engaged in fish culture, and was thought to be due to injuries arising from handling the fish. The disease that appeared in salmon in 1863 and 1868 in the Grimasto River, in the Island of Lewis, was evidently not the same as the one under consideration. The Duke of Roxburgh and others, in 1870, witnessed a great mortality of salmon in the Tweed, showing similar lesions to those of the outbreak of 1877-1883. He mentioned that ever since he had known the Tweed the disease had always existed there to a more or less marked degree. This statement has been confirmed by others, who previous to 1880 had from time to time encountered the disease for thirty or forty years previously. In 1872, Stoddart of Kelso published a paper on the disease occurring in the Tweed in that year; he had also seen it fourteen or fifteen years before in the Sprouston Water. But the most notable epizoötic was that breaking out in 1877, and continuing to 1883, when hundreds of thousands of salmon, sea trout, fresh-water trout, and other fish, were destroyed. This outbreak began to attract attention in the River Eden, Carlisle, in 1877. Soon after this it appeared in the Tweed, where it remained for several years later. On the East Coast a few cases appeared in the Coquet, but none were noticed in the Wear. It was almost unknown in fresh-run* salmon on the Tyne, where it commonly appears on kelts (spent salmon) and on dace. It did not take on an epizoötic character in this river, nor on the Tees. In the Esk, Yorkshire, about fifty diseased fish were taken out in 1882, but there was no serious epizoötic. From 1879 to 1882 it gradually spread in an epizoötic form from the Tay in North Britain to the South, where it appeared in the Conway, in the Usk, in the Wye, and other rivers, in 1882. In California the disease was rampant in 1880, especially in the tributaries of the Sacromento River, where it has always existed to a more or less extent. In 1877 and 1878, and several years before, the disease was so prevalent and destructive in Canadian rivers that there were fears that the three most valuable food fishes—viz., salmon, shad, and gasperaux—would become totally extinct.

The disease not only occurs periodically to a more or less extent in Great Britain and Ireland, the United States of America, British Columbia, and Canada, but also in France, Germany, Italy, Russia, Northern Asia, etc. It particularly prevails from time to time in the Castries Rivers in Siberia; in Great Britain, in the Tay, North Esk, the Conway, Tweed, Eden, Lune, Coquet, Wear, Teviot, Nith, Annan, Doon, Caldew, etc. It has also been observed in the River Bush, co. Antrim, and in other Irish rivers.

In 1901 and 1902 there was a recrudescence in the Tay, Perth Lade. South Esk, etc.

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* Fish recently entered fresh water from the sea.

Mortality.—This is usually very great, and in consequence the price of such a food fish as the salmon becomes high, and also the sport is lessened. Hundreds of dead fish may be taken out of a river in a very short period.

Period of Incubation.—According to experimental evidence, the period of incubation varies from two or three days to two, three, or more weeks before the disease manifests itself after exposure to infection. The common carp, gold carp, and minnow are not so susceptible as the Salmonidæ.

Time of Year—Occurrence.—It may occur at any time of the year, but it is more prevalent from January to April. It is not uncommon in May and June, and also in September and October.

Etiology.—All the factors considered by the old school of etiologists and sportsmen to play such an important part in the causation of various diseases of man and animals have been invoked to account for this truly contagious disease of fishes. Such are river pollution from factories, sewers, and drainage from lands highly impregnated with artificial and other manures ; over-population of limited areas of water ; insufficient oxygenation of the river water ; damming or barrage (obstruction by weirs) of rivers to prevent fish from ascending the upper reaches, where the water is more suitable for spawning or their nature ; not getting rid of a superabundance of the kelts or spent female fish ; lack or presence of frost or snow ; prolonged drought or wet ; leaving dead fish in the water ; rivers becoming iced over ; injuries inflicted by fish fighting, anglers, handling, whammel or hang-nets, grit, etc. But it has been shown by most conclusive evidence that all these alleged causes may be in play when the disease does not exist, and that when the disease prevails they are often absent. Aquatic birds have been blamed for its transmission from one river to another.

There is strong evidence to indicate that the disease is present at the time of the fish leaving the sea to gain the mouth of the rivers, when it frequently first manifests itself.

Many clear-sighted authorities in the early bacteriological days attributed the malady to some special bacterium, but this view was stoutly opposed by the large majority of those who investigated the great epizoötic extending from 1877 to 1883. Stirling, Sir Robert Christison, Worthington G. Smith, Justin P. Moore, Buckland, Walpole and Young, H. Lee, Rolleston, M. C. Cooke, G. Murray, W. Anderson Smith, J. Clark, and numerous others, contended it was due to the *Saprolegnia ferax*, a fungus of the class Oomycetes, and of the species *diœcious*.

Huxley was at first rather doubtful whether this fungus was the cause or the camp-follower (secondary infection) of the disease ; but in consequence of the strong evidence brought forward by Murray, he succumbed to the general view that the fungus was the active agent. G. Murray, Stirling, and others, were successful in transmitting the disease by means of the fungus, but Patterson has since shown that they could not have used pure cultures, which will not give the malady. The distinguished botanist De Bary, in 1884, when commenting on Huxley's results of his study of the action of the fungus on fish, evidently foresaw the untenability of this general view, for he says: "Disregarding Huxley's results for the moment, we may gather from the statements before us that the fungi in question are ordinary Saprolegniceæ, which must have migrated to the living animal as facultative parasites, since they usually vegetate as saprophytes. If this is so, there must have been some peculiarity in the fishes before they were attacked by the fungus, which is not found in the same fishes in a natural state; there must be some special reason for their being attacked by the Saprolegnia, perhaps a disease of some other kind, which we must not inquire further into here, for the ordinary species of Saprolegnia are so abundant in our streams and lakes that, if they could attack the fish indiscriminately as facultative parasites, not one could possibly be free from the fungus. Direct experiments also have shown me that healthy goldfish may continue lively and free from the fungus for months in water in which Saprolegniceæ, kept purposely in large quantities, were forming an abundance of spores. It would, of course, alter the case if there were distinct parasitic species of Saprolegnia, different from the common ones, but we know of no such species at present. The remarks here made apply, on the whole, to the epizoötic among salmon investigated by Huxley, but some points require further explanation."*

Nearly twenty years later (1903) J. Hume Patterson demonstrated most conclusively that the *Saprolegnia ferax* was not the cause of salmon disease, which was due to the invasion of the tissues of the fish by a special bacillus—*Bacillus salmonis pestis*. This bacillus set up necrotic lesions, on which the fungus freely developed. The peculiar part of this bacillus is that it can be transmitted from diseased dead fish to healthy dead fish in the same water, and that it cannot gain access to the healthy fish's system unless there be an abrasion or ulceration of the skin. If this be so, and the explanation of the cause given by Patterson be correct, it is surprising that during an epizoötic so many fish should receive wounds for the transmission of this bacillus without an explanation being given of their probable origin. Even Stirling, Huxley, and others observed that the disease could not be transmitted unless there were a wound on the skin, which they thought was set up by grit or some mechanical injury. Murray, on the contrary, obtained infection with impure cultures in the absence of a previous injury. It is, however, well known to ichthyo-pathologists, zoologists, and naturalists that fish leaving the sea for rivers often have their skins injured by the presence of various infusorial fish-lice, protozoa (including many species of the families of Myxidiidæ, Chloromyxidæ, and Myxobolidæ), and the larger ectozoa. The injuries received in this manner would ulcerate, and thus form an entrance to the incriminated bacillus.

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* *Vide* Saprolegniosis.

The late Dr. Cobbold considered that parasites played a great part in the mortality from "salmon disease"; but Huxley pooh-poohed this conception, and thought the presence of parasites was more beneficial than hurtful. Assuming the theory to be correct—viz., that the disease only gains an entrance to the system through an abrasion or other breach of continuity of the cutaneous surface, and that such breach in the skin was generally in nature set up by parasites—then Cobbold's views would be justified, as they have been ultimately justified in the case of other diseases.

It is probable that the prevalence of fish diseases during the last forty years is due to the fact that artificial rearing of food and other fishes has increased, and that diseases chiefly developing in young fish reared in hatcheries are spread with their distribution. Introduction of foreign fish into our waters may also account for some mysterious outbreaks. River pollution, although usually not having a direct influence in causing disease, must render fish living in such waters more liable to develop disease when the exciting cause—*causa vera*—comes into play. Wherever man has interfered with Nature's processes without observing her laws carefully and supplying compensations, disease has always become rampant. The history of the progress of the great epizoötic of 1877 to 1883 has shown that it has taken the usual course of epizoötics, and was spread by contagion. A great number of practical observers have stated that this disease usually occurs in various rivers in a sporadic form, but for some unknown reason it has suddenly, after a more or less lengthy period of quiescence, appeared in an epizoötic malignant form, which is characteristic of the majority of contagious maladies. But it is very probable that many different fish diseases have been confounded under one generic term—"salmon disease."

Bacteriology.—According to Patterson, the causal organism of the salmon plague—*Bacillus salmonis pestis*—is a short, thick bacillus, with rounded ends, varying in length, and occurring singly and in pairs, lying end to end. It is actively motile, non-spore-bearing, does not take Gram, grows rapidly and profusely at the room temperature, but shows little or no growth at 37° C., and is killed at this temperature in about six days. The microbe exposed to a mixture of ice and salt for a week not only survived that temperature, but grew profusely in the mixture. Involution forms were only observed in glucose media. It appears to be strictly aërobic. It is only pathogenic to fish. Frogs, mice, and guinea-pigs are refractory. It *rapidly liquefies gelatine,* on which it grows profusely. It grows well on agar, agar-glucose (in which it forms a cloudiness in the neighbourhood of the growth), blood-serum, bouillon, and glucose bouillon. It gives a distinct acid reaction in litmus milk, which is coagulated, and then gradually digested in about seven days. In peptone water it produces a diffuse marked cloudiness, but gives no indol reaction. On potato it gives a very profuse yellowish-brown growth at the end of eighteen hours, which is raised on the surface of the media like blisters, with a moist, shining surface. On anaërobic agar no growth is observed. The bacillus withstands the effect of ordinary water, sterile water, and *sea-water* for a considerable time. It does not, however, survive more than a week in distilled water. It keeps well on subcultures, as tubes of agar inoculated from subcultures of about a year old give profuse growths in about eighteen hours.

The specific organism is capable of infecting a normal dead fish—that is to say, a diseased dead fish may infect a normal dead fish in running water. The disease is not, according to Patterson, transmitted to a healthy fish except after the infliction of a wound, "or perhaps by a degenerated mucous gland in the sickly fish." Healthy fish in a tank into which a culture of the bacillus is poured do not contract the disease; but if the fish are previously wounded, or the water contain grit, the disease manifests itself in two or more days by the appearance of the fungus. Healthy fish thrive in tanks containing profuse growths of pure cultures of the *Saprolegnia ferax* on boiled sheep's liver or on healthy dead fish; but before they can develop the disease the specific bacillus *plus* an abrasion must be present. The bacillus breaks down the superficial tissues, and forms a suitable nidus in the necrotic parts for the fungus to grow on. Saprolegnia disappear when the fish are put in salt water.

Lesions.—Beyond the lesions on the surface of the body, the liver may be occasionally found to be soft and enlarged, and the brain rarely congested. The abdominal organs, muscles, deep-seated bones, and blood may appear absolutely healthy, even in the case of very extensive disease. The scales are covered more or less by patches of a fungus, resembling when dry, moist brown paper. If the fish is placed in water, the fibres of the fungus will separate one from the other, and the appearance presented will be that of very delicate cotton-wool. There is hardly a part of the body that is not likely to be attacked, but usually the head is the first part affected, then the tail and other regions. The lesions are essentially necrotic ones, extending more or less deeply into the softer tissues even down to the bone, especially of the head and fins. The dead parts are covered with the fungus, which, however, does not extend into, but stops short at, the living tissues. The bacilli are found in large numbers outside and invading the partly destroyed epidermis, passing into the softer cellular tissue underneath, and in considerable numbers boring their way along the healthy skin between the softer layer of the epidermis and the dermis. They are also found in large numbers passing through the dermis into the lymphatic spaces between the muscle fibres. During their progress they set up marked necrosis, and where this lesion is very marked in the muscle fibres the microbe can be seen in large numbers invading the muscle itself. In the snout and fin a similar appearance is quite marked. No bacilli are found in the heart, spleen, or liver, but a considerable number are observed in the cavity of the stomach, but not invading its walls; they have not been encountered in healthy parts of living fish, or of those dead of ordinary causes.

Symptoms.—The disease first appears in the form of small fluffy, whitish patches, resembling very delicate cotton-wool or swan's down, on the scaleless portions of the skin, notably on the top and sides of the head, giving rise to the term "salmon with white nightcaps," or at the end of the snout; but the adipose fin and the soft skin at the bases or axillæ of the paired fins are also among the first parts to be affected. The patch may be as large as a sixpence to a half-crown, almost circular in outline, having a well-defined margin and a somewhat raised softer centre, from which faint ridges radiate towards the circumference. In severe cases the patch rapidly spreads and runs into other patches which may have appeared in its vicinity, until almost the whole body is covered with an ashen-grey, fluffy coat, which completely overlaps or masks the underlying scales, and then gives the fish, when in the water, an appearance like sheep covered with wool. The patches may be few in number or very numerous, up to twenty, forty, or more, and extend over various parts of the surface of the body. On brushing off the crust or coating of fungus, the scales are usually found intact. The central part, however, takes on the consistency of wet brown paper, and can be removed in flakes, like a croupous membrane from the true skin which it covers. In mild cases, especially in the earlier stage, the patch of fungus may be easily wiped off, leaving only a slight stain, and no loosening or desquamation of scales or ulceration of the subsequent surface. When the fungus is prolific, deep-seated, and felted, the dermis beneath the slough is deeply ulcerated, leaving an open, bleeding ulcer, which may extend down to the bone, while it opens outwards in burrowing sinuses. On the head the cranial bones may be exposed, and the fins are eaten away and become ragged, the fin rays standing apart from one another. Very often one or both gill-covers become partially adherent to the shoulder and the gill openings obstructed. The gills themselves rarely become involved, but the disease frequently invades the oral cavity, and often more or less destroys the membranous veil which lies behind the teeth and plays a peculiar part in respiration. The respiratory organs, however, are not usually attacked directly, but respiration may be seriously impeded by a profuse growth of the fungus, which forms a deposit not unlike glue.

Although the disease usually appears at first in the scaleless portions of skin, it may gradually involve the whole of the back and sides, and even the ventral surface, of the fish.

When the disease has reached the sloughing or ulcerative stage, the fish appear to suffer from severe irritation and pain, evidenced by spasmodically dashing or throwing themselves about, and rubbing themselves against obstacles. Probably this irritation is due to the innumerable particles of sand getting into the wounds, as when fish suffering from an advanced stage of the disease are kept in an aquarium free from grains of sand this evidence of irritation is not manifested. When they get to this advanced state, the fish become exhausted, or weak and sluggish, seeking shallow spots near the banks of the river, where they ultimately succumb.

Some think death is due to the Saprolegnia hiding the light and preventing endosmosis and exosmosis of the skin, or is caused by suffocation. Others believe it is the result of a toxin formed by the fungus and absorbed into the system. There is, however, no evidence to support any of these views.

Diagnosis.—This disease is likely to be mistaken for other diseases in which various species of fungi of the family Saprolegniaceæ are often encountered accompanying them. It is only by bacteriological examination that it can possibly be distinguished from them.

Various species of the Saprolegnia and Achlya are encountered on fish confined to tanks, aquaria, or fish-ponds, but not enough is known of the diseases with which they are associated to enable one to differentiate them from the epizoötic salmon disease.

It cannot well be mistaken for *furunculosis*, an ulcerative disease due to a pigment-forming bacillus—*Bacterium salmonicida*—which is destroyed by salt-water. Other fish diseases, as lepidorthosis contagiosa, purpura contagiosa, morbus maculosis, ulcerative septicæmia, xanthosis leuciscorum, and many other ulcerative affections due to Myxosporidia and other sporozoa, are not usually accompanied by fungus deposits on the surface of the body.

The enzoötic disease which appeared in 1868 in the salmon of the Grimasto River, in the Island of Lewis, although manifested by more or less superficial ulcers with everted edges, was not accompanied by any fungous appearance.

Prognosis.—During malignant epizoötics the mortality is tremendous, and in some rivers the majority of fish are destroyed. In other rivers the death-rate appears to be low. It is usually a fatal disease, especially in young Salmonidæ.

Treatment.—Whatever curative means may be adopted to overcome this disease in fish confined in tanks or aquaria, no therapeutical measures can be put into practice when fish are free in rivers or lakes. Some sportsmen and naturalists speak highly of immersing them from time to time in salt water, others by putting them into tanks fed by a continuous running of oxygenated water. But although the fungus may be destroyed by such means, the disease produced by the primary infection still remains to be a source of danger to fish not already affected. It is very probable that those who had successful results by that treatment were dealing with a totally different malady to the epizoötic that decimated the fish during the period between 1877 and 1883.

The preventive measures against contagious affections of fish are almost impracticable in large streams, unless a disease, when confined to a particular stream, is stamped out by destruction of all the fish in that stream before it has had time to spread to other watercourses. Some have advised netting the fish at the mouth of the rivers as soon as they leave the sea, and before they have had time to ascend the higher reaches of the river, destroying and burning or deeply burying all those that show evidence of disease, and allowing those which appear healthy to go up the river. This method is not of much use, as apparently healthy fish may have the infection in their systems, to develop after they have passed inspection.

All dead fish should be taken out of the watercourses and burnt or buried deeply some distance from the banks of the river. River pollution with organic matter should be prohibited, as, although it may not produce disease in itself, it must lower the vitality of the fish, and thus render them susceptible to infection. Predatory birds, fishes, and animals such as the otter, should be encouraged to multiply, and not be shot at sight on the mistaken notion that they are enemies of fishes, and therefore to mankind. By their presence they kill off and eat up all weakly, diseased, or dead fish, and thus maintain a balance of nature which is beneficial to the surviving fish. They also prevent overstocking. Supposing they do destroy healthy fish, the amount they consume is out of all proportion to those lost by disease in consequence of the balance of nature being destroyed.

As to destroying water-birds in order to prevent them distributing the disease from one stream to another, the remedy would be worse than the disease, as such birds must necessarily destroy a large amount of sickly, dying, or dead fish.

Public Health.—Diseased salmon and other fish, when cooked, cannot be distinguished by taste or effect from that of perfectly healthy salmon. Numerous expert authorities have consumed it without detecting any difference or suffering from ill-effects. It seems to be a foolish policy to condemn such fish for human food, especially when salmon is usually at a high price. But where sentiment reigns, reason must succumb. Cooked salmon, although diseased, is preferential to uncooked oysters, which not rarely contain the germs of typhoid, and so often prove fatal to those partaking of such shellfish.

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II. FURUNCULOSIS OF THE SALMONIDÆ.

Synonyms.—*French:* La furonculose des salmonidés.
German: Die Furunkulose der Salmoniden.

This disease has been observed in Germany, especially in Bavaria, France, United States, and England, in the von Behr, brown or brook trout (*Salmo fario*); in the North American red-spotted brook or fresh-water trout or char (*Salvelinus* [*Salmo*] *fontinalis*); in the peal or sea trout (*Salmo eriox*); in the salmon (*S. salar*); and in the grayling (*Thymallus thymallus*).

The tench (*Tinca tinca*, or *vulgaris*), eel (*Anguilla anguilla*), pike (*Esox lucius*), chub (*Leuciscus cephalus*), and dace (*L. leuciscus*), and other fish, may also be the victims of its invasion.

During the months from May to August, 1911, the disease appeared in salmon, sea trout (peal), grayling, and even in the eel, chub, pike, and dace, in certain rivers in the South-West of England—namely, the Wye, Exe, Teign, and Dart. This epizoötic was mainly ascribed to the extreme drought and high temperature which prevailed generally throughout the late spring and early summer.

It was first noticed in the Exe on May 12, and was thought to have been introduced by a certain definite run of salmon in which the mortality first appeared, and afterwards spread to brown trout and grayling. It was first observed in the Teign on June 2, and seemed to abate about July 29. During this period dead salmon, sea trout (peal), and brown trout were found, and a ten days' rain prevented the occurrence for a time. In this river there was no indication that the salmon were first affected, salmon, sea trout, and brown trout being found together. On July 8 the disease was first noticed in the Dart; all three of the just-mentioned fish were simultaneously attacked, though it was stated that the trout became affected soon after its appearance in salmon. In the Wye, it was remarked that the disease was for the first time noted in April, but the actual epizoötic appears to have been dated from May 25, and continued up to the end of July. All kinds of fish seem to have been found dead, including salmon, trout, grayling, eels, pike, chub, and dace. The different epizoötics had a duration varying from seven to ten weeks.

Bacteriology.—According to Emmerich and Weibel, Hofer, Marianne Plehn, and de Bouville, the disease is due to a special organism, named by Emmerich and Weibel *Bacillus salmonicida*—a short, Gram-negative, immotile bacillus, which does not form spores. It forms a slight late brown pigment in agar, and gives no growth on potato, at a temperature of 37° C. It is also a facultative anaërobe, and liquefies gelatine. Plehn states that this medium becomes brown in ten days. Marsh, in America, describes a bacterium (*Bacterium truttæ*) occurring in an epizoötic amongst trout in capitivity, but he has not found the bacillus in trout at freedom. This organism is a short, Gram-negative, sporeless, immotile bacillus, which forms a brown diffusion pigment in agar, gives a very slight growth on potato, liquefies serum a deep brown colour, gives an acid reaction in glucose and in milk, which is digested, does not develop at 37° C., is slightly a facultative anaërobe, and liquefies gelatine. The pigment is soluble in alcohol. Arkwright found a bacillus in the 1911 English epizoötic having nearly all the characters of the *Bacillus salmonicida* and the *Bacterium truttæ*. Like these two, it is a short, Gram-negative, immotile, sporeless organism, forming a brown, diffusing pigment in agar, and liquefying gelatine. It gives a very slight growth on potato, liquefies serum a deep brown colour, gives an acid gaseous reaction on glucose and mannite, turns milk acid and digests it, develops very slightly at 37° C., remains alive in sea-water only a few hours, and is a facultative anaërobe. Marianne Plehn made in 1911 a fresh study of the *Bacillus salmonicida* in an epizoötic of furunculosis of the Salmonidæ which occurred in Germany in 1909. From her report it appears almost certain that the *Bacillus salmonicida* is identical with the *Bacterium truttæ* and the bacillus isolated and studied by Arkwright in the English outbreak of 1911.

This pigment-forming and Gram-negative bacillus appears in young cultures either in a round, oval, or oblong form, the long axis being equal to, or at most twice as long as, the short axis. In older cultures it may form long bacilli, whose length is four or five times longer than their breadth. The diameter of the coccal forms was about 1 μ or rather less. When stained with Löffler's blue, or carbothionin blue, or with Leishman's stain, the bacillary forms showed a tendency to bipolar staining. The bacilli rapidly die in sea-water. The bacillus is constantly, and often in almost pure cultures, found in the blood, liver, spleen, peritoneal fluid, and in deep red spots in the muscles, and in other tissues. In the tissues and blood they are uniform in shape, about 1 to 1·5 μ in length by 1 μ in breadth, and often found in dense clumps.

The disease may be transmitted by intradermal, intramuscular, and intraperitoneal inoculations; also by ingestion of pure cultures poured into the water containing fish. It is very contagious.

A bacillus of the Proteus group, and another corresponding in its cultural character to the *Bacillus fluorescens liquefaciens*, are found in the pus of discharging abscesses, mucus from the vent, and the intestinal contents.

Saprolegnia appeared on several salmon during the month of September.

Etiology.—This epizoötic disease is far from rare, and seems to be somewhat widespread in its distribution. It occurs not only when fish are confined to fish-ponds, aquaria, and tanks, but also when they have their freedom in a natural state. It is very contagious, and spreads in a manner similar to many other epizoötics. Affected fish ascending a river communicate the disease to other fish with which they come in contact during their migrations. When confined in artificial ponds and tanks, they also transmit it to their healthy neighbours. Prolonged droughts and high summer temperature, with a lowering of waters, apparently depress the vitality of fish, and thus render them an easy prey to microbial infection. Probably these factors also increase the activity and virulence of certain saprophytes existing in rivers and fish-ponds, which would under ordinary circumstances be quite harmless. To what extent a high temperature plays in this direction is not known. Some fish, like the whiting, may thrive in a tank of water up to 79° F.; beyond this they sicken and die; others, like trout, thrive in a temperature up to 85° F.; and carp show no distress below a temperature of 120° F. High temperature certainly deoxygenates water continuously exposed to the sun's rays. This is more marked when the water-mark is low, and the stream in consequence is more sluggish. Luxurious plant-life is beneficial to water by taking up the carbonic acid and giving off oxygen. Waters polluted with sewage and other organic waste, especially the wasted food that accumulates at the bottom of fish-tanks and is allowed to decompose, are alleged to be a fruitful source of the malady.

The *Bacillus salmonicida*, which is rarely encountered in healthy fish or fish coming from healthy stock, gains an entrance to the system chiefly through the alimentary canal, and more rarely by the skin or the respiratory organs. Susceptibility plays some part in its virulence. Healthy, vigorous fish reared under the best hygienic conditions are more resistant than those reared under adverse conditions. Some species are more susceptible than others—for instance, salmon more than trout, and trout more than carp, eels, tench, dace, and chub.

It is not known if fish just leaving the sea, and losing their ectozoa parasites on entering fresh water, have cutaneous abrasions or wounds remaining after the parasites have escaped, by which the infection may gain the system, and thus account for the disease appearing soon after they have commenced to ascend rivers.

Symptoms.—The period of incubation is said to be from two to three weeks. In acute infection arising during the early part of an epizoötic a great number of fish only show a loss of power to move in a forward direction and to maintain their equilibrium by turning on one of their sides, and gradually becoming immobile before they expire, which occurs somewhat rapidly after the first symptoms. In this case very few external lesions are observed. In others a few abrasions or punctures, probably resulting from ectozoa, are encountered on the skin. In a few instances there may be a slight discharge of blood from the vent.

In those cases not running such an acute course ecchymotic and raised spots, varying in size from a grain of millet to that of a hempseed, pea, or even nut, appear on the skin. These spots vary in number from three to five, eight, and even fourteen, and are usually situated on the lateral surfaces of the lighter coloured zone of the skin ; they may sometimes be seen on the head or other parts. They are mostly discrete, but a few now and then run into one another, forming larger patches. Their superficial covering, gradually losing its reddish colour and acquiring a yellowish or pinkish tint, become necrotic, and then burst, discharging at first a yellowish, and afterwards a thin reddish (carnivorous-like) pus, leaving rather deep ulcers, with a yellowish-red or pinkish centre, and a deeper red irregular or ragged outline.

Lesions.—In a great number of instances there are no external lesions ; in others there are a few small punctured wounds or slight abrasions, probably left by the usual ectozoal parasites becoming detached from their host on entering fresh water.

In others the skin shows lesions, as described under Symptoms. The deep ulcers sometimes resemble abscess cavities, and occasionally have one or more small openings in the skin, or they may be completely closed. Again, the ulcers may extend towards the bones of the ribs or fins, which are thus exposed. They may even penetrate into the abdomen or thorax. In other cases the small ecchymotic spots become softened and leave ulcers, with a greyish centre and a deeper red periphery; below the floor of the ulcer the tissues have a deeper pinkish or red hue, dotted with one or more small ecchymotic spots. They may be found in any part of the subcutaneous and muscular tissues.

The *liver* is occasionally mottled with different tints, and sometimes softened. The spleen not rarely is intensely hæmorrhagic and soft, and appears like a dark blood-clot. Under the microscope the liver, kidney, and affected muscular tissue show patchy necrosis and disintegration of the tissue cells, but no increase in the leucocytes or connective-tissue cells. The bloodvessels and capillaries are crowded with the pigment-forming bacillus. The intestines are frequently congested, in some cases to a very great degree, but this lesion is unevenly distributed; sometimes it is in one part of the intestinal tube, and in others in the pylorus and pyloric cæca. On pressure of the abdomen bloodstained material may be discharged from the vent in many instances. It should be mentioned here that healthy dead fish not rarely show a congested appearance of the intestinal tube, and may also occasionally discharge a reddish material from the vent.

Various entozoa and ectozoa, not excluding protozoal parasites, are often encountered in fish, even when otherwise absolutely healthy. In this respect fish resemble the mammalia and birds, which are only exceptionally free from parasites even when they are in the best of health. Hence the presence of parasites is not always evidence of a diseased condition.

Diagnosis.—This disease somewhat resembles myxoboliasis tuberosa, due to the *Myxobolus Pfeifferi*, but in that affection there are several large external tumours, which ultimately discharge pus and leave large crateriform ulcers, while the pus contains besides bacilli, spores of the myxobolus. It also resembles the epizoötic disease of fresh-water fishes, commonly known as the "salmon disease," but in that disease ulcers generally first appear on the scaleless parts of the skin, notably that on the head, and are usually covered with a saprolegnial fungus; it is, moreover, due to a motile, non-pigment-forming bacillus, which can thrive in salt water, whereas the *Bacillus salmonicida* is destroyed by *salt water*. It may be confounded with some forms of saprolegniosis when fungi form on the ulcerous patches.

Prognosis.—This is always unfavourable. The mortality is generally great.

Treatment.—As there does not appear to be any curative treatment, prevention should be aimed at. In order to fulfil this object it might be advisable to form a double wire-netting barrier across narrow non-navigable rivers as soon as the disease is discovered, to prevent the diseased fish further ascending the river and so contaminating healthy ones farther up the stream. The affected fish might also be netted, and either placed in salt water or taken back to the sea. River pollution should be strictly guarded against. All dead fish should be taken out of the river and burnt or buried deeply in quicklime some distance from watercourses.

The tanks or aquaria in which fish are confined or reared should be kept clean by occasionally evacuating them, and then cleansing and ultimately disinfecting them before the fish are returned to them. The remains of food should not be allowed to accumulate on the bottom of the tanks. Should the disease break out, the diseased fish should either be destroyed or isolated in salt-water tanks, and the apparently healthy put into fresh tanks. The infected tanks or fish-ponds should be thoroughly cleansed, and then disinfected with permanganate of potassium or a milky solution of freshly slaked lime. These disinfectants should be allowed to remain in the tanks for a week or a fortnight.

Public Health.—Although fish dead of this disease are not injurious to man when consumed by him, they should not be exposed in a public market for human consumption. The *Bacillus salmonicida* is not pathogenic to mammalia. Owners or lessees of running streams should be compelled by law to speedily remove dead fish from the water and bury them some distance from the watercourse.

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III. SPOTTED DISEASE OF THE RED-SPOTTED TROUT.

Synonyms.—Ulcerative disease of trout.

French : Maladies des taches du saumon de fontaine.

German : Fleckenkrankheit des Bachsaiblings ; Morbus maculosus.

The red-spotted or North American brook trout (*Salvelinus* [*salmo*] *fontinalis*) is very often attacked during the spawning season by an ulcerative disease of the skin, especially along the back in front and behind, and also below the dorsal fin, which is not as a rule involved in the morbid process. The epidermis is detached in elongated patches, somewhat oval in shape, and very irregular or rugged in outline, the long axis taking the direction of the long axis of the body. These patches are soon covered by one of the Saprolegnia. They are usually accompanied by acute enteritis, which is not long in destroying the affected fish.

Nothing is known of the cause of the disease beyond that it seems associated with pollution of the water with the excrements of the fish and the remains of their food, which accumulate at the bottom of the tanks.

It is a very fatal disease, and greatly feared by pisciculturists.

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IV. ULCERATIVE DISEASE OF SALMON.

In 1868 a disease appeared on salmon in the Grimasto River, in the Island of Lewis. The affection was confined to the head. The whole of the upper part of the head was found covered with ecchymosed spots and ulcerations, the ulcers being more or less superficial with everted edges. In some instances the nasal cartilages were also involved, appearing as if one side had been cut out by a corroding sore. When cut into, the bones and cartilages were soft, and the brain and meninges showed signs of inflammation. The gills were pallid, but otherwise sound. None of the fins were affected. In those cases that recovered the gill-covers had a dull white, leprous appearance, and in all the fish the head was more or less white, and remained so for years afterwards. There was no trace of fungus in any case. It did not attack the brown trout or the sea trout. It was thought that the disease was contracted in the sea before the salmon gained the river. The condition of the diseased fish was good, the body being plump, fat, and pink in colour. The flavour of the cooked fish did not differ from that of a healthy one. This malady destroyed a great number of salmon, and evidently it differed from the epizoötic salmon disease described on p. 946. In the same river, after a prolonged drought, in 1863 some 1,500 fish were picked up on the shores of the bay and mouth of the river.

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V. INFECTION IN FISH.

Charrin observed in September, 1892, an epizoötic that decimated the fish in one branch of the River Rhône. He isolated several microbes, but only one of them was pathogenic. This bacillus was cultivable on the majority of the usual culture media.

On agar it determined a thin layer, having a bluish iridescent reflection ; sometimes around a colony some bacterial trails were disposed in the form of a cart-wheel. It liquefied gelatine somewhat slowly. When deposited on the surface, it produced a liquid cone, having its summit inferiorly situated. On potato a salient, mammellated, moist, yellowish or brownish zone promptly arose at the points where the organism was inseminated. Milk was coagulated in forty-eight to seventy-two hours, and gave an acid reaction. Bouillon was rendered fairly uniformly opalescent. Cultures gave off a marked odour of trimethylamine. Growth was rapid when fully exposed to the atmosphere, and only showed a more feeble vitality under a layer of oil. It is motile, measures about 2 μ in length by 0·8 μ in breadth. Its growth is not impeded at 33° C., but still very active at 20° C. Inoculated into carp or barbel, it gives rise to hæmorrhage, and generally death. These hæmorrhages are either found in the muscles or in the scales. Its products, which are insoluble in alcohol, act in the same manner. However, large doses of either the cultures or the toxins—that is, 1 c.c. of the first for an animal of 200 grammes, 6 c.c. of the second—are necessary. Charrin was not successful in exalting the virulence of the virus. Its virulence was not great for the frog, guinea-pig, or rabbit, which required 3 to 4 c.c. to produce fatal results. When 3 grammes of the virus was introduced into a glass globe containing 4 litres of water and ten bleak, seven of these fish died in less than three weeks ; whereas in another jar containing the same quantity of water and the same number of fish, but no virus, only two fish succumbed, and these did not contain the bacillus, which was found in the blood and muscles of the fish in the infected glass globe.

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VI. STAPHYLOCOCCAL EPIZOÖTIC IN GUDGEON.

In September, 1893, Charrin observed an epizoötic in gudgeon in the River Rhône, near Lyons. In one day he collected forty-one, and the next day seventeen dead fish, which were without exception gudgeon.

The external surface of the anterior third of the body was slightly œdematous, and on post-mortem examination there was a small quantity of effusion into the abdominal cavity.

In eleven instances he inseminated agar with the œdematous material, and in every case he obtained the same microbe, which was spherical, aërobic, and easily cultivable in the various culture media. In ox bouillon it gave a uniform layer of a yellow-grey colour, which was intense on the fourth day, if the tubes were placed in an incubator maintained at 37° C. On agar a whitish layer, which as time went on become of a more or less golden colour, developed. This layer was salient and moderately moist; the pigmentation was rapidly or extremely slow in forming, according to the manner in which the agar was made; it was either of a moderate intensity, or almost nil, and it quickly disappeared. Gelatine was not slow in liquefying under the influence of the growth of this organism. On potato it formed a yellow coating.

Gudgeon inoculated with cultures developed the disease. The rabbit was destroyed, but large doses (2 c.c.) were injected. Pure cultures poured into water also gave the gudgeon the disease.

Charrin considers his organism to be none other than the *Staphylococcus* or *Micrococcus pyogenes aureus.* The *Staphylococcus pyogenes aureus* derived from a case of osteomyelitis appeared at first sight to be inoffensive for the guinea-pig, but by the aid of an exalted culture positive results were obtained.

In the same year an epizoötic having similar characters appeared in the Seine, in the vicinity of Paris. It was attributed to the dry season.

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VII. ULCEROUS SEPTICÆMIA OF GOLDFISH.

Synonyms.—*French :* La Septicémie ulcereuse du poisson rouge.
German : Die Ulcerativen Septikämie des Goldfisches.

Ceresole has described an ulcerative septicæmia in the goldfish (*Carassius auratus*) confined in aquaria. It is due to a special bacillus, which is pathogenic to goldfish and to the rabbit. This bacillus somewhat resembles in gelatine cultures that of anthrax. It varies from 2 to 2·5 μ in length by 0·8 to 0·9 μ in breadth.

This bacillus is found in the discharges of the ulcers, and reproduces the disease when inoculated into healthy fish. It is a very contagious malady.

The disease is characterised by the presence of ulcers on the upper and posterior part of the head.

In the case of an enzoötic the tanks in which the fish are contained should be evacuated, cleansed, and disinfected, and the apparently healthy fish isolated, and the dead ones burnt.

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VIII. RED PLAGUE OF THE CYPRINIDÆ.

Synonyms.—*French :* La peste rouge cyprins.
German : Die Rotseuche der Karpfenartigen fische ; Purpura Cyprinarum.

Under this name the Germans describe a disease which destroys a prodigious number of carp and tench in tanks or in winter ponds. It receives this denomination from the fact of the affected fish showing on the belly and sides a more or less, sometimes very slight, red discoloration of the skin, which corresponds to hæmorrhagic effusion into the skin or subcutis.

The fish do not appear to suffer pain, but rise towards the surface of the water, where they seem immobile, and die, on an average, about the tenth day of their seizure.

It is caused by a special bacillus—*Bacterium cyprinicida* (Plehn)—which is 1 μ in length by 0·8 μ in breadth. It is aërobic, and grows on the usual culture media. It is found in all the organs, especially the kidneys, and in the blood. The disease is witnessed where the water is polluted with putrefying organic material.

The prevention of contamination of water is the best means of giving assurance against this enzoötic.

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IX. THE RED PLAGUE OF EELS.

Synonyms.—*French :* La peste rouge de l'anguille.
German : Die Rotseuche des Aals; Pestis Rubra Anguillarum.
Italian : Peste rosse delle anguille.

Epizoötics of eel diseases have frequently appeared, more notably in Italy, to a lesser degree in Herzegovina, and occasionally in Holland, Germany, and Denmark.

Giän Francesco Bonaveri witnessed a fatal malady in eels in the year 1718, when thousands of eels in the lagoons of Comacchio were destroyed. In 1790 a similar outbreak in the same place was observed by Spallanzani. It reappeared in Italy again in 1825, 1850, 1864, 1867, 1884, 1885, 1889, 1892, 1893, and 1901. In 1898 and 1899 it was observed in Herzegovina. In 1901 it appeared in the Ortobello, in Tuscany.

It is an intensely contagious disease, and causes a great mortality. It occurs during very hot weather, and is determined by a bacterium—*Bacillus pestis rubræ.* The bacillus found by Canestrini in the liver of diseased eels, and named by him the *B. anguillarum*, is from 1·5 to 2·4 μ long and 1 μ broad. It somewhat resembles the cholera bacillus. It is pathogenic to eels, sticklebacks, goldfish, frogs, and salamanders. Inghilleri and Gosio found a bacillus in the Ortobello red plague that was pathogenic to the guinea-pig, rabbit, and white rat; it was 2 to 3 μ in length by 0·4 to 0·5 μ in breadth, facultative anaërobic, and did not take Gram. Canestrini's microbe stained by Gram's method, and was exquisitely aërobic.

The fins are injected with blood, and reddish, somewhat diffuse patches appear on the skin of the belly, sides, and head. The sick fish rise towards the surface of the water, and soon die. Putrefaction sets in almost immediately after death.

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X. LEPIDORTHOSIS OF WHITEFISH.

Synonyms.—*French:* La lépidorthose des poissons blancs.
German: Die Schuppensträubung der Weissfische; Lepidorthosis contagiosa.

This disease attacks chub (*Leuciscus cephalus*), dace (*L. leuciscus*), roach (*R. rutilus*), rudd (*Scardinius erythrophthalmus*), common or carp bream (*Abramis brama*), vendace (*Coregonus*), *Idus melanotus*, carp (*Cyprinus carpio*), etc.

It is caused by a bacillus—*Bacillus pestis astaci*—which is also the cause of great epizoötics of disease in crayfish. When this disease occurs in this crustacean, the French have denominated it *La peste des écrévisses*, and the Germans *Krebspest.*

It has been especially noticed in France, Germany, Italy, and Russia. The small bacillus is from 1 to 1·5 μ in length by 0·25 μ in breadth. It grows on the usual culture media. It is found in the blood, exudates, and organs. It is pathogenic from fish to crayfish and the frog, and reciprocally.

It is characterised by a swelling and standing out or opening of the scales over the whole or on the posterior part of the surface of the skin, due to an accumulation of a clear fluid or exudate in the scale covering. There is also an ecchymosis on the parts attacked. The affected fish do not appear to suffer, but they finally lose their activity, become weak, and then die.

The causal organism seems always to penetrate the system through lesions in the skin, so that in order to prevent this disease care should be taken to remove any cause likely to wound the fish, and also to keep the ponds and tanks as clean as possible.

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XI. YELLOW PLAGUE OF ROACH.

Synonyms.—*French :* La peste jaune du gardon.
German : Die Gelbseuche der Rotaugen ; Xanthosis Leuciscorum.

Oscar Wyss studied an epizoötic that decimated the roach (*Rutilus rutilus*) in the Lake of Zurich in the months of July and August, 1897, and isolated a very common bacillus, designated *Bacillus vulgaris* (*Proteus*).

The body was covered with slightly salient yellow spots of 2 to 3·5 centimetres in diameter. There were also some ecchymoses. The fish lost their vigour, ceased to swim, and succumbed. In the blood of the dead fish the bacilli were found in abundance.

The disease was attributed to the exceptionally hot weather, the high temperature of which so depressed the system of the fish that the microorganism invaded and grew rapidly in it. Probably the microbe also increased in virulence in consequence of the high temperature.

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XII. TYPHUS OF THE PERCH.

In 1867, 1870, and 1873, an enzoötic practically decimated the perch in Lake Leman until there were few fish left to catch, so that angling had to be abandoned. The disease ran its course in about ten days, and was manifested by the fish floating towards the surface of the water and quickly dying. They showed blood effusions at the base of the fins, and whitish spots on the skin. It was attributed to pollution of the lake with factory waste. Nothing is known of its pathology.

XIII. COLIBACILLOSIS OF THE ALOSA FINTA.

A variety of the *Alosa finta* (*A. finta* var. *lacustris*), a fresh-water non-migratory herring, confined to the Lake of Lugano, between Switzerland and Italy, is frequently decimated by an enzoötic. During the winter of 1901-02 a million of these fish perished from an acute inflammatory disease, set up by the *Bacillus coli communis*. It was supposed that the fish, which are fond of frequenting the mouths of sewers, were contaminated by the ingestion of human and animal dejecta poured into the lake from the sewers.

XIV. TUBERCULOSIS OF CARP.

In 1897, Bataillon, Dubard, and Terre observed carp in a stream of a fish-culture establishment die presenting large tumours on their flanks. They found in these tumours numerous bacilli of tuberculosis. The stream was contaminated by phthisical people expectorating into the water, and the disease was coincidental with this action. Nicolas and Lesieur gave five carp and eight goldfish no other nourishment but tubercular sputum for several months, and although these fish succumbed to a diffuse tubercular infection, they did not develop any tubercles or tumours.

From these facts it would seem advisable not to give fish either milk or meat, especially that coming from tubercular cows, unless previously boiled or cooked.

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XV. OTHER FISH INFECTIONS.

Babes and Riegler have described an epizoötic in carp (*Cyprinus carpio*), Crucian carp (*Carassius carassius*, or *vulgaris*), tench (*Tinca tinca*, or *vulgaris*), pike (*Esox lucius*), and roach (*Rutilus rutilus*), in May, 1902, in Bucharest. These authorities considered it to be caused by a bacillus, which they termed *Proteus piscicidus versicolor*.

Fischel and Enoch studied an enzoötic in carp, due to a bacillus, which they termed *Bacillus piscicidus*. This bacillus was 1·2 to 3 μ in length by 0·25 μ in breadth. It did not take Gram, but was pathogenic to the mouse, guinea-pig, dog, and pigeon.

Madame N. Sieber studied in St. Petersburg an enzoötic in pike-perches (*Lucioperca sandra*). She isolated a bacillus to which she gave the name *Bacillus piscicidus agilis*.

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THE QUAIL DISEASE

By H. GRAY, M.R.C.V.S.

Definition.—The quail disease is an acute virulent infectious disease affecting various species of quail, and said to be due to a bacterium of the coli group. It is characterised by acute congestion of the lungs, necrotic foci in the liver, and intestinal ulceration.

History.—This disease was first observed in April, 1906, in a bird-dealer's establishment in Washington, U.S.A., a large number of birds gradually dying off; many of which seemed well in the evening were found dead in the morning. Some time later this dealer received twenty-four blue quails from Wichita, Kans., when on arrival they were nearly all found to be dead. The year previous the same dealer received great numbers from Birmingham, Ala., the mortality then being 100 per cent. In May, 1906, the disease was present in Boston, Mass. In January, 1907, it affected the ruffed grouse in Worcester, Mass.; the stock birds had been obtained from Alabama. A month later the Washington dealer received from Alexander City, Ala., sixty-five birds, of which thirty-four were dead; twelve days later fifteen other birds were dead. These quail had been for several months on the same place, and had appeared perfectly healthy. The first lot had been brought in contact with these acclimatised ones, and ten days later the disease appeared in them, and in two days all were dead. During the same month, February, five dozen birds arrived from Alexander City to replace the previous lot, and on arrival two were found dead. The remainder were sent to the Laboratory of Pathology, Bureau of Animal Industry, and within eight days all but three were dead. Still during the same month another Washington dealer received fifty-three bobwhites from Indian Territory, but they came via Wichita, Kans. There was uncertainty as to whether hese were recrated at Wichita. On arrival twelve were dead, and four died later on, and ultimately four others. Another consignment from Marlow, Ind. T., arrived at the first dealer's establishment on March 4, 1907. Of this lot twenty-six were dead. On March 5 a Californian quail came from Elizabeth, Pa. On the 17th of the same month the second Washington dealer received from Wichita, Kans., a lot of forty-eight bobwhites. On arrival six were dead, and two were received later for examination. On the 21st a sharp-tailed grouse was received from the first-mentioned Washington dealer; the next day two more died. The disease also appeared at Yarmouth, Nova Scotia, the bobwhites coming from Alabama.

According to these facts, the disease exists, or has existed, in Alabama, Kansas; Indian Territory; Washington, D.C.; Mount Vernon, Va.; Elizabeth, Pa.; Boston and Worcester, Mass.; and Yarmouth, Nova Scotia.

It is uncertain if this disease exists in various species of quail imported from America and elsewhere and confined in aviaries in Great Britain. Numerous deaths, however, occur in an enzoötic form from time to time, and the clinical history of the birds during life is similar to that of the American outbreak.

Species Affected.—As far as is known, the disease is prevalent in the common bobwhite (*Colinus virginianus*), the Californian quail (*Lophortyx californicus vallicola*), the mountain quail (*Oreortyx pictus*), the scaled quail, called also " cotton-top " or blue quail (*Callipepla squamata*), and the sharp-tailed grouse (*Pediocœtes phasianellus campestris*).

Etiology.—This disease was at first thought to resemble the British grouse disease, and then the infectious enterohepatitis of turkeys, but no amœba could be found. All the diseased birds examined yielded a bacterium of the coli group. Inseminations from the blood, lungs, liver, and intestines gave a rather actively motile, rod-shaped organism, appearing often as ovoid cocco-bacilli, single or united rods with rounded ends, and even filaments. Polar staining was constant, none with Gram, and no spores were detected. Growth occurred without or with oxygen. Its growth on gelatine, the character of gas formation on glucose, lactose, and saccharose bouillons, and its milk coagulating properties, prove it to be a form of *Bacillus coli.*

It did not prove pathogenic to the fowl, pigeon, or rabbit. Mice and guinea-pigs succumbed with the characteristic lesions. Feeding experiments proved negative.

Symptoms.—The period of incubation seems to be about ten days. There is dulness and a tendency to squat in the corner of the cage with ruffled feathers. The appetite is diminished or *nil*, and in three days the bird is dead. When death is delayed and the disease runs a more chronic course, diarrhœa, although not well marked, may be present, and emaciation becomes profound.

Lesions.—The carcass is generally plump, occasionally emaciated.

The lungs are slightly congested ; the liver is also congested and shows a few small areas of superficial necrosis. The intestines are studded with minute ulcers. These are the characteristic lesions in the majority of cases.

Rarely the lungs show areas of consolidation, in the form of small, dark red spots. The liver is often free from necrotic areas, but merely congested ; sometimes a large portion of a lobe, in depth as well as in extent of surface, is destroyed. The spleen is always congested, sometimes enlarged, and at other times quite dark. The constant lesions are minute or pin-head-sized areas of necrosis, scattered throughout the length of the intestine ; these are seen through the wall of the intestine as small yellowish spots. Sometimes there is, however, patchy redness or minute erosions or necrotic foci, covered with a croupous exudate. The necrotic process may continue until a true ulcer forms, sometimes even penetrating the wall of the gut, and followed by the attendant small yellowish masses of necrotic material coating the intestines or the walls of the abdominal cavity.

Diagnosis.—This disease might be confounded with intestinal coccidiosis, but there are no coccidia in the lesions. It somewhat resembles bird fever. But diagnosis must be doubtful until the correct etiology has been definitely settled.

Treatment.—Prevention should be aimed at. Strict isolation, cleansing and disinfection of cages, water-troughs, food-trays, etc., should be carried out. Intestinal disinfection by the use of β-naphthol, salol, or cyllin, is worth a trial. The subcutaneous injection of atoxyl might also be attempted.

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CHOLERAIC GASTRO-ENTERITIS IN BIRDS

By H. GRAY, M.R.C.V.S.

Synonyms.—Spirillar, or vibrionic cholera of birds; Gastro-enteritis cholerica avicum.

French: Gastro-entérite cholérique des oiseaux.

Definition.—A contagious gastro-enteritis of poultry, characterised by a profuse liquid diarrhœa, and due to a bacterium (*Vibrio metchnikova*, comma bacillus, *Spirillum* or *Vibrio choleræ avicide*) closely resembling Koch's comma bacillus of Asiatic cholera (*Bacillus virgulus*), and secreting an analogous toxin.

History.—This disease was observed in Odessa in 1888, attacking fowls, and was studied by Gamaleia. Palmirsky and also Bruhl have made important researches into its nature. Its chief interest is due to the fact that it closely resembles Asiatic cholera of man.

Bacteriology.—The *Vibrio metchnikova* in the pigeon's blood varies in size and shape. It may be wide, short, and curved, and have rounded extremities; or it may sometimes be united in the form of spirals, having from four or five to ten spirals, resembling a piece of slack, unrolled or untwisted cord. It is very motile, and possesses a single vibratile flagellum. It is Gram-negative, and stains with the ordinary aniline dyes. In *veal broth*, peptonised or not, it renders the medium turbid in six to seven hours, with silky undulations; the following day the surface is covered with a white mist. On *gelatine* a stab puncture shows after two or three days gas bubbles, which gradually increase, and in the lower part the medium is liquefied in the form of a cylinder, showing in its centre a white line twisted in a spiral manner. Later on the gas bubbles burst through the surface, and the whole mass becomes liquefied, and then white granular particles are deposited on the bottom. Plate cultures show visible colonies on the third day, which form round liquid patches with a white central spot. On *agar* a white layer with a thick centre, which has a yellowish, shining appearance, is formed. On *potato* a brown layer with a darker centre than the circumference appears; it is more profuse than with Koch's organism, which gives yellowish-brown striæ. *Milk* at 35° C. is coagulated after a week; it becomes intensely acid, and the vibrios are destroyed. A typical culture is formed in eggs. After ten days the dissolved white is transformed into a yellowish, turbid liquid, whilst the yolk is distinctly black. In peptone solution nitrites and indol are formed.

Under certain conditions spores are formed. When the cultures are injected into the fowl, pigeon, and guinea-pig, they cause death. Absorption does not produce any effect in man. Ingestion kills young fowls. Adult fowls are less receptive, and they always resist by ingestion. Pigeons are very susceptible to subcutaneous and intramuscular inoculation, but they also resist by ingestion. The guinea-pig is more susceptible to the fowl vibrion than to Koch's Asiatic cholera organism; it succumbs to intraperitoneal and subcutaneous inoculations, and even by ingestion without previously rendering the stomach alkaline. The rabbit and the spermophile are only killed by large doses. Young suckling rabbits and the spermophile are very susceptible to Koch's bacillus of Asiatic cholera.

Gamaleia believes that natural infection takes place by the air-passages, and from thence gains the intestine, and afterwards reaches the capillaries and enter their walls. Death is due to absorption of the toxin secreted in the intestinal wall. Intra-intestinal culture does not give any sign of intoxication. The vibrion exposed to a temperature of 50° C. is destroyed in five minutes; it resists this temperature for one or two minutes, and at 45° C. for ten minutes. Its virulence is increased by successive passages through the pigeon. It is the same with the rabbit if inoculations are made directly into the lung. By these means a virus is obtained that will kill the fowl, lamb, dog, etc. On the other hand, its virulence is decreased if the microbes are injected under the skin or into the vein.

The disease is said to be *not contagious*. It is supposed that the germs, of which several varieties or species have been described, exist in soil and water in certain localities. The affection clinically resembles to a certain extent some of the protozoal diseases in man and animals, and occurs in the summer months. Pettenkofer and Emmerich, and Metchnikoff and his pupils have ingested pure cultures of Koch's bacillus without experiencing any ill-effect by so doing. On the other hand, when the stomach was previously rendered alkaline with sodium bicarbonate, only a choleriform diarrhœa without any serious general symptoms was obtained. In one case, however, Metchnikoff observed all the symptoms of cholera after a man had swallowed a gramme of bicarbonate of soda, and then a variable quantity of culture on agar emulsified in a little sterile bouillon.

Symptoms.—As with a good many avian diseases, the affected birds do not move, appear drowsy or sleepy, have their feathers open, the wings drooping, or they form themselves into a ball. There is a profuse watery diarrhœa. Adult birds may last in a state of sleepiness for forty-eight hours or more. The temperature remains about normal. It resembles in many of its clinical features fowl cholera.

Lesions.—The crop is filled with a thin liquid material. The small intestine contains a great quantity of yellowish-grey fluid, sometimes mixed with blood. The intestine is intensely hyperæmic or congested. The other organs are usually normal in appearance. The spleen is not enlarged, but always remains small and pale. The vibrios are copiously present in the intestinal fluid and blood in young poultry, but not always in the blood of adults.

Immunisation.—Cultures in calves' feet broth, after sterilisation by heat at 120° C. for one hour, give a toxin which kills the guinea-pig. A dose of 4 c.c. leads to death within twelve to twenty hours, after a subnormal temperature. Smaller doses produce a temporary disturbance; on recovery, they become immune against the vibrion. Immunised animals give a serum that is endowed with preventive and curative properties.

Pfeiffer has shown that, while the *Vibrio Metchnikova* is virulent for the pigeon, the vibrio of Koch is not; and that a pigeon that has received a large dose of Asiatic cholera bacillus is afterwards killed by an injection of the avian vibrio, just like one not previously treated by the former microbe.

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PIROPLASMOSIS

INTRODUCTION.

The mass of our knowledge with regard to the cause and nature of this important group of diseases has been gathered within recent years. The stimulus to investigation of the piroplasmoses was first given in 1893 by the publication of the classical experiments made by Smith and Kilborne in connection with Texas fever in cattle. They showed that this disease was caused by a small parasite living and propagating in the red blood-corpuscles of affected cattle, and that it was transmitted by the agency of a tick.

The name "pyrosoma" was given to the genus to which these organisms belonged, because they were commonly pear-shaped; and because the organisms were frequently found in pairs attached by their narrow ends, they called the parasite the *Pyrosoma bigeminum*.

The name "pyrosoma" had, however, already been given to a form of Tunicate, and so in 1895 Paton termed the genus "piroplasma." Starcovici had already called the organism "babesia," and it is probable that by this term it will afterwards be commonly known.

Since Smith and Kilborne's discoveries, piroplasmata have been found in various species of mammals, causing serious disease, and different varieties of piroplasmata have been discovered in the same species. Each variety of piroplasmata appears to be able to live and propagate only in one species of mammal. Hence it is necessary for the experimental transmission of the disease to use as the experimental animal an individual of the same species as that in which the piroplasmosis has been discovered.

All the piroplasmata are essentially intracorpuscular animal parasites. Only occasionally are they found free in the blood-plasma of their ultimate host, the mammal. They belong to the phylum Protozoa, subphylum Sporozoa, class Telosporidia, order Hemosporidia, suborder Acytosporea, genus Babesia or Piroplasma. They do not carry pigment. Their general form is spherical or pear-like, varying from 0·5 to 5 μ in size.

They may be single, paired, or multiple in the blood-corpuscles. They are very active, throwing out pseudopodia in their movements, and forming crescents, ringforms, triangles, rods, and presenting at times long filaments. The parasite consists of cytoplasm and a nucleus. The nucleus of the cells is of indefinite shape, consisting of chromatin granules arranged in rod, ring, or semicircular form, excentric in position, and frequently flattened against the periphery of the cell.

The attempts to cultivate these parasites in artificial media have not so far been successful. For the identification of the parasite the blood of animals showing symptoms of piroplasmosis is examined. Blood films are prepared, fixed, and stained with any one of the various modifications of Romanowsky stain, Giemsa, Jenner, or McConkie.

The exact life-history of the piroplasmata has not yet been fully worked out. Dr. Gonder has recently investigated East Coast fever in cattle, and discovered the process of development of the parasite causing that disease. Because of certain peculiarities of that disease, the inability to transmit it by inoculation of the blood of affected animals, by the complete disappearance of the parasite from its host after recovery from the disease, and by the peculiarities in the morphology and development of the parasite, it has been regarded as a variety of a genus distinct from the piroplasma called " Theileria."

The organism is named the *Theileria parva.* The disease caused by the Theileria is, however, for the sake of convenience, described under the heading of Piroplasmosis. Each piroplasmosis is transmitted under natural circumstances by inoculation by means of a tick. The variety of tick transmitting varies with the disease, and the exact stage at which the tick conveys the disease varies. In Texas fever in cattle the adult female tick sucks the blood of an infected animal, afterwards falls off its host, and on the ground lays eggs, from which the larvæ develop and inoculate the disease into any susceptible animal which they may have an opportunity of attacking. In malignant jaundice the disease is transferred only by the adult tick, the piroplasmata being carried from the adult female tick which has sucked the blood of an infected dog, through the egg to the larval, nymph, and adult stages of the progeny. In East Coast fever the adult tick does not appear to infect its young. The disease is transferred by the tick between its larval and nymphal, or nymphal and adult stages. In other forms of piroplasmosis the disease may be transmitted as in East Coast fever, but also from the adult tick through the egg to the larva of the tick.

When an animal is showing symptoms resulting from the presence of piroplasmata in the blood, it is easy to demonstrate the parasite in blood-films. After recovery, the parasites decrease rapidly in number, and may not be found in examination of blood-films, but they remain in small numbers in some portion of the blood-stream for a long time after the attack, perhaps for life. After recovery from an attack, the acute symptoms may reappear after weeks, months, or years, because of some circumstances acting adversely on the defences of the body. In all cases a marked and lasting immunity usually results from an attack of piroplasmosis. In East Coast fever the organisms disappear entirely from the body of the host after an attack, so that, unlike the piroplasmosis, the animal no longer remains "the reservoir of the virus."

Artificially the disease may be transferred to the susceptible animal by the inoculation (subcutaneous or intravenous) of a few cubic centimetres of blood from an infected sick animal. The chief exception to this rule is East Coast fever.

In the blood-stream the piroplasmata cause the destruction of the red blood-corpuscles they invade, and a consequent great reduction in their number (oligocythæmia). The hæmoglobin from the broken-down corpuscles is dissolved in the blood-plasma, and discharged in the urine by the kidneys. Destructions of the red blood-corpuscles and oligocythæmia are not features of East Coast fever.

In the treatment of these diseases typan blue has been used with success in several varieties of piroplasmosis. It is of no service in East Coast fever.

In regard to prophylaxis, attempts have been successfully made to eradicate the disease by exterminating the tick or purifying the tick by changing the species of mammal on the pasture. In some forms control of the disease only can be hoped for, and this has been done by breeding from immunised animals, immunising by inoculation of infected blood, or rearing young animals on infected land.

I. BOVINE PIROPLASMOSIS.

By S. B. WOOLLATT, F.R.C.V.S.,
Mooi River, Natal.

Piroplasmata are protozoal organisms which invade the red cells of the blood, and are, under natural conditions, transmitted by ecto-parasites. They may be classed under the Hæmosporidia, and thus be considered relations of the malarial parasites of man. Their definite host is the tick, and their intermediate hosts are cattle and other vertebrates.

The diseases caused by piroplasms in cattle are—

A. Texas Fever (see p. 983).
B. East Coast Fever (see p. 994).
C. Ailments caused by the Piroplasma mutans (see p. 1005).
D. British Redwater (see p. 1005).

East Coast fever, however, in view of recent investigations by Theiler and others, should not strictly be included under piroplasms, as the causal agent is not really a piroplasma, and for this reason Bettencourt has created a new genus for it, which he has named "Theileria."

As it is so closely allied to piroplasmosis, and has in the past been considered under this head, for convenience it is so considered here.

Further, in South Africa the disease East Coast fever is to a great extent allied to true piroplasmosis, as regards its symptoms, method of dissemination, and treatment.

The first to observe the presence of endo-globular parasites was Babes, who recorded them, in 1888, as appearing in the blood of cattle suffering from hæmoglobinuria in Europe. He did not at this time recognise the true nature of these bodies, and it was not until Smith and Kilborne's researches into Texas fever in 1890 that their real nature and their method of transmission was shown. These latter workers were also the first to demonstrate the part played by ecto-parasites in the transmission of diseases.

Diseases produced by true piroplasms are communicable by direct blood inoculation from an animal suffering from the disease, or from one which is immune or indigenous to infected pastures, into a susceptible animal.

Formerly Texas fever (*P. bigeminum*) was described under the name of "bovine piroplasmosis," and East Coast fever was also termed by some "tropical piroplasmosis." If confusion is to be avoided now that three distinct bovine maladies are known to be caused by piroplasms, we must in future apply the term "piroplasmosis" in a general sense.

The parasites responsible for all forms of piroplasmosis are named piroplasms. That causing Texas fever, which was the first to be discovered was originally named *Pirosoma bigeminum* by its discoverers Smith and Kilborne, on account of its pear-shaped form, and the fact that two parasites usually hang together in one blood-corpuscle (twins).

The parasite in Texas fever is the prototype of the genus Piroplasma, and all forms of piroplasms found in the blood of bovines were, until the researches of Theiler and Koch into East Coast fever, and of Theiler into *P. mutans* and *Anaplasma marginale*, considered to be different forms of the one parent parasite (*Piroplasma bigeminum*).

All forms of bovine piroplasmosis are communicated by ticks.

Piroplasms having a different refractile index to the blood-cells may be seen as colourless bodies within the corpuscles in unstained blood. With a watery solution of any of the basic aniline dyes the parasites may be seen, owing to their taking the stain more deeply than the corpuscles. These are not, however, satisfactory stains to use, as they do not show up the structure of the parasite. Generally, in staining piroplasms, the Romanowsky or its more recent modifications, Giemsa or McConkie's, are by far the best, as with these stains the structure of the parasites is clearly shown.

With these differential stains the karysome or nucleus is usually seen as a deep, violet-stained chromatic body, in some cases appearing as a diffused body staining a faint red. This nucleus is most frequently on the margin of the parasite, in some cases appearing to be outside the cytoplasm. It is always a distinct body, and is positive evidence of piroplasmata. In the smaller forms of the parasite (bacillary forms) its presence enables them to be distinguished from other bacillary bodies.

In some parasites two nuclei are seen representing the condition of the parasite preceding division. Frequently, in the larger parasites, other chromatic particles exist distinct from the actual nucleus, but taking on a similar stain. The cytoplasm of the parasite stains a bluish tinge.

Piroplasms multiply in the blood by direct fission, the first evidence being division of the karysomes. Some observers, however, claim to have seen flagellated forms (microgametes). This process of multiplication and the invasion of fresh blood-cells is an auto-infection, and corresponds to the schizogony or monogony phase of the life-cycle of the malarial parasite. Ross showed that the sexual cycle (sporogeny or amphyony) of the malarial parasite takes place in the body of the mosquito, and from analogy we may suppose that sporogeny in the case of the piroplasms takes place in the body of the tick, their definite host. With piroplasms the deposition of pigment in the tissues, which is so characteristic of malaria, is absent, and the morbid changes which take place in the blood vary with the specific piroplasma concerned, and are described separately later.

A. TEXAS FEVER.

Definition.—Texas fever is a specific fever affecting bovines, caused by the presence in the blood of the *P. bigeminum.*

Nomenclature.—It has been described under several names. In North America it is known as Texas fever, tick fever, and has been described by Law as Portuguese cattle fever. In the Argentine Republic it is known as "la tristeza"; in South Africa as Bush-sickness and gall-sickness; and it is described in many other parts of the world as bovine malaria and bovine piroplasmosis.

Geographical Distribution.—It occurs in East, West, and South Africa; in North Africa (Tunis, Algeria, and Egypt), in North America (the Southern States), in Queensland, the Argentine Republic, Uruguay, Costa Rica, Venezuela, and at one time it devastated the cattle of Jamaica. In India it was first discovered by Raymond and Stockman, and it also occurs in Java. According to Laveran, it was first observed in Europe on the Danube, and has since been met with in many parts of the Continent.

General Remarks.—This disease is the most common malady affecting bovines in tropical and semi-tropical countries. The knowledge of the geographical distribution of Texas fever is of value in guarding against its importation into clean countries, and for obtaining cattle capable, to some degree, of resisting the infection from infected countries when required for restocking purposes.

When considering the importation of cattle for restocking, however, it should be recognised that there is no true immunity against Texas fever, and that the degree of tolerance possessed by animals from different countries, and even from different localities in the same country, may vary to such an extent that animals from one locality or country fail to withstand the infection in another. That this is so has been proved by animals imported into South Africa from infected areas of Queensland dying to the extent of 80 to 90 per cent., and in other cases, where exposed to less grossly infected veldt, the importation has been attended with successful results.

Of all the cattle imported into South Africa for restocking purposes, those from Texas have proved to possess the highest degree of tolerance, as they have withstood successfully the veldt infection in some of the most grossly infected areas of South Africa. Cattle from the infected districts of the Argentine have also been. successful in South Africa. Although a country or locality is known to be infected with Texas fever, it does not necessarily follow that cattle from such areas can with impunity be imported into other infected countries. It is known, of course, that any conditions that lower the vitality of animals in whose blood a living organism exists, as is the case with immune Texas fever animals, may render such organisms active.

Etiology.—Smith and Kilborne in their researches proved conclusively that the common blue tick, known in America as the *Rhipicephalus annulatus* or *Boophilus bovis*, is the definite host of this parasite, and communicates the disease to susceptible bovines. In Africa it is the *R. decoloratus*, the *R. appendiculatus*, and the *R. evertsi*; in Australia the *R. Australis*. In Europe it is thought that the common tick *Ixodes ricinus*, or *I. Reduvius*, is the responsible agent.

There is no doubt that ticks are the agents responsible for the dissemination of Texas fever, but it is thought that blood-sucking flies may act as disseminators. Lignières cites a case, and the writer has seen an outbreak of acute Texas fever amongst highly susceptible animals, where all the evidence pointed to flies acting as the transmitters. The animals were cows imported from clean areas of the Argentine into South Africa, and taken direct from the ship by rail into stables and fed on imported fodder.

For immunising purposes 5 c.c. of blood from a healthy South African bred calf were injected into four animals, and induced a chronic form of the disease in the animals inoculated. Twenty days after the appearance of piroplasms in the blood of the inoculated animals cases of acute redwater appeared in the uninoculated ones in the same stable. Of twenty-six animals uninoculated, twenty died from acute redwater, and the remaining six remained healthy until exposed to veldt infection. No ticks could be discovered on those which succumbed, although an exhaustive search was made for them.

This in itself, however, must not be accepted as positive evidence of non-infection from ticks, but the conditions under which the cattle were kept precluded the probability of this taking place.

The common blue tick, *R. decoloratus*, passes the whole of its life upon its host—that is, it does not become detached to moult, but remains attached to its host during the periods of moulting. It is the larvæ of this tick, hatched from eggs laid by females which have matured on sick or immune animals, that communicate the infection to other susceptible cattle on becoming attached. From this it will be clear that the infection in the case of Texas fever passes through the egg stage of the tick responsible for its dissemination. The brown tick (*R. appendiculatus*) and the red tick (*R. evertsi*) may rarely transmit redwater, and they have a definite life-history, described later.

The disease can be produced either experimentally by the aid of ticks, or by direct inoculation with the blood of sick or immune animals; and because actual piroplasms cannot be demonstrated in the blood of immune animals, it does not follow that such blood will fail to infect; in fact, in practice, if animals are known to be immune—that is, to be capable of withstanding infection when exposed to infected pastures—it may safely be assumed that their blood is infective.

The explanation of this is that the piroplasms may be so few as to render it impracticable to demonstrate them by examining several fields of a blood-film.

Smith and Kilborne obtained ticks from infected cattle, and distributed them on to clean pastures. Cattle from clean districts were subsequently put on to these pastures, and contracted the disease after an interval sufficient to allow for the development of a fresh generation from these infected matured ticks, which interval would occupy from five to six weeks or longer, depending on the temperature—*i.e.*, whether summer or winter.

Similar results occurred when tick-infected cattle from disease areas were taken on to clean veldt, the local healthy cattle becoming infected after certain intervals which depended on a new generation developing from the matured infected ticks dropping off the cattle brought from infected areas.

Young ticks hatched from mature females obtained from cattle from infected areas produce the disease when placed on susceptible cattle.

The disease cannot be produced by contact of susceptible healthy animals with those immune or suffering from the disease. Ordinarily, the disease is carried from one centre to another by cattle carrying and dropping infected ticks. Ticks off their natural host do not travel any appreciable distances under natural conditions.

The causal agent, *P. bigeminum*, as previously described, is a pear-shaped body, usually found in pairs within the body of the corpuscle. It is rare that they are seen free outside the corpuscles.

Formerly this parasite has been described as occurring in many forms, such as the typical form, which is a pear-shaped body; also as a willow-leaf-shaped body, a round or bacillary form, and as a ring. We must now, however, dissociate these various forms from the *P. bigeminum*, and look on them more as separate piroplasms. As the parasites occur in pairs, they occupy the greater portion of a red corpuscle. They have their body with their long axes parallel or in a straight line. It is not usual to find more than two parasites together, but as many as four have been observed. The largest pear-shaped bodies are 4 μ long and ½ μ wide at their widest end. It is only usual to find a small percentage of the blood-cells—from 2 to 3 per cent.—which contain the parasites in peripheral blood. They are more numerous in the blood of internal organs, and first appear in the bone-marrow.

In the examination of cattle in South Africa one will always encounter other forms of piroplasms, as *P. mutans* is as generally distributed as *P. bigeminum*, and with the present rate of extension of East Coast fever infection (*P. parvum*), this disease will soon be more or less generally distributed also.

In view of recent discoveries, we now know that the chronic form, particularly in inoculated imported animals, is in reality due to gall-sickness (*Anaplasma marginale*) or to *P. mutans* infection.

Both these latter diseases are inoculable, and the parasites are present in the blood of recovered animals. What happens, therefore, is that in using blood from an indigenous animal to immunise an imported bovine the blood frequently contains the three parasites—viz., those of redwater, gall-sickness, and *P mutans*.

The redwater, with the short incubation period, reveals itself first; then *P. mutans*, with an incubation period of twenty-five to forty-five days; and, finally, gall-sickness, with an incubation period of from sixty to eighty days.

Theiler has shown this to be the case, and has worked out the separate inoculation periods by using pure infections only of these three separate parasites.

Lignières has studied the behaviour of piroplasma in defibrinated blood kept at blood heat, and he claims to have observed the formation of spores, which pass from the corpuscle into the serum, and invade fresh blood-cells, forming again into the pyriform body.

MICROSCOPIC APPEARANCE OF THE BLOOD.—The disease is essentially an anæmia, and in acute cases the rapid destruction of blood-cells is very considerable. The number of red cells may be reduced in a few days from 5,000,000 or 6,000,000 to 1,000,000 or 2,000,000 per cubic millimetre. In the most acute cases the blood may show little change as regards the variation in the size of the corpuscles, but in cases of a semi-acute nature the variation in size of these cells is most marked. The variation in the size of the blood-cells may be to the extent of many of them being not more than half the size of the largest cells.

Another constant change which takes place in attacks of any duration is the so-called "poikilocytosis." This is the term employed to denote marked abnormalities in the shape of the red cells. Many may be pear-shaped or sausage-shaped, with crenated edges.

Another change seen in severe cases is the appearance of nucleated red cells, which may be so numerous as to be found in almost every field. These nucleated blood-cells are really young cells thrown into the blood-stream before maturity, and may be found in all cases of excessive anæmia.

The nucleus of a red cell, or normoblast, is spherical, and usually stains intensely and uniformly, and can be easily distinguished from the nucleus of any white corpuscle. It occupies the greater portion of the corpuscular body. With the Romanowsky stain the blood may show the normal eosin tint, but it more frequently takes on a kind of mauve tint.

Another condition seen is that in which many red cells, normal in form and size, stain an abnormal tint with Romanowsky, and instead of the normal eosin tint, they show a mauve tint.

A most characteristic condition found, particularly in cases of some duration, is the so-called "punctate basophilia." A red cell showing this condition is considerably increased in size, and, when stained with Romanowsky, shows a number of blue-stained, very minute particles in its substance, the rest of the cell taking on the ordinary eosin tint. These blue-stained particles appear to be due to degeneration of the substance of the corpuscle. This condition is of particular value, owing to its appearance usually taking place in the later stages of an attack, when difficulty is frequently experienced in detecting the causal parasite.

Period of Incubation.—If by this we understand the period which elapses between actual infection taking place and the onset of the symptoms—it is by direct inoculation from five to six days—the parasites appear to multiply in the blood at once. If by the period of incubation we understand the time which may elapse before cattle give evidence of the disease after exposure to infected veldt, it will depend, first, upon the intensity of the tick infection; and secondly, to some extent, on the time of the year (ticks being more virulent in summer and in autumn), and may be put down at from ten to twenty days. When the disease has been introduced on to a clean farm by infected ticks—that is, a farm free from ticks—the period of incubation is usually about six weeks, which corresponds with the time required for the eggs laid by the infected ticks to hatch out. In summer the hatching takes place after an average time of thirty-six days, but would be longer in winter.

Morbid Anatomy.—The lesions vary, depending upon the attack being of an acute or chronic nature, and in the most acute cases, where the lungs are affected, there is a large collection of froth around the nostrils, which may be tinged with blood similar to that seen in cases of anthrax, horse sickness, or East Coast fever.

On opening the carcass, the subcutaneous veins are found to be full of blood of a pale colour, which usually coagulates fairly well, the blood-serum remaining red from the hæmoglobin set free from the corpuscles. Yellow pigmentation of the subcutaneous tissues and serous membranes is frequently present, as in the case of East Coast fever. Œdema of the lungs, as above stated, may be present, involving the greater portion of both lungs, accompanied by a pleuritic effusion, which is of a pale yellow colour and free from flocculi. The pericardial sac may contain an excess of fluid, and hæmorrhagic spots are frequently present on the epicardium and endocardium—in fact, these hæmorrhagic spots and blotches may be distributed generally over the serous membranes, and the fat around the base of the heart is usually infiltrated with a gelatinous exudate.

The Spleen.—This is invariably enlarged, and often mistaken for an anthrax spleen. It is usually three or four times its normal size, and its pulp is of the consistency of black-currant jam, but not so diffluent as in the case of anthrax.

The Liver is enlarged and congested, and the gall-bladder usually contains a quantity of dark inspissated bile, depending upon the duration of the attack.

The Kidneys.—The fat around these organs is usually infiltrated with a gelatinous exudate, and the kidneys themselves congested and enlarged.

The Bladder.—This usually contains a quantity of dark-coloured urine, which may be black in some instances.

The Abomasum invariably shows gastritis. The mucous membrane is swollen and claret-coloured, and the gastric lesions have frequently been mistaken for those of rinderpest; but it is more of the nature of an ecchymosis than of a diffuse inflammation.

The Intestines frequently show acute enteritis, particularly the cæcum and large intestines. The lesions, however, are also more of an ecchymosis than of a true inflammation.

The Muscles.—If the destruction of the blood-cells has been excessive, the muscular tissue is markedly pale and flabby, particularly the heart muscle.

Chronic Form.—The carcass is emaciated, but otherwise no visible outward signs of disease are observed, and frequently on post-mortem, besides the watery condition of the blood, and perhaps a slight swelling of the spleen, no lesions are found to exist.

Usually, however, the muscular tissue is pale and flabby, and hæmorrhagic spots are scattered more or less generally throughout the serous membranes. The *spleen* is enlarged, the *liver* yellow and friable, and the *gall-bladder* contains a thick yellow or green bile. which is often stringy, due to the presence of a quantity of mucus.

The *kidneys* are pale in colour, and may be embedded in an œdematous tissue.

Gastro-enteritis is usually absent, but the abomasum may show hæmorrhagic spots, the latter being also present in the intestines, particularly in the cæcum. The *bladder* is usually free from lesions, and the urine is normal.

Symptoms.—All observers agree that an acute and chronic form of the disease should be recognised, and in my experience we should also recognise a benign form, or perhaps, more correctly, a recrudescence where Texas fever is endemic.

Benign Form.—This form occurs in animals possessing a varying degree of immunity or tolerance, being endemic to infected veldt. This immunity can be broken down to a great extent by any conditions which tend to lower the vitality of an animal, such as overwork, cold, other diseases, etc., and animals capable of withstanding the infection on one pasture or locality may suffer from this form of the disease, or even a more acute form, if subjected to veldt with a higher degree of infection.

In these benign cases the symptoms shown are slight, and are frequently overlooked. Hæmoglobinuria is rare, the only symptom being an elevation of temperature lasting four or five days, running from the eyes, a dribbling of saliva, slight staggering gait, and a tendency to constipation. These attacks most commonly end in recovery. There are also the symptoms of gall-sickness and fever produced by *P. mutans*.

When accompanying a disease such as rinderpest and East Coast fever, animals usually show hæmoglobinuria, etc. With East Coast fever it is always fatal, and with rinderpest recovery is rare.

Acute uncomplicated redwater is frequently seen in indigenous animals.

Acute Form.—The symptoms are a high fever, the temperature ranging from 106° to 107° F.; staggering gait, the hind-quarters being particularly affected; a discharge from the eyes; a dribbling of saliva; constipation in the early stages, frequently followed by diarrhœa and dysentery; also hæmoglobinuria, which may commence early or be delayed until the fifth or sixth day of the disease. With this condition the urine varies in colour from a light claret to black, and is always albuminous, whether discoloured or otherwise.

The mucous membranes show evidence of a great reduction of the blood-cells, and may be icteric. When the lungs are affected, which is common in the most acute cases, the symptoms are those arising from pulmonary œdema and pleuritic effusion, with all the accompanying symptoms of distressed respiration, etc.

The heart-beats are increased in number and in force; the animal frequently becomes comatose, or may show evidences of delirium. These acute cases usually end in death in from two to eight days after the onset of the symptoms. In some cases the chronic supervenes on the acute form.

Chronic Form.—In imported susceptible cattle this form more usually results from the effects of artificial inoculation than from natural infection. The chronic form from natural infection among imported cattle is rare in countries where the degree of infection is at all high. In this chronic form the rise of temperature is intermittent, being high in the evening (105° to 106° F.) and low in the morning (102° to 103° F.). There are all the signs of a progressive anæmia, and the emaciation increases. The appearance of the mucous membranes may be icteric, and they usually show hæmorrhagic blotches. The appetite is capricious, but in some cases may continue, and rumination is maintained.

The hæmocytometer will show a reduction of the red blood-cells, which is not so great as in acute cases, and the production of new cells may almost keep pace with this reduction. Hæmoglobinuria is not a constant symptom, the broken-up blood-cells being carried off by the liver, but albumin is invariably present in the urine. The bowels may be normal or constipated, or a chronic diarrhœa may exist. In milch cows the supply of milk is greatly reduced or ceases, depending on the severity of the attack; but the animal invariably continues to lick itself, and shows other normal signs. If stabled, bedsores are common. Death may take place from exhaustion, or it may be sudden, due to heart failure. It is conceivable that death may also take place from asphyxia when the blood-cells fall below a certain number per cubic millimetre. Few animals will live with less than 2,000,000 per cubic millimetre.

Treatment.—In the case of animals indigenous to infected veldt, outbreaks of redwater are usually confined to individual cases. It does happen, however, under certain circumstances, that extensive outbreaks of the disease may occur among animals born on infected veldt. These outbreaks are accounted for by the fact that infection of a greater virulence has been introduced by susceptible animals dying, or that conditions prevail which, by lowering the vitality of the cattle, render them more liable to infection. In such cases it is always advisable to remove to fresh pastures, even if such removal is limited to the extent of taking them from one paddock to another. The mortality under such conditions invariably ceases, owing to their being removed from the source of infection ; but in order to guard against introducing the more virulent form to the new pasture, cattle should be periodically dipped, at an interval of not less than fifteen days, to prevent the re-establishment of the more acute form of infection.

Dipping, as a means of combating Texas fever, is practicable, owing to the fact that the tick responsible for its dissemination passes the whole of its life, from the larva to the mature adult, on its host, and this period of its existence occupies some twenty-one days. It therefore follows that by dipping at intervals of fifteen days we insure the destruction of this tick at some period of its existence, and thus prevent it maturing and propagating.

Although it has been found from experience that dipping will not in all cases destroy the more adult forms of tick, yet the eggs laid by such ticks, after being dipped, are not fertile. It is conceivable that with the most careful dipping all the ticks on an animal are not destroyed, and some may reach maturity and actually propagate, but they are reduced very considerably.

It has been shown in practice that with a policy of systematic dipping and fencing, to prevent the reintroduction of ticks by stray animals, the mortality from redwater is very considerably reduced, but with this reduction of the ticks the immunity or tolerance to the disease is also considerably reduced, and it therefore follows that, short of a general policy of dipping in a country where redwater is endemic, owners who prefer to pursue the system of tick extermination must also be prepared to retain their cattle on their own farms, unless they recognise that by removing them they incur a considerable risk of heavy mortality.

In cases of outbreaks where dipping is not practicable, it is still desirable to remove to fresh pastures, as the mortality is always checked thereby.

In practice, the dips which have been found to give the most beneficial results—and, in fact, the only ones of any practical use for the destruction of ticks—are those containing arsenic in solution. Cattle so dipped, particularly on the first few occasions, may show symptoms of arsenical poisoning at any period within eight days after being dipped, and these symptoms become particularly evident if the cattle are worked.

The question of dipping in the treatment of tick-borne diseases is more fully dealt with under East Coast Fever (see p. 1002).

For the treatment of individual animals many remedies have been tried, but there is no doubt the success which may be claimed for any particular remedy is far more dependent upon the natural resistance of an animal to the effects of the piroplasma than to any merits of the treatment. The success of trypan blue in the treatment of canine piroplasmosis warrants its use in bovine piroplasmosis.

In Cape Colony considerable success has been claimed for the use of an oleaginous purgative containing drachm doses of carbolic acid, but all kinds of remedies, from sheep-dip to paraffin, have been used with varying success.

Quinine, which has been put forward by many as a most valuable drug in the treatment of Texas fever, has not given any satisfactory results in the hands of the writer. It has been tried in large and small doses, given hypodermically or by the mouth, and in no case has any beneficial result been observed from its use.

In the treatment of valuable animals purgatives must be avoided, but laxatives, antiseptics, and stimulants, combined with good nursing, are the most rational lines to follow.

When the acute stages of the disease have passed off, blood tonics are indicated; but it must not be supposed, because an animal may recover from the acute attack, that he will not suddenly develop a second acute form of the disease, and die suddenly—in fact, this is probably the most disappointing feature of the treatment of redwater, which one is so often called upon to combat in the case of imported bulls introduced into countries where the disease is enzoötic. In cases where such animals have been exposed to veldt infection they should be at once stabled or removed from the possibility of reinfection.

Immunity.—Cattle which are indigenous to infected veldt are, under ordinary circumstances, immune to the infection to which they have been exposed. This immunity, which, perhaps, should be more correctly termed a tolerance, appears to exist in proportion to the degree of infection to which they have been exposed. That this is so is shown by the fact that animals capable of withstanding the infection in one locality may succumb to an attack of Texas fever if removed to a farm or locality where the infection is greater, although such removal may only be a matter of a mile or so.

Again, animals may be capable of withstanding the infection on a farm during winter, when ticks are less active, but succumb to the disease on the same farm in summer.

The blood-serum of an immune animal is not protective, however severe the attack may have been from which it suffered, neither can the blood-serum be made protective by hyperimmunisation. The immunity is not an active one, as may be proved by removing animals from infected veldt for a number of years, and then again exposing them. It, however, lasts practically the life of an ox.

Animals born on infected veldt acquire immunity early in life, young calves contracting a mild form of the disease, which passes off unobserved. The earlier the calves are exposed to infection, the less they suffer from the effects of an attack. In the summer months, however, when tick infection is most intense, considerable mortality may be experienced among even newly-born calves, and if they be housed during the first few months of their lives, and exposed during the autumn, when the ticks are most active, mortality may be excessive. Young animals are, however, far less susceptible to the disease than adults, and are, in consequence, more suitable for inoculation purposes.

INOCULATION TO CONFER IMMUNITY.—This has been largely carried out in some countries. Its efficacy does not depend upon any protection in the serum, but is due to the blood used for inoculation containing the piroplasmata of diminished virulence, which produces a mild, non-fatal attack. This system of inoculation has only been successfully carried out to any extent in Australia. The method consists of subcutaneous injections of from 5 to 10 c.c. of defibrinated blood taken from an animal recovered from Texas fever into the animal to be immunised.

We have no means of determining by microscopical examination whether the blood of any particular animal is suitable for inoculation, and the effects which may result from the use of the blood of any animal can only be determined by the results upon the animal inoculated.

With our present knowledge, the mortality attending inoculation of susceptible animals is far too high to justify its adoption. Lignières claims, however, to be able to successfully vaccinate susceptible animals, and, presuming he is able to do so, we should still in practice have to demonstrate whether his method would establish an immunity of sufficient strength to withstand the varying degrees of infection animals may have to be subjected to.

It has been shown in practice that this system of inoculation can be usefully employed in increasing the degree of immunity of an animal already possessing some resistance.

Attempts to immunise by controlling the tick infection have not been successful. One may obtain with equal certainty either fatal or negative results.

Experience in Africa has shown that in attempting to immunise susceptible animals with the blood of those indigenous to the country the mortality has chiefly been experienced from what has been termed a second reaction—that is, the inoculated animal has appeared to have recovered from the first infection, which commences from the fifth or sixth day after inoculation, but succumbs to the second reaction, which commences from the thirtieth to the fortieth day. With the appearance of this second reaction, ring forms of the piroplasms appear.

Such cases occur where the blood used for inoculation contains both the *P. bigeminum* and the *P. mutans*, the primary reaction being due to the former, and the second reaction to the latter.

Theiler has shown that these piroplasms are distinct, and, in practice, he thinks that if animals are immunised separately against each of these parasites, an immunity may be established enabling animals to withstand veldt infection in South Africa.

To this must now be added another tick-borne disease indigenous to South Africa—viz., gall-sickness, due to the *Anaplasma marginale.*

B. EAST COAST FEVER.

Definition.—A specific disease affecting bovines, caused by the *Piroplasma parvum*, or, as more properly termed, the *Theileria parva.*

General Remarks.—It exists in German East Africa, where the cattle appear to possess an immunity, probably inherited through generations; also in Portuguese East Africa, Rhodesia, the Transvaal, Cape Colony, and Natal; and it has been observed by Dschunkowski and Luchs in the Transcaucasias, and termed by them "tropical piroplasmosis."

The disease has been described under several names, owing principally to its being confused in the early days of its investigation with Texas fever or ordinary redwater. It has been termed "Rhodesian redwater" and "Rhodesian tick fever," which names were inadequate and misleading. Subsequently it was termed "East Coast fever," which name is also misleading, as the disease is not confined to Eastern Africa, but has been observed, as mentioned above, in Asia Minor, and termed "tropical piroplasmosis."

The writer thinks that, although tropical piroplasmosis might scientifically be the most suitable name, yet, as other piroplasmoses exist in tropical countries, distinct from this disease, it would lead to confusion, and the name "East African Coast fever" is probably the most applicable, as it is chiefly prevalent on the East Coast of Africa.

Recent discoveries have also shown that the disease is not a true piroplasmosis.

This disease first came into prominence in Rhodesia, where it appeared in a cargo of cattle from New South Wales introduced for restocking purposes in 1901. This troop of cattle undoubtedly became infected with Texas fever at Beira, where they were retained on account of railway difficulties, that disease appearing amongst them while remaining at Beira. The cattle were removed to Umtali as soon as possible, to lessen the mortality which was taking place in the low country at Beira, it being thought that their removal to the higher latitudes would prevent further loss.

East Coast fever was introduced into Umtali by cattle intended for slaughter that were imported from German East Africa prior to the arrival of the Australian cattle.

After arrival at Umtali, the Australian cattle eventually all died from a mixed infection of Texas fever and East Coast fever, and later the local cattle there contracted East Coast fever and died. At this time it was thought that the disease was a virulent type of redwater, and in consequence no severe restrictions were placed on the movement of cattle in Rhodesia, with the result that it spread considerably. The Transvaal was invaded in 1902, and the disease obtained a good footing in that colony before its nature was recognised, it being confused with rinderpest, and the unsettled condition of the country, as a result of the war, precluding any proper supervision or investigation.

The disease was also introduced into other centres in the Transvaal by cattle from the East Coast through Delagoa Bay, and there is no doubt that for years past it has been indigenous to the East Coast of Africa north of Beira, but has only made itself evident of recent years, since cattle from these North-Eastern Coast districts have been imported to the more southern States.

Etiology.—From the very beginning it was observed that the disease was not contagious, and that healthy cattle could mingle with diseased cattle without danger for the time being.

Koch investigated this disease at Dar-es-Salaam in 1897, when he thought it to be a modified form of Texas fever, which opinion was then shared by Laveran, both considering that the small piroplasms found were young forms of the bigeminum. Gray and Robertson, who first investigated East Coast fever when it appeared in Rhodesia, also at the outset thought it to be a form of Texas fever, assuming a more virulent type, and they were no doubt influenced in forming this opinion by Koch's statements concerning the disease at Dar-es-Salaam in 1897.

It was known that Texas fever existed generally throughout Rhodesia, and it was thought that with the introduction of susceptible cattle (the Australian cattle), the disease assumed a more virulent form.

It has subsequently been shown that the *P. parvum* is distinct from the piroplasma of Texas fever, but its confusion with that parasite can easily be understood when it is known that, so far, in South Africa all cattle which have died or suffered from the disease have also been infected with the *P. bigeminum.*

The presence of the ring form of the piroplasm, now described by Theiler as the *P. mutans*, in the blood of animals inoculated with blood taken from those recovered from an attack of East Coast fever was considered by Koch as evidence of immunity having been produced in the inoculated animal; but Theiler soon showed that this form of piroplasm had nothing to do with East Coast fever, and he originally considered that it was a form the bigeminum assumed in the blood of cattle immune to that disease (Texas fever).

To further show the confusion which existed regarding the several forms of piroplasms, Koch considered that East Coast fever existed on the coast of Natal and the Cape Colony because he found ring forms in the blood of cattle from those districts years before the disease appeared in Natal, and up to the time of writing it has not extended to the coast of Cape Colony.

There is no doubt but that the Australian cattle referred to at first died of Texas fever; they acquired Texas fever at Beira, and East Coast fever infection after arrival at Umtali. The former (Texas fever) has a shorter incubation period than the latter, and the cattle first succumbed to Texas fever, and, in consequence, the first diagnosis of redwater was correct, and masked the disease which followed immediately with it—namely, East Coast fever.

Theiler has done more than any other investigator to unravel bovine piroplasmosis, and it is he, and, later, Meyer, Gonder, and Lounsbury, who have been chiefly responsible for working out the life-history of the parasite outside its bovine host—namely, in the tick.

It was early shown that ticks were responsible for the dissemination of East African Coast fever, and Koch first considered that the common blue tick, *Rhipicephalus decoloratus,* was the intermediary bearer; but this has not been confirmed, and this tick is now considered to play no part.

It has been shown that the ticks responsible are the *R. appendiculatus,* commonly known as the brown tick, which exists generally throughout South Africa; the *R. simus*, commonly known as the black-pitted tick; and Theiler has recently shown that the nymphs of the *R. evertsi* may also act as the disseminators. *R. cepensis* and *R. niteus* may also be considered as hosts of *P. parvum*. The principal ticks, however, concerned are the two first named, and the *R. appendiculatus* in particular. These two species leave their host at each moulting period—namely, as larvæ when changing to the nymph stage, and as nymphæ when changing to the imago or adult stage. As either nymphæ or adults they are capable of living many months without a host, and it is this power to remain active for such periods without feeding that maintains infection in pastures for as long as twelve to fifteen months after the last death from the disease. The infection in the case of East African Coast fever is not transmitted through the egg stage, but is communicated by a nymph which in the larval form fed on an animal suffering from East Coast fever, or by an adult which as a nymph fed on a sick animal. The nymphæ and imagos, however, play the most important part in the dissemination of the disease.

Ticks in any stage which may feed on animals recovered from East Coast fever do not acquire infection, and infected nymphæ or adults, when once they have bitten, lose their infection unless it is reacquired. With this knowledge, all restrictions on animals other than bovines could with safety be rescinded, as being non-susceptible. Ticks attached to them would lose their infection, and be unable to reacquire it. They could be of danger, however, in carrying infected ticks which did not attach themselves, but this in practice could be neglected, as the object of all ticks is to bite.

It has been shown that this disease is communicated by certain species of ticks, and as ticks do not travel any appreciable distance off their host, it follows that the disease is chiefly carried by sick cattle dropping infected ticks. In South Africa ticks which act as intermediary bearers exist generally, and although in clean districts they are at present harmless in this respect, they supply the one feature necessary for its dissemination, and render dipping as a practical means of dealing with and guarding against East Coast fever of great value.

The *P. parvum* is the smallest of piroplasms, and was, as already stated, at one time considered to be an intermediate form in the life-cycle of the *P. bigeminum*. It is now known to be a distinct species, and closely resembles the *P. mutans* when the bacillary form of that parasite is chiefly present—in fact, there were cases where a positive diagnosis of East Coast fever was impossible, owing to these parasites so closely resembling each other. Now, however, with the knowledge that Koch's bodies are specific of East Coast fever, the disease can with certainty be diagnosed microscopically.

The development of *Theileria parva*, the causal parasite, has been very fully described by Dr. R. Gonder in his report to the Secretary for Agriculture of Pretoria.

The most important feature microscopically of East Coast fever is that form of the causal parasite known as Koch's bodies, Koch's granules, blue bodies, etc.

Koch was the first to discover these bodies in the organs of animals which had died of East Coast fever, and we now recognise them to be specific for this disease.

Theiler, Meyer, Gonder, and others, have since recognised their true nature, and worked out the rôle they play.

The knowledge we now possess concerning these bodies warrants our belief that the tropical piroplasmosis (Transcaucasian), described by Dschunskowsky and Luchs, produced by *P. annulatum*, and occurring in Russia, the Egyptian piroplasmosis, and perhaps the *P. bacilliforme* of Tunis, are all infections identical with East Coast fever.

Koch's granules led to the identification of a disease of bovines in Uganda known as "anakeli," but now known to be East Coast fever.

Koch's granules, unstained, are round or oval bodies containing a few or several granules in their plasma.

In preparations stained after Giemsa or Leishman, they are described by Meyer as light blue or dark violet discs, containing a variable number of round or irregular light reddish or more bluish-violet granules, which are pure chromatin.

As before shown, these bodies are of chief importance in the diagnosis of East Coast fever. They appear early in the disease in the lymphatic glands and in the spleen. For diagnostic purposes, any of the superficial glands may be punctured, and slides examined from them. The glands most convenient to obtain films from are the prescapular and the precrural—the latter in particular.

By this method Koch's granules have been found on the second day after a rise of temperature in the animal. These granules represent a stage in the development of the *Theileria parva*, and are found in the following organs: the spleen, where they are usually very frequent, and in lymphatic glands, which are the most convenient.

Morbid Anatomy.—On viewing the carcass, there is usually a discharge of froth from the nose and mouth, often tinged with blood, such as is seen in cases of anthrax and in South African horse-sickness. With this condition there are always more or less morbid changes in the lungs. The carcass is usually well nourished; the subcutaneous fat frequently shows yellow pigmentation. This is also a common condition of the peritoneum, particularly of that covering the omentum. This pigmentation cannot be taken as an exclusive lesion of East Coast fever; it is more or less generally observed in cases of Texas fever also. When the lungs are affected, the chest cavity contains a considerable quantity of yellow liquid, which is free from flocculi. There is usually evidence in such cases of a chronic pleurisy, and the lungs may be found attached to the chest wall. The lungs themselves are usually œdematous, which condition may be very slight or involve a considerable portion of one or both organs. When pulmonary œdema is present, that portion of the lung tissue affected shows a condition very similar to that met with in cases of horse-sickness, the lung tissue being infiltrated with a pale straw-coloured fluid, the bronchi being filled with the same fluid in a frothy condition. The pericardial sac may contain an excess of liquid, and hæmorrhagic spots are common on the epicardium, the fat around the base of the heart being infiltrated with a gelatinous exudate of a distinctly yellow colour. Large hæmorrhagic blotches may exist on the aorta and under the endocardium, particularly in the left ventricle; and, in fact, these blood-spots, as with other piroplasmal infections, are general on the serous membranes.

The spleen, in my experience, is usually enlarged and the pulp diffluent. Some observers, however, state that there is no enlargement of the spleen in the great majority of cases.

The liver is enlarged, congested, and may be the seat of infarcts, which may be so large as perhaps to be more properly described as necrotic areas.

The writer has seen cases in cattle on the East Coast, which no doubt possessed some immunity and were suffering from a semi-acute form of the disease, in which more or less the whole of the liver was necrosed, and the endocardium covered with a kind of diphtheritic membrane.

The gall-bladder usually contains a quantity of inspissated bile, and large hæmorrhagic blotches are commonly present under its peritoneal covering.

Gastro-enteritis is always present. The abomasum is the seat of ecchymoses, accompanied by ulceration, and these blood-spots are common throughout the intestines, particularly in the small bowels, and may be seen as numerous bright red spots the size of split peas.

If the intestines be washed, they have quite a characteristic appearance, the hæmorrhagic spots regularly studding the intestinal mucous membrane, and, the membrane being swollen and yellow, the contrast in colour is most striking.

The kidneys show the most characteristic lesions of the disease. They are usually enveloped in an œdematous tissue, and show infarcts in their substance. These infarcts may vary in size from a pin-point to that of a pea; in colour from a very dark red to pale yellow, depending on whether they are recent or of long standing with the onset of necrosis. The yellow and necrotic infarcts usually show a dark inflammatory ring around their base. The infarcts in the kidney are always triangular, with their base on the surface of the organ. They may be raised above its surface or depressed below it. These infarcts are considered to be characteristic of East Coast fever, but the writer has seen them in the kidneys of animals suffering from nagana (*Trypanosoma Brucei*), where the most searching examination enabled him to exclude East Coast fever infection.

The bladder is usually normal, and contains a quantity of pale-coloured urine. The blood itself is normal in appearance, never watery or anæmic, as in Texas fever, and the blood-serum is clear.

In East Coast fever the lymphatic glands are often enormously swollen and œdematous.

Period of Incubation.—The period of incubation after exposure to infected veldt is about thirteen days. The shortest period is ten days, and the longest twenty. Theiler has, however, recorded a case of a period of incubation of thirty days. When cattle were artificially infected by placing infected ticks on them, the shortest period observed was nine days. This period of incubation must not be confused with the time an animal may run on an infected farm before showing evidences of the disease, as the infection may be quite localised, and an animal may not for some time have passed over an infected area.

Symptoms.—The earliest sign of the disease is a rise in temperature from 104° to 106° F., or even to 108° F. The animal usually exhibits a slight cough, is listless, and shows evidence of ill-health by lagging behind the herd. It is a characteristic symptom that an animal, although visibly sick, still continues to feed until the disease is advanced, when rumination ceases, and the appetite is then, of course, impaired. As with all piroplasma infections, there is muscular weakness, which is particularly evidenced by a staggering gait, affecting the hind-quarters in particular. Saliva dribbles from the mouth, and the bowels are constipated, followed, as the disease advances, in most cases by diarrhœa. There is a falling-in of the flanks and swelling of the glands at the base of the ear, which may also be observed in any of the superficial lymphatic glands. Œdema of the throat is a frequent symptom. Hæmoglobinuria is absent in uncomplicated cases, and anæmia, if any, is very slight.

The mucous membranes are sometimes icteric, and the membrana nictitans frequently shows hæmorrhagic spots, which are so characteristic of piroplasma infection. Œdema of the lungs and pleurisy may exist, and are very common in some outbreaks. When animals show marked symptoms of lung trouble, the disease has been mistaken for contagious pleuro-pneumonia. In some cases there is a marked inclination to delirium, which is also observed in Texas fever; in other cases animals may become comatose. The disease may last from six to twenty days, and the mortality is fully 95 per cent.

In investigating a suspected outbreak of the disease, with the knowledge that it is mainly introduced into a fresh centre by cattle dropping infected ticks, evidence should be sought for of animals having been brought to the locality and dying within thirty days of arrival, or of animals passing through and dying farther on the road. If such evidence can be obtained, it is most useful; but in practice, in countries such as South Africa, it is very difficult to obtain reliable evidence. Infected ticks may be carried by intermediary hosts, to which they do not attach themselves in the sense of biting, but adhere to clothing, etc.

It is known in practice that the disease usually makes its appearance from six weeks to three months after it has been introduced, and it is also found that when the crop of infected nymphæ or adults resulting from the ticks (infected larvæ or nymphæ) which have been dropped from sick or dead animals are ready to infect, they usually are only able to secure one or more animals, depending, of course, upon the intensity of the infection and the area of the grazing of the local cattle. That this is so is known by the fact that in practice it is a rule for one or two animals to die at intervals of a week or two, or even a month, until the infection becomes well established, when many animals become sick, and unless action is taken the troop or herd is annihilated.

It will be recognised that the experience in practice of isolated animals dying at intervals, as above stated, is very misleading in the detection of outbreaks, particularly so to the lay mind, who cannot conceive such a fatal disease as East Coast fever killing only an odd animal, and not making itself grossly evident for many weeks or even months; and it is this fact that leads to the concealment of many deaths, an owner putting out of his mind the possibility of the existence of a fatal disease when no further trouble occurs after the lapse of a few weeks from the first death. In practice, at all events in South Africa, there is no doubt, even with the best supervision possible, the great majority of outbreaks are not detected when the first local animal dies from the disease. It follows, therefore, if we are to deal with outbreaks successfully, we must always recognise the great possibility of infected animals having been moved from the new seat of infection prior to the existence of the disease becoming known.

Treatment.—During the past few years considerable strides have been made in checking the ravages of this disease. There are no drugs of any known value in the treatment of it.

Theiler has shown that animals may be immunised by subcutaneous and intraperitoneal injection, and insertion of spleen pulp and lymphatic glands, no doubt based on the knowledge that the forms of the parasites found in the spleen and glands are in an undeveloped stage, and the development in the animal so treated producing immunity or death.

So far, however, the mortality appears to be too high to warrant its adoption, particularly in view of the excellent results obtained by other methods described later.

Dipping in the Treatment of East Coast Fever.—The value of dipping in cleansing cattle from ticks has long been recognised, but as a mode of combating East Coast fever, particularly in areas already infected, it was not, until the recent valuable work of Watkins-Pitchford, considered to be of great practical value.

It was thought that dipping could not be carried out at a shorter period than ten days if the lives of the cattle dipped were to be considered, it being argued that if cattle were dipped to-day they would become tick-infected to-morrow, and thus contract East Coast fever from infected veldt. It has long been known that the tick chiefly responsible for the dissemination of East Coast fever—the *R. appendiculatus*—changes its host three times, and its shortest feeding-time as a larva or nymphæ is seventy-two hours, and its longest almost four days; and in consequence it was thought that until an agent was forthcoming by which cattle could be dipped every third or fourth day, dipping could not prevent the mortality on infected pastures.

Watkins-Pitchford was the investigator who showed that cattle could with perfect safety be dipped every third day, and that in grossly infected pastures the mortality from East Coast fever could be at once arrested if the dipping was carried out effectively. Watkins-Pitchford's results have been borne out in practice, and it is now a well-established fact, notwithstanding reports to the contrary, that cattle may be kept on grossly infected pastures if thoroughly and systematically dipped every third day. The writer has also seen the mortality at once arrested among cattle in badly infected pastures where the dipping had only been carried out weekly. Weekly dippings, as we know from our knowledge of the life-history of the responsible ticks, leave a loophole of escape for infected nymphæ or adults, but in practice this is of very small account. The dipping, to be thorough in badly infected tick areas, must be accompanied by the application to such predilection seats of the adult *R. appendiculatus* as the ears, around the anus, root of the tail, and the sheath, of some oily adherent material possessing tick-destroying properties. The dressing of such parts is known in South Africa as "hand-dressing."

Practical experience showed that when cattle were dipped every ten days the mortality from East Coast fever was in no way arrested, and the great success which now results from dipping is entirely due to the short-period dipping thoroughly carried out.

Further, with thorough short-period dipping the necessity for temperature camps—*i.e.*, moving cattle from infected to clean pastures, taking their temperatures daily for twenty-five days, and isolating or destroying those showing a temperature denoting the initial stage of the disease—is not so essential, and in practice can safely be dispensed with if the dipping and hand-dressing is thorough and repeated every third day. Under such conditions, any animal already infected when the treatment commenced would, of course, develop the disease, and in all probability die; but it could not infect pastures, as no living ticks would exist on such an animal.

Whatever arguments may still be put forward theoretically, the plain practical facts remain, that any owner can at once arrest the mortality from the disease by proper dipping methods, and, by a systematic policy of dipping, guard against infection.

From the above remarks it will be gathered that with a systematic course of short-interval dippings ticks can be practically eradicated from a farm, and with their diminution and eradication the many tick-borne diseases which are, and have been, the cause of 99 per cent. of the bovine mortality in South Africa (outside of rinderpest and contagious pleuro-pneumonia), and, further, the cause of the high mortality among all imported stock, will disappear. In reality, this is actually taking place. Farmers now find that their losses in stock are *nil*, and the mortality, which was very high among calves, particularly in the summer months, since the advent of dipping, has reached almost vanishing-point.

It must also be remembered that in carrying out the dipping process with reference to East Coast fever the stock-owner is at the same time stamping out the other tick-borne diseases, which, although now indigenous to South Africa, cause in the aggregate enormous losses annually.

There are many dips now on the market which can be used every third or fourth day with safety, but the one almost universally employed is from the formula of Watkins-Pitchford, as follows:

Arsenite of soda	8½ pounds.
Soft-soap	5½ pounds.
Paraffin oil	2 gallons.

To make 400 gallons of dip.

The soap is dissolved in about 5 gallons of hot water; while still hot add this soap solution in small quantities to the paraffin, and stir to a creamy lather. This makes a soap emulsion. Dissolve the 8½ pounds of arsenite of soda in sufficient hot water (about 1 gallon), and, when completely cold, add cold water up to 50 gallons. This mixture may be made in the tank. The soap solution is then gradually added, stirring thoroughly all the time. The mixture is then made up to 400 gallons by the addition of water. Most cattle-dipping tanks at the 5 foot 6 inches or 6 foot level hold about 3,000 gallons, and in practice sufficient quantities of the above ingredients may be made and added to the tank already full of water.

This dip can be used every fourth or fifth day, and its employment has saved South Africa as a whole from the ravages of East Coast fever. It is very cheap and very effectual. Some cattle-owners use the arsenite of soda alone, but the soap is desirable as an emollient, and the paraffin is of use in guarding against reinfection with ticks.

In the writer's experience, this dip is too strong for use on cattle not accustomed to being regularly dipped, and if it is used at full strength at first considerable excoriation and injury to the skin will result. Six pounds of arsenite of soda is sufficient to commence with, and lately Watkins-Pitchford has recommended a dip consisting of 4 pounds arsenite of soda, 3 pounds soft soap, 1 gallon of paraffin oil, to 400 gallons of water. In this dip cattle may be immersed every third day.

In badly infected tick areas it is as necessary to carry out hand-dressing systematically as it is to dip. The ears, around the eyes, under the tail, the sheath, the trunk of the tail, are the parts necessary to dress. It is the adult *R. appendiculatus* which chiefly attach themselves to these parts, and as the dipping fluid does not readily reach these regions, hand-dressing is the only way to destroy this variety of tick.

In the writer's experience, the best formula to use for this purpose, both for the destruction of ticks and to prevent others attaching themselves, is equal parts of bell grease (ordinary waggon-axle grease) and paraffin oil.

C. PIROPLASMOSIS DUE TO THE PIROPLASMA MUTANS.

This parasite is indigenous to South Africa, and produces fever in cattle in that country. It was named " mutans " by Theiler, who has shown it to be communicated by the red tick (*R. evertsi*) and the brown tick (*R. appendiculatus*). The disease produced has an incubation period of from twenty to fifty days, and anæmia is a prominent symptom. The *P. mutans* was thought at one time to be a form of the *P. bigeminum.*

The symptoms produced resemble those of a mild attack of redwater, and, like the *P. bigeminum*, the *P. mutans* exists in the blood of a recovered animal. Morphologically, this parasite resembles the endoglobular form of the *Theileria parva.*

D. BRITISH REDWATER.

By J. F. CRAIG, M.A., M.R.C.V.S.,
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Synonyms.—Blackwater; Moor-ill; May disease; Red murrain; Bovine hæmoglobinuria; Bovine piroplasmosis.

Definition.—Redwater is a hæmoglobinuria of cattle due to the presence in the blood of an intracorpuscular parasite, the *Piroplasma bovis.*

Occurrence.—It is enzoötic in various portions of the British Isles—in the South-West of England, in many parts of Scotland, and almost throughout Ireland. It is most common on low-lying rough pastures, such as moors (hence the name " moor-ill "), and on lands in the neighbourhood of woods. Farms on which the disease is prevalent are commonly called " redwater farms." In such farms which are afterwards given over to tillage, or where land cultivation advances, redwater decreases, and becomes almost or entirely unknown. This is due to the destruction of the breeding-places of ticks.

Redwater is most prevalent in the spring—May and June (hence the term " May disease " applied to redwater)—and in the autumn. These periods are probably the seasons at which the ticks are most numerous or most infective. At these times the cases of redwater are most frequent after showers of rain.

The animals affected are bovines. Cattle from three months upwards are susceptible. Calves are more or less immune. The disease is observed chiefly in cattle at pasture. Animals reared on infected farms show a greater power of resistance to the disease than those imported from clean districts. This is due to the fact that the former acquire a certain degree of immunity by repeated inoculations. One attack of the disease confers a very high degree of immunity. Redwater is sometimes observed in cattle which are housed; then the attack is of the nature of a recrudescence. Some writers describe a special form of redwater appearing in cows a few days after parturition. The symptoms exhibited are those described for ordinary redwater, but are always very serious. In cases where the disease has been thoroughly investigated the *Piroplasma bovis* has been found in the red blood-corpuscles. No ticks may be attached to the animals, because the attack is a secondary one, the defences of the body being broken down by the parturient state.

Etiology.—It was once thought that the disease was due to some dietetic error. Long rank grass, large quantities of turnips, or a diet too rich in carbohydrate, were regarded as causal agents. About ten years ago attention was drawn to the great similarity between this disease and Texas fever, and the blood of affected animals was examined by Nocard, Mettam, Montgomery, and others.

This led to the discovery of the true cause—the *Piroplasma bovis.* Probably there are at least two forms of the piroplasma, distinguished according to their size: (1) The large form, identical with the *P. bigeminum;* (2) the small form, which occupies only a very small portion of a red blood-corpuscle. In the latter variety the piroplasma contains much less protoplasm; the pear-shaped form measures on an average only about 1 μ long by 0·5 μ broad. The twin pairs often lie at the margin of the red blood-corpuscle, and the angle formed between is very obtuse. Of these two forms, the latter is by far the most common in Ireland. The disease is transmitted by inoculation, and the inoculating agent is a tick.

The ox ticks of the British Isles are the *Ixodes ricinus*, the common species, and the *Hæmophysalis punctata*, found in the South of England. They abound chiefly in the grass and bushes in rough, low-lying land. On the ground they do not travel far, and hence redwater is confined to certain districts or fields. They differ from the *Boophilus bovis,* which transmits Texas fever, in that they leave their host to pass through their ecdyses on the ground. They may therefore be called "three-host ticks." They attach themselves to cattle on three occasions: in the larval, nymphal, and adult stages. On each occasion they remain attached for from three to five days, and gorge themselves with blood. The adult female tick, while it is attached to the host, is impregnated by the male, which does not suck blood. After gorging itself with blood, it drops off the host; and on the ground, under rough herbage, it lays its eggs in about a week.

The number of eggs varies from 100 to 1,000. The time taken for the development of the tick depends upon conditions of temperature and moisture and the presence of a suitable host. The eggs, larvæ, and nymphs, may hibernate during the winter months. It usually requires at least five months for the complete life-cycle of the tick. The tick may transfer the disease during any of its stages—larval, nymph, or adult. It ingests the virulent blood from an affected animal. The virulent material is retained in the succeeding stages of the tick, and probably there undergoes some as yet unascertained changes. This material is injected, possibly with the secretion from the salivary glands, into the next host, and if the host happens to be a susceptible animal, redwater results. Stockman has shown that the *Hæmophysalis punctata* can transmit the disease in the adult stage, and considers that the tick is chiefly infected in the nymphal stage and infects its host when it becomes adult.

After recovery from redwater, the bovine has a high degree of immunity against the disease, but it harbours the parasite in some portion of the circulatory system for an indefinite period, probably for life. Even although one cannot detect the piroplasma on microscopic examination of the blood of a recovered animal, yet this blood, if injected in quantity into a susceptible animal, will set up the disease. This has been done after an interval of a year or more subsequent to an attack of redwater.

Animals reared on land in which the disease is prevalent become immune through moderate inoculations by ticks, but although immune, they have the piroplasmata in their blood. The disease is more commonly observed when bovines from clean districts are brought on to redwater land. Sometimes in animals which have grazed on redwater land, or have recovered from an attack, the defences of the body are broken down by some accident or untoward circumstances, such as parturition (the so-called "parturient redwater"), and redwater appears in a virulent form. These secondary attacks would explain cases appearing in animals which are housed or are grazing on land where there are no ticks. Occasionally second attacks appear after an interval of a few weeks, sometimes months or years.

Although the tick is the natural inoculating agent, the disease may be transmitted artificially by injecting under the skin of a susceptible bovine a few cubic centimetres of blood from an animal suffering from redwater. In experiments carried out with the small variety of piroplasma at the Royal Veterinary College of Ireland, direct inoculation produced no reaction. McFadyean and Stockman have recently described a small form of piroplasma (*Piroplasma divergens*), found in a case of redwater in England, which they were able to transmit directly by inoculations of blood.

The number of red blood-corpuscles containing piroplasmata in clinical cases varies from 2 to 20 per cent., but no indication is given by the number present as to the course and termination of the disease. Piroplasms are found chiefly in the red blood-corpuscles, but occasionally also in the blood-plasma. Those present in the plasma, at times present some resemblance to minute trypanosomes.

Virulence.—It is difficult to make an accurate estimate of the mortality from this disease. Probably it is less than 5 per cent. Frequently post-mortem examination of animals dead of redwater reveals some complication, such as cirrhosis of the liver. Redwater is most severe in cattle imported from clean districts. It is very fatal in the so-called parturient form.

Period of Incubation.—After direct inoculation with the large variety of the piroplasma, the average period of incubation is about nine days, but it varies from four to twelve. Under natural circumstances, when a susceptible animal is put on an infected pasture, the symptoms of redwater appear at the earliest in a fortnight, during the most favourable period of the year—viz., spring and autumn. Sometimes months elapse before the disease appears.

Symptoms.—The symptoms vary according to the intensity of the disease. In the mild form the animal does not appear to be much inconvenienced. It does not seem quite so well as usual, the coat is staring, but the chief symptom is the discoloration of the urine. At first the urine is slightly darker than usual, and forms a distinct froth in dropping on to the ground. Later it becomes red, and even black, in colour. This discoloration is due to the presence in the urine of hæmoglobin and its derivatives. The hæmoglobin is derived from the red blood-corpuscles. These corpuscles are broken down by the piroplasmata which have invaded them, the hæmoglobin is dissolved in the blood-plasma (hæmoglobinæmia), and is excreted by the kidneys with the urine. If the temperature be taken in the early stage, it will be observed to be raised to 104° or to 106° F., but later it falls to the normal again. In a few days, sometimes within twenty-four hours, the urine becomes quite normal without the animal showing any other marked symptom, and recovery promptly results. In subacute cases the animal is dull, ceases to feed or ruminate, the coat is staring or dry, the mucous membranes are slightly paler than normal, the pulse is increased in frequency, the temperature is raised up to 105° or 107° F.; later on it declines, the urine is red or black, diarrhœa sets in, but occasionally constipation is observed.

As the disease proceeds, the animal rapidly loses condition, becomes very weak, occasionally grunts or moans, the eyes sink in the orbits, the mucous membranes become yellow, the pulse dicrotic. The action of the heart is tumultuous, and may be heard at some distance from the patient. The diarrhœa is followed by an obstinate constipation, which may be accompanied by straining. This is one of the most serious clinical symptoms of the disease. In some fatal cases the urine becomes quite clear, although the constipation remains. No doubt this symptom is due to anæmia. The milk in milch cows is decreased in quantity, and in severe cases may be discoloured with blood-pigment. On examination of the blood it is found to be thin and watery, and flows very freely even from small punctured wounds, also the hæmorrhage is sometimes difficult to control. The piroplasmata are easily found in blood-films, after appropriate staining, such as that already described for other forms of piroplasmosis, and polychromasia, poikilocytosis, and basophilia, may be observed. The red blood-corpuscles are reduced in number from 7,000,000 to as few as 2,000,000 per cubic millimetre. In this condition the animal finds it difficult to swallow, and if fluid medicines are administered, they may enter the trachea and bronchi, and set up a fatal pneumonia.

In acute cases the animal soon becomes unable to rise, and may die in three to four days. In subacute cases the course is protracted for a week or ten days. Where recovery sets in, the bowels begin to act freely, the urine clears up, and the animal begins to feed and ruminate. The piroplasmata decrease very rapidly in number, and soon cannot be found on examination of blood-films.

Morbid Anatomy.—The post-mortem appearances are identical with those noted for Texas fever. The carcass is emaciated. The fat is in part replaced by a gelatinous and yellow mucoid material. The flesh is pale, moist, watery, and does not set. The blood is thin, but forms a firm clot. The spleen is usually larger than normal. The spleen pulp is firm and dark. The liver is frequently enlarged, soft, and of a yellow ochre colour, the result of fatty degeneration. This degeneration also affects to some extent the heart and kidneys. Subserous and subendocardial hæmorrhages are also present. The blood-serum is red in colour from the presence of free hæmoglobin (hæmoglobinæmia).

Treatment.—Remove the affected animals from pasture, and put in a comfortable shed. Pick off the ticks from the skin. These ticks will be found chiefly on the under aspects of the body, between the thighs and fore-arms, under the neck, on the dewlap and under aspects of the thorax and abdomen, the udder and perineum. Allow a liberal, nutritious diet, such as bran or linseed mashes, oatmeal, or maize-meal gruel. In very severe cases milk, or eggs and milk, should be given. If the animal is ruminating, grass and green food may be allowed; but hay should not be given in quantity, because of the tendency to constipation.

As for medicinal treatment, first give a laxative, such as sodium chloride (½ to 1 pound) administered in a large amount of water, or preferably in linseed tea; linseed oil (1 to 2 pints) may also be given. A few pounds of treacle may also be administered. A liberal supply of drinking water should be allowed. Frequently this is all that is required. Agents used in addition are various, and have been employed with more or less apparent success. Lead acetate (½ to 1 drachm), quinine sulphate (1 to 3 drachms), tartar emetic (2 to 4 drachms), have all been regarded as specifics, but their usefulness is not constant. Various hæmatinic stomachics and stimulants are used to combat the anæmia and the alimentary troubles—*e.g.*:

℞. Ferri carb. Potass. chlor. Sodii bicarb.	āā ʒii.
P. Nucis vom.	ʒi.
P. Gentianæ	℥i.

M. Ft. pulv.

Sig.—To be given two or three times daily in cold gruel.

Dialysed iron and hæmoglobin, given hypodermically, have also been used. Infusions of normal saline, and even of blood in severe cases, have also been suggested.

Lately, Nuttall has recommended trypan-blue as a specific against redwater. The dose used is 100 to 200 c.c. of a 1 per cent. solution of the drug in water. This solution is injected into the jugular vein. One dose is sufficient to effect a cure, but if improvement does not result, a second injection is of no service.

The trypan-blue so injected colours the tissues, and causes the skin throughout the body temporarily to turn a deep blue colour. It appears to have a destructive action on the piroplasmata. This agent has not yet been so widely employed as to permit of a correct estimate being given of its usefulness. In some forty or fifty cases in which it was tried in Ireland a fair amount of success was obtained, but it was thought desirable to administer a laxative in addition.

Preventive Measures.—The preventive measures recommended against redwater are suggested by what is known of the etiology of the disease and the life-history of the tick. The various methods for eradication of the disease are the following:

1. The extermination of ticks on redwater land will assuredly prevent the transmission of the disease. The cattle must be removed from the land, and the rough grass and the herbage burned. Where practicable, tilling the land will effectively get rid of the breeding-places of the tick. That, of course, cannot be carried out in all lands. Rock-salt on the pastures is also beneficial.

2. The attempt may be made to prevent the inoculation of susceptible animals by ticks in infected districts. This is done by periodically spraying the bovines with an arsenical or carbolic dip, or applying some dressing, such as a solution of creolin or carbolic acid, or sulphur liniment. Arsenical dips appear to be the most satisfactory for the purpose, and even destroy the ticks attached to the animals. To be effective, this treatment must be carried out at intervals of a few days during the spring, summer, and autumn. On that account this method does not appear to be an easy or economic means of prevention. It has been suggested, therefore, that the applications should only be carried out at the periods of the year at which the disease is most prevalent.

3. Another method recommended is to purify the ticks—*i.e.*, get rid of the infective material in them. This is done by leaving the land free from cattle for a period of about fifteen months. During that time sheep may be grazed over the land, these animals not being, apparently, susceptible to this form of piroplasmosis. After this period has elapsed, care must be taken that all bovines introduced into the field must never have been affected with redwater or grazed on redwater land. It would, therefore, in many cases be necessary to rear calves on the land.

To control the disease and prevent losses immunisation has been suggested. This appears to be very practicable and worthy of serious attention. The immunisation is to be carried out on all susceptible bovines about to be introduced to redwater land. The immunising agent is the defibrinated or citrated blood of affected cattle, or cattle reared on infected land. The amount suggested is 10 or 20 c.c., introduced either subcutaneously or intravenously. After the injection the animal must be isolated for about a fortnight before setting it free. The inoculation is not likely to produce, as a rule, a very marked reaction, but this reaction may vary considerably in different cases.

What is the Relation between British Redwater and Texas Fever

This question is not definitely settled. It would appear that the variety of redwater in which the large piroplasms are present is a modification of Texas fever. It differs from the latter in not being nearly so virulent, in the constant presence of hæmoglobin in the urine in clinical cases, in the absence of the chronic form, and the variety of tick which transmits the disease. That it is a variety of Texas fever is evident, judging from certain immunity experiments which have lately been made. Cattle inoculated with redwater blood have been imported to South Africa, and have shown a very high degree of resistance to South African redwater. The reason that the resistance shown by these bovines on their importation to South Africa was not so great as one might expect was due to the fact that the cattle ticks in that country not only inoculated the *P. bigeminum*, but also the *P. mutans*, the *Anaplasma marginale*, and spirilla.

II. OVINE PIROPLASMOSIS (CARCEAG).

By J. F. CRAIG, M.A., M.R.C.V.S.

This disease has been observed in Italy by Bonomo (1896), by Babes and Starcovici in the valley of the Danube, and has been carefully studied in Roumania by Motas (1902). It has also been observed in Montana, U.S.A., by Williams and Knowles.

Animals Affected.—The disease is confined to sheep, and lambs as well as adults are susceptible.

Etiology.—The cause of the disease is an intracorpuscular parasite, the *Piroplasma ovis*, which is similar in form to that causing Texas fever in cattle.

The disease is transmitted by inoculation by a tick, *Rhipicephalus bursa*. It is chiefly noted during the warm months from May to October. The adult ticks alone can inoculate the disease, but as the development of the tick may take place in twenty to twenty-five days, propagation is very rapid. Artificially the disease may be transmitted directly by injection of infected blood into healthy sheep. Recovery from one attack confers a lasting immunity.

Virulence.—The condition is very serious, and the mortality is greatest among sheep imported into infected districts. By experimental inoculation the acute form may be reproduced in three or four days.

Symptoms.—According to Motas, an acute and a benign form may be recognised. In the acute clinical form the affected animals become very dull, move with difficulty, frequently moan, eat little or nothing, fall down, and die in a few hours.

In the acute experimental form the temperature is raised to 105° or even 107° F., the sheep ceases to eat and ruminate, becomes weak, insensible, and paralysed in its hind-quarters. The pulse and respirations are very rapid. The urine presents rarely a red coloration, but resembles in colour an infusion of tea. It contains hæmoglobin. The number of red blood-corpuscles is seldom reduced beyond 4,000,000 per cubic millimetre. Death results in six to eight days.

In the benign form there is a little febrile reaction for two or three days, the general condition is very little affected, anæmia is present, but it is difficult to find piroplasms in examination of blood-films.

The **Diagnosis** is made from examination of the blood and the presence of ticks.

Lesions.—The muscles are pallid, the serous cavities may contain blood-stained effusions, the liver is lighter in colour than normal, the spleen moderately enlarged, and the kidneys friable.

Treatment is similar to that of bovine redwater. Motas maintains that injections of bile from affected into healthy animals have produced beneficial results in preventing the disease.

III. EQUINE PIROPLASMOSIS.

By Major F. S. H. BALDREY, F.R.C.V.S., D.V.H., I.C.V.D.

Definition.—Piroplasmosis is an acute or chronic relapsing fever caused by an endoglobular protozoön parasite, causing a variety of symptoms which have given rise to a multiplicity of terms more or less descriptive of the disease, and by which it is known in various localities. The disease is common to all animals, but the parasite affecting each species is specific to that particular species.

Nomenclature.—*Horses*—Piroplasmosis equi (equine piroplasmosis); Acute or chronic bilious fever of the horse; Equine malaria; Horse-sickness; Piroplasmosis bigeminum of the horse; Pyrosoma; Babesia; Typhoid fever of the horse.

French : Malaria du Cheval; Piroplasmose du Cheval.
German : Seuchenhaften Hamoglobinurie des Pferdes.

History.—Wiltshire,[1] in the Natal Almanac in 1888, described the disease under the term of "anthrax fever," and his description is similar to the one given by Hutcheon of bilious fever. Guglielme,[2] in 1899, was the first to discover the *Piroplasma equi.* Pierre,[3] in 1896, mentions "malaria" as occurring amongst horses in the French Soudan; and Dupuy,[4] in 1889, mentions that "malaria" caused great loss amongst Algerian horses in Senegambia. Rickmann,[5] in 1902, found the piroplasma parasite in the blood of horses that had died of South African horse-sickness. Pallin[6] mentions biliary fever as occurring in the plains of Bengal, Punjab, and other low-lying and feverish districts. He says that the death-rate is low; imported horses suffer the most, more especially those newly imported of English and Australian breeds; Arabs are less frequently affected, and country-breds possess a high degree of immunity. Dale,[7] in 1904, describes the disease amongst donkeys in the Transvaal, imported donkeys being practically immune. Lingard,[8] in India, has diagnosed it in English, Australian, Arabs, and country-bred horses, in 1904. According to Eassie,[9] in 1905 the disease occurred in Burma in indigenous ponies and in mules imported from the Punjab and Western China. Axe,[10] in 1906, describes the disease as occurring in India and China. According to Williams,[11] in 1907, it occurs in Central India, Rajputana, and Gujarat, amongst artillery remounts (Australian horses). Jolliffe,[12] in 1907, mentions the disease as occurring at Meerut amongst Australian horses. Berlitzer and Marzinovski,[13] in 1907, describe the disease in Russia, especially amongst imported horses, the indigenous being scarcely affected. According to Baruchello and Pricolo,[14] Piroplasmosis equi occurs in numerous districts in Italy, and also in Sardinia. Popov[15] observed in the Caucasus a disease which was curable by quinine, but the piroplasma was not demonstrated. Theiler[16] states that equine malarial fever has undoubtedly existed for a long time in South Africa, and is the same disease as the so-called bilious fever of Cape Colony and Natal. He has met with the disease in Johannesburg and other parts of the Transvaal, in horses from Basutoland, the Orange River Colony, and amongst horses imported for the South African War from England, New Zealand, Australia, and the Argentine. Verney,[17] in the Natal Monthly Reports, mentions the occurrence of biliary fever amongst horses from South America before the outbreak of the South African War. Hutcheon, quoted by Theiler, states that in Cape Colony cases occasionally occur everywhere, and at all seasons of the year, but that, as an enzoötic disease, it prevails only in certain well-defined districts. It appears to be especially enzoötic in certain low-lying farms shut in by high hills; it is seldom found in the Cape Peninsula; but, on the other hand, often on the East Coast and as far as 100 miles inland. Theiler observed it on the high and low veldt, almost always in the summer, and especially just before the rainy season. Bowhill[18] describes the disease as occurring amongst horses, donkeys, and mules in South Africa. Rogers[19] describes the disease in Algeria in three forms: (1) benign, (2) hæmorrhagic, (3) grave. Bordet and Danysz[20] found the organism in the blood of a large number of horses in the Transvaal. Peters[21] describes a form of malarial fever in horses in the west of the United States of America, but did not find the *P. equi*. Moore[22] says that the disease known in America as malarial fever has not been demonstrated as due to a piroplasma.

Geographical Distribution.—The disease is widely distributed throughout practically the whole world, but has not been verified in England, Germany, or Australia in horses, although it most probably exists in these countries.

Susceptibility.—All domesticated animals are susceptible to their own specific form of piroplasmosis in a more or less degree.

Horses in the tropics are much more susceptible to the specific fever than in temperate climes. Indigenous animals of any one particular tract acquire an immunity in that tract, but they may be susceptible to attacks in another distantly-removed area. There would appear to be a considerable racial immunity in piroplasma, but, as many observers have pointed out, this immunity is only apparent, and is due to the fact that young stock suffer from the disease and acquire a more or less resistance, although they are not necessarily free from subsequent infection. In this respect the disease closely simulates malaria in man, and the animal may carry contagion without itself showing any symptoms for an indefinite period. An animal may have acquired immunity in the district or tract of country in which it is indigenous, but this will not prevail if it be transported to another tract, and it may be attacked as severely as though it had not already suffered and recovered.[25] It is doubtful if any lasting immunity can be conferred in the tropical forms of the disease in horses as found in India and Africa. Observations have shown that animals may be attacked many times, and eventually succumb to the disease. Where the infection is indigenous, and has become of a more or less chronic nature, it is seldom that any great loss is experienced from it alone, but, owing to complications which arise, mortality may be high. This is frequently observed in India, when, if any complications intervene, death may result. It is largely the cause of death in famine years, and is responsible for many of the fatalities succeeding the various methods of preventive vaccination, when the action of such vaccine is to lower vitality temporarily, and allow the piroplasma to obtain the upper hand to such an extent that severe debility, and even death, may result. Wild animals, and smaller ones used for laboratory purposes, do not appear susceptible to any acute form of the disease, and the parasites affecting them are comparatively normal residents of the blood. I have observed in laboratory guinea-pigs that, when a high percentage of red cells is infected, there may be a rise in temperature and some loss of weight, but have never seen a fatal result. Animals imported from temperate climes to a tropical country are particularly susceptible to the attacks of piroplasmosis. This has been the cause of considerable loss and annoyance in the case of Australian remounts brought to India, and imported cattle for dairying and breeding purposes. The same has been noted by Berlitzer[25] with remounts brought to Russia from the Steppes. Eassie and Williams[36, 37] have shown that remounts in India from Australia are much more susceptible than country-breds and Arabs; also that the tropical sun has a tendency to determine attacks. The rigorous conditions of a campaign or other very hard work are also a determining factor, and induce a predisposition to it. Theiler[40] found that horses were more susceptible than donkeys, and these latter were more susceptible than mules.

Immunity.—It has already been shown under the head of susceptibility that, in certain conditions, immunity may be established; but this is not polyvalent, except in the case of *P. bigeminum*, and even here not absolutely. Experiment has demonstrated that the inoculation of blood from a recovered case is still capable of reproducing the disease, and will induce an attack of a more or less mild character, provided the dose of blood given is not too large. Recovery is rapid, and without serious complications, and subsequently a modified protection is established. This vaccination induces an actual attack of piroplasmosis, but generally in a milder form. Parasites are found in the corpuscles and plasma at the end of the usual incubation period, and may be numerous, the severity of the attack depending upon the number present. Recovery follows, but there may be a secondary reaction. In this secondary period the symptoms are more severe, although parasites are fewer than in the first period. This is due to the increased anæmia and the debilitated condition produced by the first attack.

Vaccination by means of blood from a recovered animal is strongly recommended by many authorities as the only practical means of preventing losses amongst animals about to proceed to an infected tick area. Although an animal may withstand with comparative ease the inoculation of virulent blood, it is not so with natural tick-bite infection. Such a blood vaccination will give a modicum of protection to tick-bites, but without it an animal will suffer a severe and possibly fatal attack from the biting of very few ticks. From this it will be seen that the effecting of immunity is somewhat difficult artificially. It is necessary to keep control animals for the supply of virulent blood, and the dose of this must be regulated according to local conditions. Such a proceeding is unsafe without a laboratory, and can only be done in a very empirical manner where large quantities of stock are concerned.

First Method of Immunisation.—One method adopted is to bleed affected animals during the highly febrile stage, and inject subcutaneously 1 to 5 c.c. of this blood into healthy animals. There is a marked reaction, lasting some four to seven days, with a maximum temperature of 39·5° C., recovery, and an immunity. This method is employed in countries where the disease is prevalent, or when moving animals from a tick-free area into one presumably affected with piroplasma-infected ticks. Berlitzer[25] recommends that all young equine stock be exposed to tick infection, so that they may obtain a natural immunity. It would appear that the younger the animals are at the time of the first attack the better it is, as they will the more readily recover, and even if subsequently attacked, will not suffer greatly. In the tropical forms of the disease—*i.e.*, those forms not characterised by a distinctly pear-shaped form of parasite—immunity is not so easily established artificially, although the rule holds good with regard to the immunity gained naturally by very young animals. Immunity is very hardly gained, and experiments in this direction have not been successful. Theiler[39] has shown that the protection established by an attack of piroplasmosis may be broken down by the intercurrence of horse-sickness in Africa, and a recrudescence of a severe bilious fever may result. This is due to the fact that the protozoön remains latent in the system, and not from any fresh infection, as he was careful in his experiments to exclude such a possibility. The same author[40] has found that, although a first attack induced by the inoculation of immune blood may confer some protection, it does not follow that there will be no secondary infection. This may arise from organisms latent in the system, and is often more serious than the first paroxysm. In fact, fatal results are less frequent from the first than from the secondary disease, although in the latter there may be fewer organisms demonstrable in the blood, and these somewhat modified in form.

Second Method of Immunisation.—Protective inoculation in equines is advocated, and the method adopted by Theiler[40] is to take the serum of immune—*i.e.*, recovered—animals for this purpose, and inoculate subcutaneously. This serum will contain piroplasma parasites. The intravenous method is dangerous. The use of immune mule blood is contra-indicated for horses, as it produces a severe form of piroplasmosis in them. Donkey blood may be used. The primary result is followed by a secondary reaction, often more severe than the first, but seldom fatal.

The best results were obtained by using immune foal blood. Animals that are weak or just off shipboard should not be inoculated.

Period of Incubation.—The period of incubation by natural means—*i.e.*, from tick-bites—is variable; that is to say, the time elapsing between the actual tick-bite and recognisable clinical symptoms may be as long as three weeks in duration. It will largely depend upon the amount of infection introduced and the resistance the animal possesses. By the direct inoculation of virulent blood greater uniformity may be found, but here also the time cannot be specified exactly. Theiler says that incubation from blood inoculated is five to six days. Marzinovski[41] has shown the incubation period from tick infection to be twelve days. Nuttall[44] states that sixteen to seventeen days is the incubation period in horses. The period of incubation may be stated as from twelve to twenty-one days by natural infection, and five to ten days by blood inoculation.

Method and Agency of Infection.—Piroplasmosis is entirely a disease produced by inoculation, and naturally only so by the medium of a biting insect. It is generally conceded that the mediary insect is the tick. Salmon and Stiles, Lounsbury, Theiler and Hutcheon, have demonstrated the life-history of the parasite through this arthropod in Texas fever of cattle. Owing to the fact that cases of piroplasmosis frequently occur amongst troop-horses with no ticks demonstrable upon them, it has been suggested that other biting insects may be concerned in the dissemination of the protozoön either mechanically or by cyclical means.

In some experiments I have recently made with Tabanus flies at Muktesar, it was found that piroplasmic-like bodies in the fly were found in the blood of guinea-pigs ten to twenty days after the inoculation of the salivary gland contents of the fly. These occasioned rise in temperature, but no fatal results, and the infection gradually disappeared.

Berlitzer[25] mentions *Dermacentor reticulatus* as the accepted arthropod in Russian tick fever in horses, but suspects the possibility of biting flies. In India the *Hyalomma œgyptium* is very common, and may be concerned in the dissemination of the disease, as the tick is found on all domestic animals. Jolliffe[12] has drawn attention to the absence of ticks in outbreaks of bilious fever (piroplasmosis) amongst cavalry horses in India, and suggests the possibility of some other agency being concerned in its dissemination. Lingard[8] thought that the Culicidæ were agents in this country. Brickman[38] considers that flies are the intermediary agent in what he calls "malaria of horses"—a chronic form of piroplasmosis in Africa. Theiler[39] has demonstrated that equine bilious fever is transmitted in South Africa by the tick *Rhipicephalus evertsi* (the red-legged tick). This arthropod becomes infected during the larval and nymphal stages by biting affected equines, and transmits the disease by biting during the adult stage. He failed to demonstrate that the disease was carried through the egg. Marzinovski[41] has shown that transmission in Russia is by the tick *Dermacentor reticulatus*. The larvæ are infected, and reproduce the disease in their adult stage, the protozoön undergoing a cycle in them.

Bacteriology.—Examination of blood-films from equines infected with piroplasmosis will show various forms of parasites. The type species of *P. bigeminum* is comparatively rare. These forms are occasionally met with in the erythrocytes, and more frequently free in the plasma. The commonest forms are small dot-like bodies in the substance of the corpuscle, or sometimes at the edge of the blood-cell. It is difficult to differentiate between these and precipitated stains, but by very careful focussing it will be seen if such a parasite focusses in the same plane as the cell. The bacillary and ring forms will almost invariably show a clear space around them, which stains a faint blue, and is clearly discernible in the depth of the corpuscle. Bowhill[18] has described a flagellate form of the parasite free in the plasma, or in a modified form in the blood-cells. In secondary attacks the organism is often leaf-shaped, and this same condition is noted after treatment. The size of the various forms varies considerably. The pear-shaped are from 1·5 to 3 μ in length. The cytoplasm is usually denser at the periphery, and leaves the centre often clear, or apparently vacuolated. The nucleus is situated at the broad extremity, and is a dense mass of chromatin. The widest part of the parasite is one-third that of the length. A very minute chromatin body may be demonstrated at the pointed extremity, and this is now considered to be the blepharoplast. The nucleus is single, except when division is about to take place. Parasites may be single or double. Occasionally two pairs may be seen situated in the form of a cross in the corpuscle. This form is very typical of equine piroplasmosis. Minchin[42] suggests that the flagellum seen by Bowhill may possibly be a pseudopodium, and this may be the means by which the parasite gains entry to another cell. The ring forms are apparently modifications of the above. They are small, with a dense chromatin mass—the nucleus—situated centrally or marginally. There is never any pigment, as in malaria; the second chromatin spot or blepharoplast is undemonstrable. Size varies from 0·5 to 1·5 μ in diameter. Such forms are present in the plasma also. Bacillary forms are small red-shaped organisms, containing a chromatin mass at one extremity, and measure 0·5 to 1·5 μ in length by 0·1 to 0·5 μ in width. The coccoid forms or marginal points are extremely small and difficult of demonstration. A minute chromatin spot is occasionally to be made out. The size of the parasite is from 0·1 to 1 μ in diameter.

Bacteriological Examination.—Examination of the blood is made by making very thin blood-smears and staining with any of the recognised blood stains. Leishman gives very good results, but the best are obtained with Giemsa, as follows: After drying quickly in the air, fix in methyl alcohol for five minutes. Stain in Giemsa, 1 drop to 3 c.c., in distilled water for two to twenty-four hours, preferably in incubator at 37° C. If the blue stain is in excess, wash very rapidly in methylated spirit, and then quickly in distilled water. By this stain the fine details of the parasite in the corpuscle or plasma are very well brought out. The parasite has been confounded by some observers with malaria, but it does not resemble this organism morphologically. There is never any pigment. Crescent forms and rosettes are never seen. The parasite in its early amœboid stage in the corpuscle has some resemblance to it, but very little observation is necessary to differentiate the minute differences as above. The bacillary forms have no likeness whatever to malaria, and the ring forms have no likeness to the signets of malaria. The *Anaplasma marginale* is distinctive, and the pear-shaped forms are diagnostic.

Morbid Anatomy.—The effect of the presence of piroplasmic infection is to immediately cause anæmia by the destruction of the red cells. Parasites enter erythrocytes, and use up their protoplasm in obtaining pabulum for their own existence. In the majority of cases the result is disintegration of the blood-cells, although this is not absolutely necessary, and a red corpuscle may neither break up nor disintegrate even when the protozoön partially fractures it in the act of escaping, but will regain its original shape and healthy condition. The result of this continual destruction of red cells is the breaking-up of hæmoglobin, which becomes free, and is excreted by the kidneys, giving rise to the typical symptoms of hæmoglobinuria, or redwater, which originated the name of the disease. A microscopical examination of the red urine will reveal the presence of many apparently healthy red corpuscles, showing that the excretory power of the kidney is seriously affected. This is probably due to the injury of the endothelial lining of the tubules, glomeruli, and capillaries, as a result of faulty nutrition, occasioned by the grave changes which have taken place in the blood.

The biliary symptoms are caused by the excessive work imposed upon the liver in endeavouring to dispose of the large mass of disintegrated material which tends to collect and be elaborated therein. The result is congestion, and possibly inflammation, with the inevitable consequence of jaundice, suppression of biliary excretion, etc. The loss of bile is evidenced in the intestines by constipation, foul-smelling, clay-coloured fæces, etc. Resulting from this morbid process, the intestines become gravely affected, saprophytic organisms largely increase, irritation is set up, and muco-enteritis may follow. This can only be called a complication, and is not actually piroplasmosis, although a dangerous sequel. The lungs are also frequently the seat of grave lesions, and the cause is very much the same as the above. The blood being the tissue in which serious alterations have previously taken place will induce imperfect nutrition in the lungs. This will allow of the collection of numerous saprophytic organisms, and the irritation so set up will give rise to broncho-pneumonia. This may be sufficient to occasion death, and is one of the most serious complications met with. Lingard[43] has demonstrated a toxin in the blood of animals suffering from piroplasmosis. It is conceivable that so many living organisms in the blood may excrete a substance that is inimical to the life of the vertebrate host, and seriously affects the nutritive properties of the blood and all its elements. Brain symptoms, local œdemata, etc., are all ascribable to the same cause—*i.e.*, loss of nutrition to the endothelial capillary linings, the destruction of which allows of transudation of the fluid elements of the blood, and possibly also some of the cellular structures. The consequent congestion and pressure will account for the nervous disorganisation and rapid collapse which so frequently follow. The changes taking place in the blood are not so very marked by microscopical examination. As the disease progresses, pronounced anæmia is noted by blood counts, the red corpuscles going down to 5,000,000 per c.c.

There is a varying index of leucocytosis. Eosinophilia is observed by an increase of 2 to 4 per cent. in these cells, and the large granular polynuclear eosinophiles have been observed, as described by many authors in connection with trypanosomiasis and piroplasmosis. These corpuscles are graphically known as "mulberry cells." There is some basophilic degeneration of the leucocytes, and the same degeneration may be seen in the red cells, together with poikilocytosis and polychromasis. In addition, there are the marginal points which have now been generally considered as piroplasmic organisms, and known as *Anaplasma marginale* (Theiler). It has been thought by some that these dots are a basophilic degeneration.

The post-mortem appearances are not particularly characteristic. The cause of death in horses is seldom from uncomplicated piroplasmosis. The immediate influences causing death are usually an acute inflammatory condition of the intestines, liver, or lungs; and from whichever it is, or a union of all, there will be the usual post-mortem appearances associated with such a morbid condition. In all cases there will be marked febrile lesions—*i.e.*, friable liver and engorged and pulpy spleen. The subcutaneous tissues will be more or less gelatinous, and there may be a collection of a yellowish exudate. The peritoneal cavity generally contains a scanty bilious or blood-stained fluid. The brain and nervous centres are congested, and the lungs may show a subacute broncho-pneumonic condition. The mucous membranes are congested, and frequently covered with minute petechiæ, showing the local destruction that has taken place in the capillaries. The pleura, as well as the peritoneum and other serous membranes, may be congested and the cavities contain effusion. Microscopical examination of the spleen and liver will show more parasites than the peripheral circulation—in fact, numerous organisms may be seen in the spleen when they can only be demonstrated with difficulty in any other tissue.

Symptoms.—Bilious fever, as a term describing the specific disease of piroplasmosis equi, is somewhat of a misnomer. Undoubtedly, the commonest and most frequent symptom is an acute bilious fever, but this is not always so.

Bilious fever may be divided into three varieties:

1. *Idiopathic Congestion of the Liver*, associated with digestive disorganisation, and giving rise to a sharp attack of fever, with bilious symptoms.

2. *Bilious Fever caused by Imperfect Absorption of Highly Concentrated Nitrogenous Material*, and giving rise to hæmoglobinuria of a non-specific nature. This last is a frequent condition in the tropics, and is very ephemeral in character. A horse will come in from work, and pass red-coloured urine, which upon examination will be found to contain many corpuscles, broken-down hæmoglobin, and some albumin. There is little systemic disturbance otherwise, and a dose of mag. sulph. or sodii

sulph. and a bran mash is sufficient to correct the condition, the animal being usually fit for work again the next day. The above two idiopathic diseases will be fully considered under their proper headings in Vol. II.

3. *Specific Bilious Equine Fever.*—The onset of this disease is acute, but will depend upon whether an animal has ever had a previous attack, as the first attack produces some immunity. The disease may arise subsequently, although seldom so severe as on the first occasion. Presuming, then, that the attack is the initial one, the onset is rapid. The first noticeable symptom will be dulness and a disinclination to feed. If the temperature be taken, it will be found to be 103° F., or possibly higher, even 106° to 107° F. This alarming symptom may intervene in the course of some simple ailment, such as a slight colic, laryngitis, cough, or indigestion, and diagnosis may be somewhat difficult unless piroplasmosis is suspected. Many cases of bilious fever commence with what appears to be an acute form of influenza. In uncomplicated treated cases the course is not very prolonged. There may be high temperature for a few days, and this then falls to normal or subnormal. After an interval of a day or two there may be a remission, and the subsequent anæmia and depression is more marked. Such a condition may end fatally from extreme pyrexia, or may subside and become more or less chronic, the disease continuing for a month or more, with frequent remissions and exacerbations. In this instance there will be marked wasting and anæmia. In the majority of outbreaks the initial inflammatory symptoms are followed by bilious symptoms. The mucous membranes become of a dull red-yellow colour, and petechiæ are present. The mouth is hot, sticky, and foul-smelling; the fæces scanty, slimy, and foul. The urine may be only highly coloured, but usually after a day or two will be bloody.

This latter symptom in horses is very alarming, but is not so much to be feared as the same condition in cattle. Bloody urine is not common to all cases, and in many instances is never seen. This is quickly followed by extreme debility and pronounced anæmia. There is marked leucocytosis, an increase of 1 to 3 per cent. in eosinophilia, and the so-called "mulberry eosinophile" may be found. The usual febrile symptoms are very noticeable, and extreme lassitude and asthenia are marked features. There is local œdema of the abdomen and extremities of a non-inflammatory and non-painful nature. The complications which arise are the most to be feared. Pneumonia and broncho-pneumonia are possibly the most fatal. The animal's resistance is so completely lowered that there is no vitality to fight against this dangerous condition. Colic and intestinal troubles frequently arise, giving cause for a grave prognosis. The cause of the complications has already been given. Colic is often followed by diarrhœa of an obstinate nature, and occasionally the fæces may be blood-stained and offensive.

Course.—This is either acute or chronic. In the former death may result from acute pyrexia; in the latter there is a tendency to a cachectic condition, from which an animal seldom regains his former robust health, and tends to be what is known as a "waster." In either case fatal results may occur from intervening complications, but the great tendency is to recovery, which may be prolonged or fairly rapid.

Diagnosis.—A positive diagnosis depends entirely upon the demonstration of the *P. equi* in the blood. The fact of not being able to demonstrate the parasite will not constitute a negative decision. Blood-smears at frequent intervals throughout the affection must be taken. The number of parasites in the blood varies from hour to hour of the disease, and unless frequent examinations are made, it may happen that the smears may be taken at a period of remission. If piroplasmosis is suspected, a clinical diagnosis can usually be made, even without the microscope, as the variety of the symptoms negatives the possibility of it being anything else. The disease usually occurs more or less as an epidemic, but by this I do not mean that all animals exposed to the same conditions are equally affected. It is, however, noticed that at certain times of the year, usually during the early rains or immediately afterwards in the tropics, a fair percentage of animals are attacked within a short period of each other.

Baldrey and Mitchell[45] have devised a method which may simplify diagnosis when the parasites are scanty or difficult of demonstration. It is based on the fact that when an animal cell is digested, or contains a parasite capable of digesting it, neutral red added in a 1 per cent. solution reacts to the acid production of digestion taking place in the cell, and colours the parasite contained therein red. By this means blood may be examined and the living piroplasma observed *in vitro* stained a red colour. The method adopted is to mix fresh infected blood in a Wright pipette with a 1 per cent. neutral red in pot. cit. solution. After incubating for fifteen minutes, examine under a cover-slip or hanging drop. The dilution of pot. cit. is 2·5 per cent., and this is made a 1 per cent. solution of neutral red.

Differential Diagnosis.—Having found the parasite, there can be no question as to the nature of the infection. It has already been noted that piroplasma may intervene in the course of another disease. In that case piroplasmosis itself becomes a complication, as has been noted in connection with horse-sickness in South Africa.[39] It may be mistaken for influenza, especially if typical bilious symptoms and hæmoglobinuria are not present; but there is an absence of the inflammatory coloured mucous membrane, and the microscope can usually be relied upon to differentiate. Moreover, the infection will never be so generally epidemic as influenza almost invariably is. The subsequent complications—*i.e.*, the pneumonic, intestinal, bilious, etc.—must be treated as such, and can in no way interfere, except as a complication, with the disease piroplasma *per se*. They must be considered separately and treated separately, always, of course, bearing in mind the initial cause. The microscope can differentiate between piroplasma and other sporozoön diseases, such as trypanosomiasis and filariasis. Anæmia and debility due to the presence of intestinal parasites and other causes should not cause any trouble in this respect.

Prognosis.—In uncomplicated piroplasmosis treatment may be considered to cause a satisfactory response, and a reasonable hope may always be entertained of recovery. When complications arise the anæmia and debility are so profound that one must expect bad results. They may not necessarily be fatal, but they leave considerable weakness and predisposition to disease generally.

Prophylaxis.—It is difficult to lay down any rule for the prevention of this infection. If an animal has not had an attack when young, and is exposed in an infected area, there is every chance of his contracting the disease. It is practically impossible to prevent bites from ticks in such an area, and even amongst army horses, where every care is taken by grooming, etc., attacks are frequent. The cause of the spread of infection is without doubt a tick, but it is possible that some biting flies are also concerned, and prevention of such bites is quite impossible. Many authorities recommend that all animals which are about to be exposed should be inoculated with a small dose of virulent material obtained from a recently recovered animal's blood. This induces a mild attack, and so prevents any danger from a subsequent severe attack. Theiler has had very good results by this method, and it has been employed in Russia and Germany with success. The better method of inducing immunity is to expose all young animals to natural infection by turning them out in infected paddocks. If animals are imported, and especially susceptible, they should be first given an inoculation from the blood of a recovered animal; this will induce a mild form of the disease, and give a subsequent comparative immunity. Theiler found that he always got a secondary attack in these cases, which was often more severe than the initial one. Every care should be taken of such vaccinated animals during their reactionary period; good food and careful nursing are necessary in order to prevent undue complications. Isolation of the sick during outbreaks is indicated, but is hardly of much practical use, as it is purely a disease of contagion, and not acquired except through biting insects or by actual inoculation. It is well to adopt measures of prevention by disinfection of all utensils, stable clothing, etc. It is also important that all fæces, litter, urine, etc., from infected animals should be burnt. This obviates the possibility of any infected insect remaining alive in such refuse. Many flies, such as stomoxys, lay their eggs in manure, and it is possible that the progeny of such infected flies are themselves infective, and capable of reproducing the disease. For this reason no precautions of disinfection should be neglected.

Treatment.—Medicinal treatment is very much on a plane with all other drug treatment of disease—that is, it is more or less empirical. No really satisfactory method has yet been devised. In the beginning of the disease it is advisable to give a purgative consisting of calomel, with a small dose of aloes. I have found in the tropical forms of the disease 1 drachm of calomel plus 3 drachms of aloes to prove very satisfactory. The calomel acts on the liver and relieves bilious congestion, as well as being an intestinal disinfectant by the amount of perchloride of mercury that is liberated. The aloes prevent any stasis, and relieve the large bowel of accumulated toxic matter. Quinine has but little action upon the parasite, which again differentiates it from malaria. Salicylates and salts of mercury have been found of special value in the treatment. The former, however, have a deleterious effect upon digestion by the irritation they tend to set up. Berlitzer[25] has found great benefit from the administration of perchloride of mercury subcutaneously, alternated with 5-gramme doses (about 90 grains) of calomel. Salicylate of mercury in 0·5-gramme doses (9 grains) in ball, alternated with 5 grammes of atoxyl given subcutaneously in a 4 per cent. solution, has succeeded in improving symptoms. Arsenic cannot be given in sufficiently high doses to have any effect on the parasites without causing grave intestinal troubles. The digestive mucous membrane is already very irritable, which complicates the method of treatment. Throughout the disease symptoms must be treated as they arise. A sufficiency of nourishment and easily digested food is a very important matter for consideration. Strict attention to all hygienic details is also of importance, with plenty of fresh air, warm clothing if necessary, and every comfort that can be devised for the patient. Frequent grooming maintains the activity of the skin, and hand rubbing and massage will prevent metastatic œdematous swellings. Horses that refuse all food in the form of grain, bran mashes, etc., will often eat freely of lucerne, carrots, green grass, etc., in which case they can be given these *ad lib.* Oatmeal mixed with the drinking-water is often freely partaken of. Treatment to a large extent must depend upon keeping the animal as fit as possible, with the intention of aiding it to throw off the infection.

Treatment of Complications.—These are always serious when they arise, and care should be taken to inhibit them as much as possible. The most dangerous are the lung complications. The symptoms are those of a more or less acute broncho-pneumonia, and can only be treated as such. Enteric symptoms are frequent, and evinced by diarrhœa and dysentery, accompanied in the early stage by colic. This latter is never very acute, but tends to be persistent in its intermittency. Violent purgatives are contra-indicated on account of the irritability of the bowel. Oil in combination with sedatives and antiseptics should be given, and if the diarrhœa is very excessive, mild astringents and small doses of carbolic acid and turpentine may be prescribed. The bilious symptoms and hæmoglobinuria, being symptomatic of the actual infection of piroplasmosis, are best treated as already advised. The latter symptom is not always present in acute cases, and is seldom seen in a mild case. If very acute, this symptom bodes a grave prognosis. Throughout the course of the disease, and especially during paroxysms of fever, diaphoretics, mild diuretics, and liver stimulants, in conjunction with vegetable tonics and general stimulants, are indicated. Excessive use of drugs is, however, to be avoided, and medicines are to be given as little as possible, especially if the animal reacts to one or other of the specifics mentioned earlier. Cases of sudden collapse are not frequently observed, and usually the animal becomes weaker and weaker, and dies from general inanition in untoward cases. Experiments in connection with the treatment of piroplasmosis in horses by the subcutaneous inoculation of solution of trypanblau have not yet been recorded. This treatment has met with some success in dogs, and may act as a specific in horses. It is worth trying in chronic and acute cases. Jowett[46] and Nuttall[47] have described this treatment in dogs, which has yielded considerable success. A 2 per cent. solution of trypanblau is used. It is prepared by dissolving the agent in boiling water, and filtering after cooling in order to remove any undissolved particles. The dose used in dogs is according to weight—*i.e.*, about 5 c.c. of the above solution per 15 pounds of body-weight—and injected intravenously. An animal so cured still remains infective, and its blood is capable of producing virulent and fatal attacks months after recovery, and when it is showing absolutely no symptoms, thus showing that the dog has acquired a tolerance to the parasite, and is unaffected by it. The same treatment might be tried on horses, using a 2 per cent. filtered solution of trypanblau, and inoculating intravenously or subcutaneously in doses of 50 to 100 c.c. The above author (Jowett) recommends a maximum dose being given at the beginning, tending to an over rather than an under dose. The curative action is manifest at once, and one dose would appear to effect it. The intravenous method is more rapid in action than the subcutaneous method, but it requires care, and can in no case be attempted by a layman. Animals so cured may have a relapse if subjected to adverse or trying circumstances; but they are practically immune for six months, although their blood remains infective to susceptible animals.

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IV. CANINE PIROPLASMOSIS.

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Synonyms.—Tick fever; Malignant jaundice; Infective icterus; Biliary fever of dogs.

Historical.—Hutcheon, in Cape Colony (1893), was the first to recognise this condition as a distinct disorder, and to distinguish it from distemper, with which it had, and has since, been frequently confused. Two years later Piana and Galli-Valerio discovered the parasite *Piroplasma* in the blood of a sporting dog at Milan, and shortly afterwards the South African observers confirmed this discovery, and announced the constant presence of *Piroplasma* in all cases of the disease. Canine piroplasmosis has now been recognised in most parts of Africa and in India; in Europe it has been detected in Italy and France. No case of natural infection has yet been recorded in Britain.

Loundsbury, in South Africa, was the first to prove the tick-borne nature of the disease, though the earliest workers, having in view the similarity to bovine piroplasmosis, realised and commented on the association with ticks.

Clinical Study.—The incubative period following the exposure to infected ticks is commonly between ten and twenty days; by artificial inoculation this period is shortened. The disease is ushered in by symptoms of a febrile nature, increased respiration, quick pulse, and a temperature of 103° to 105° F., and occasionally higher. In acute cases the dog rapidly becomes very weak, the visible mucous membranes are pale and sometimes icteric, and emaciation is pronounced; as death approaches the temperature becomes subnormal, and the enlarged spleen can be readily palpated. In such cases hæmoglobinuria may be seen, but this is not a constant symptom. A chronic form is frequently met with in which progressive emaciation with anæmia, and sometimes a jaundiced appearance of the skin and mucous membranes, are the most pronounced symptoms; the temperatures of these animals may show very little abnormality. Keratitis and partial paralysis have been noted in long-standing cases.

Untreated cases suffering from the acute form usually die in five to ten days from the onset of the fever, but in the case of dogs imported into an endemic area this period may be shortened. Occasionally hyperacute cases are met with, the duration of which is only a few hours. Chronic cases may last several weeks. The mortality from these forms varies much in different localities, and also according to the class of dog. Pure-bred animals and dogs imported from an absolutely clean locality suffer more severely than local born and mongrel dogs. Prior to the introduction of trypanblau treatment, the mortality in all breeds at Nairobi, British East Africa, exceeded 80 per cent.

Morbid Anatomy.—The serous cavities may contain an excess of hæmoglobin-stained fluid; the visible mucous membranes are pale and jaundiced. The alimentary tract shows little abnormality; the liver may be enlarged and congested, or is of a yellow colour, and the gall-bladder is usually distended with a thick or grumous green bile. The spleen is enlarged, often to a considerable degree, and the Malpighian bodies are prominent. The kidneys are usually congested, and the hilum may contain blood-tinged urine, in which case the contents of the bladder will be similar. The blood in the heart and other organs is pale and watery, the serum being tinged with hæmoglobin.

The Causal Parasite.—This is the *Piroplasma* or *Babesia canis*, a parasite of the red blood-corpuscles which is transmitted by means of a tick.

To the genus *Babesia* belongs also the parasite of redwater in cattle (*B. bovis* or *bigemina*), and the clinical and pathological picture produced by the species of cattle and dog is very similar.

The technique for the examination of blood or organ slides of suspected animals is similar to that recommended in the chapter on Trypanosomiasis (pp. 1041-1043); the fresh preparation is, however, rarely employed in clinical practice. Carbol thionin, which stains the parasites blue and the red blood-corpuscles green, may be employed in the absence of Giemsa or Leishman's stain.

The parasite varies in shape from round to an elongated pear-shaped body, the former being about 1 to 2 μ in diameter, and the typical pyriform bodies measure from 3 to 4·5 μ in length. Stained by one of the Romanowsky modifications, the cytoplasm appears blue, and contains a deeply red staining nuclear body.

The development and life-cycle of this piroplasma within the canine host has been well studied by Nuttall and Graham-Smith. With the maturition of the pear-shaped bodies the red blood-corpuscle breaks down, and the liberated piroplasma, being free in the blood-plasma, may attack a new corpuscle. Having entered this, the pear-shaped body assumes a rounded form and becomes larger; later it becomes amœboid, and eventually two symmetrical buds are protruded, which gradually enlarge at the expense of the parent body, and assume a pear shape, the tapering ends of which temporarily remain united. Later they separate, and each pair, the red corpuscle being destroyed, is capable of redevelopment in an erythrocyte. Somewhat complicated nuclear changes take place with this cycle.

The number of corpuscles which may be infected varies considerably during the course of the disease, and does not necessarily bear any relation to the temperature curve. In some acute cases upwards of 40 per cent. may be invaded, but an infection of about 10 per cent. is fairly high. In lingering or chronic cases, and at the commencement of infection, parasites may be so scanty as to escape microscopical detection, although inoculation of a susceptible dog with this blood will prove that the piroplasma are present. The parasite remains in the blood of a recovered animal, and for this reason relapses may occur at any time, especially in the course of other intercurrent affections.

Although single rounded forms and twin pear-shaped bodies predominate in the red blood-corpuscles, it is not rare to find multiple invasions by the latter, as many as twenty parasites being present in a single red cell. It is in smears of internal organs that these heavily-invaded corpuscles are generally found. Forms, usually pear-shaped, are also seen free in the plasma ; these are probably parasites seeking to enter new corpuscles.

Transmission.—The earlier work of Smith and Kilborne on the transmission of Texas fever (*P. bovis*) by means of ticks pointed to these parasites being responsible for the spread of the canine piroplasma. After a lengthy investigation, Loundsbury, in Cape Colony, proved that the common dog-tick of South and East Africa, *Hæmophysalis leachii*, could convey infection.

In the case of African canine piroplasmosis, the infection is also taken by the adult Hæmophysalis, but it is not transmitted until the offspring again reaches the adult stage—that is to say, infection exists, but remains dormant through each of the intervening metamorphoses, larval and nymphal. As has been noted, the piroplasma remain in the blood of a dog long after apparent recovery, although in such small numbers as to be undetected on microscopical examination; and Loundsbury showed that the progeny of female ticks taken from such a host were infective just as much as those born of a mother tick taken from a dog in the acute stages of the disease.

The transmitting agent in Europe has not yet been proved, but is believed to be *Ixodes ricinus* and *Demacentor reticulatus*, the former being found on sick dogs in Italy, the latter in France.

In India, Christophers has proved that *Rhipicephalus sanguineus* can convey the parasite. This worker showed that adults originating from mothers taken off a sick animal could give rise to the disease. This is similar to Loundsbury's observations with *H. leachii*; but Christophers further demonstrated that nymphæ (but not larvæ), the progeny of infected females, were also capable of conveying infection, and he considers it probable, but not yet experimentally proved, that a nympha feeding on a sick dog may become infective at the succeeding (adult) stage.

Both Loundsbury and Christophers have demonstrated the correctness of their views by sending ticks to England, where at Cambridge and London the disease was reproduced.

Christophers has described the development of *P. canis* within the body of *R. sanguineus*. In this he finds that certain forms of the piroplasma pass into the egg, either within the ovary or when in the oviduct, and there become zygotes, which develop in the embryonic tissues of the larva. Subsequently the zygotes break up into sporoblasts, which may be seen disseminated through the tissues, and after the following metamorphosis, these in turn break up into sporozoites, which accumulate in the salivary glands and provide the infection for the succeeding bite.

Treatment.—Prior to 1909 there existed no form of treatment which could in any way be regarded as efficient. Symptomatic therapy was adopted in most cases, though quinine, turpentine, and arsenical preparations, were from time to time advocated as possessing superior properties to previously tried drugs. The influence of aniline dyes in the experimental treatment of other protozoal infections (trypanosomiasis) led workers to conduct experiments with a variety of these dyes.

In 1909 Nuttall and Hadwen published the results of their observations upon trypanblau, a toluidin compound of sodium. The results obtained were striking, and the later publications by Jowett, in Cape Colony, on field experiments and by Nuttall leave no doubt that this drug, when efficiently administered, is practically a specific.

The dose employed by Nuttall and Hadwen ranged from 0·2 to 0·65 c.c. of a saturated solution per pound of the dog's body-weight—say from ½ to 2 drachms of a saturated solution for a 20-pound dog. The minimum effective dose has not yet been definitely ascertained, but it would appear as though a very small quantity will suffice. Administration *per os* is without effect; the solution of the drug is therefore injected subcutaneously, with aseptic precautions, as there is a great tendency to abscess formation if this be neglected. In East Africa it was found that abscesses frequently formed when the solution of trypanblau had not been sterilised, the result being that the Veterinary Laboratory undertook to put up the required doses of sterile solution in sealed tubes. It has been found there that a 2 per cent. solution of the drug obtained through Messrs. Baird and Tatlock can be prepared in the cold, and that a very small sediment remains. This solution is filled into tubes, which are sterilised in the steamer at 100° C. on each of three successive days and then sealed. The dose for a dog is fixed at 5 c.c. for an animal up to 20 pounds in weight, and in proportion above this. The results obtained have been excellent, the veterinary officers recording success in over 95 per cent. of treated cases, which now number over 300.

The action of the trypanblau upon the piroplasma has been watched by Nuttall and Hadwen, and is seen to be almost immediate, the pyriform bodies becoming rounded and finally disintegrating. Slight relapses about the tenth day are not uncommon, and a second dose of trypanblau may be indicated, but as a rule simple nursing and symptomatic treatment will suffice.

The tendency to abscess formation being overcome by the use of sterile solutions and aseptic methods of administration, the only disadvantage that remains is the blue colour imparted to the skin and mucous membranes of the dog. This effect passes off in a few weeks, and may be neglected when compared with the benefit to the animal.

GALL-SICKNESS OF CATTLE

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THE term "gall-sickness" (South African Dutch "galziekte") is commonly applied to a variety of diseases, the dominant symptom of which is anæmia and icterus, and the post-mortem finding an abnormal liver or gall-bladder. According to Theiler and Hutcheon, it is a name given by stock-owners to almost every obscure complaint affecting the digestive organs, and the latter cites hyperæmia of the liver, hepatitis, acute yellow atrophy of the liver, gastro-duodenal catarrh, and gastro-enteritis, as diseases which have been grouped under this popular name.

Theiler has made a study of the prevailing diseases expressed by this term, with the result that he has isolated four distinct organisms, all Protozoa, which on inoculation reproduce a train of symptoms coinciding with those of the natural condition. These are *Trypanosoma theileri*, *Spirochæta theileri*, *Piroplasma mutans*, and *Anaplasma marginale*. It is, however, accepted that simple digestive disturbances due to improper feeding, and also to the eating of poisonous plants, may bring about symptoms which would be identified by the South African stockman as gall-sickness.

Symptoms.—With a medley of exciting or specific causes, it is natural that no precise information can exist regarding the disease in nature. Malaise, with staring coat, a dry muzzle, and capricious appetite, ushers in the attack, and the temperature is usually elevated. The visible mucous membranes are in turn congested, anæmic, and jaundiced; the breathing is accelerated, and the initial constipation gives place to a fœtid diarrhœa, often very black in colour, though in some cases the excreta have the clay-coloured appearance associated with hepatic disturbance. Emaciation is rapid in adult animals, amongst which the mortality, especially in imported stock, is considerably higher than in calves.

Post-mortem Appearances.—These will vary according to the actual causal agent involved, but as a general rule there is an anæmic or a jaundiced condition of the subcutaneous tissues and organs, the blood is watery, and the liver may be the seat of pathological changes, ranging from an active congestion to a state of acute yellow atrophy. The gall-bladder is frequently distended with bile, which may vary in colour from light yellow to dark green, and in consistency from fluid to grumous. The spleen is usually enlarged, softened, and dark on section, and the lymphatic glands are commonly enlarged and œdematous. The lesions in the intestinal tract may be so severe as to suggest rinderpest, but they rarely exceed intense hyperæmia, with hæmorrhagic points, and in many cases œdema of the mucosa, especially that of the duodenum, is the only change observable.

Etiology.—In addition to poisonous plants and other dietetic causes, the following organisms have been met with by Theiler in association with disease presenting certain clinical analogies with gall-sickness:

1. *Trypanosoma theileri* (see p. 1040).
2. *Spirochæta theileri.*
3. *Piroplasma mutans.*
4. *Anaplasma marginale.*

Spirochæta Theileri.

This spirillum was found by Theiler in 1902, and named after the discoverer by Laveran.

Morphology.—*S. theileri* measures about 20 to 30 μ in length, and about 0·3 μ in diameter, and presents itself under the classical form of a series of S-shaped curves or undulations, or under various modified shapes—single and double loops, forms of a figure 8, etc.—such as can be assumed by a long flexible organism.

It stains by simple aniline dyes, but for detailed examination one of the Romanowsky modifications should be employed.

Transmission.—In his first experiments Theiler failed to reproduce infection by the inoculation of spirilla containing blood into cattle and other animals; but in a second communication he shows that cattle and sheep could be successfully inoculated, the incubation period being only two or three days. The blood of an animal recovered from the infection is still infective on inoculation to a clean animal.

In nature the infection is conveyed by the "blue tick" (*Boophilus decoloratus*), the larvæ born of a mother which fed on a sick animal having given the parasite to cattle in South Africa and in Paris after an incubative period of from thirteen to seventeen days.

Pathogenicity.—The disease produced by a pure infection with *S. theileri* is probably not often fatal; the most noticeable symptom is that of anæmia. *Piroplasma bigeminum* and *Anaplasma marginale* are also conveyed by the same tick, with the result that a mixed infection so frequently occurs as to render observation difficult.

Treatment.—The usual mildness of the disease demands few medicinal measures, but the drug "salvarsan" ("606") might be tried in certain cases, as it has a very pronounced beneficial effect on other diseases due to spirochæta infection.

Piroplasma mutans.

The parasite represented by this name was originally described as the "*P. bigeminum* of the immune ox" by Theiler, who later proved it to be a distinct species, and gave it its present appellation.

P. mutans is a small piroplasma, very closely resembling *Theileri parva* of East Coast fever, and occurs in the blood most frequently as rings or bacillary forms rarely measuring more than 2 μ in length, and is therefore considerably smaller than the pear-shaped *P. bigeminum*.

Transmission.—The inoculation into a susceptible bovine reproduces infection in about thirty to forty days; this at once distinguishes *P. mutans* from *Th. parva*, which is not inoculable by means of blood. It is expected that a tick will be proved as the transmitting agent in nature, but up to the time of writing Theiler's experiments with various species have been without positive result.*

Pathogenicity.—When *P. mutans* appears in the blood there occurs a rise or an irregularity in the temperature curve, the parasites become numerous—though rarely come to infect more than 30 per cent. of the red blood-corpuscles—and indications of anæmia appear. A blood-count shows diminution in red corpuscles, and microscopic examination reveals poikilocytosis, basophile granulations, and other lesions associated with anæmia. It would appear that death does not frequently occur as a result of *P. mutans* infection alone; but in fatal cases in which this parasite predominates Theiler has found gelatinous infiltration of the subcutaneous tissues of the dependent parts, an excess of fluid in the body cavities, œdematous lungs, enlargement of the spleen and lymphatic glands, an anæmia and jaundiced appearance of the mucous membranes, and in some cases an enlarged and distended gall-bladder.

Anaplasma marginale (Theiler).

For many years Theiler has spoken of bodies which were frequently observed in the blood of South African cattle under the name of "marginal points." Other observers in various parts of the world would appear to have seen at certain stages of redwater infection similar bodies. which were regarded as coccus-like forms of *P. bigeminum*.

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* It has now been demonstrated that the *P. Mutans* is transmitted by the *R. Appendiculatus* (brown tick) and the *R. Evertsi* (red tick).—Editor.

As a result of Theiler's researches it seems probable that they have nothing to do with the *Piroplasma* of redwater, but are independent parasites, to which he has given the name *Anaplasma marginale*, the generic name being due to the apparent dearth of protoplasm in their structure.

Bodies similar in appearance have been seen in various animal species which were not suffering from any recognisable disease; but in the case of cattle, although they may occur in the blood of healthy animals, as does also the redwater parasite, there appears small room for doubt that when numerous they are in etiological relationship with a disease identical with the classical gall-sickness, to which, therefore, the name *Anaplasmosis* has been given.

Geographical Distribution.—*Anaplasma marginale* has been found throughout South Africa, in British and German East Africa, Uganda, Soudan (Balfour), Trans-Caucasia (Dschunskowsky and Lühs), and there is little doubt that the "peripheral coccus-like body" of writers on Texas fever in South and North America is also of the same nature. There is no evidence of it having been found in cattle of India or Australia, but bodies of similar appearance have been seen in the blood of other animals in Australasia.

Morphology.—Viewed in an ordinary Giemsa-stained blood preparation, *A. marginale* appears as a homogeneously stained dot about 0·75 μ in diameter, situated very frequently at the periphery of a red corpuscle. By special selective staining, Sieber has drawn attention to a small central dot which is surrounded by a protoplasmic substance, all of which is deeply stained by the usual methods of staining. This writer has drawn attention to the similarity between *Anaplasma* and the *Negri*, *Guarnieri*, and other bodies found in rabies, variola, etc., and considers it should be placed in Prowazek's group of *Chlamydozoa*.

Pathogenicity.—The incubative period after inoculation is roughly twenty to forty days, but it shows great tendency to variation. With tick infection (Theiler's single successful experiment) it was somewhat longer. After their appearance in the blood, the *Anaplasma* multiply very rapidly, until within a few days as many as 30 per cent. of the red corpuscles may be invaded. Pathological changes of anæmia follow rapidly; polychromasia, anisocytosis, and poikilocytosis, are observed microscopically; to the naked eye the blood appears watery, and a great diminution in the number of red corpuscles (falling as low as 1,500,000) can be observed. This is expressed clinically by the pallor of the mucous membranes, which often become intensely icteric. Emaciation is very rapid, and the animal's breathing is often markedly distressed.

The acuteness of the symptoms varies much in different animals, being less severe in calves and animals born in an infected area than in those introduced at a later age. The mortality is also higher in this latter class of stock.

Transmission.—We have already drawn attention to the varied parasites commonly found in the blood of African cattle, of which two at least have been shown by Theiler to be spread by the *B. decoloratus*. These are *P. bigeminum* and *S. theileri*. This worker has, however, on one occasion succeeded in transmitting a pure infection of *A. marginale* by means of this tick.

Treatment.—The Transvaal Veterinary Department recommends the administration of small doses of calomel (1 drachm), followed by Epsom or Glauber's salts, and succeeded by repeated doses of quinine (2 drachms at a time). In East Africa the veterinary officers have found the necessity for accompanying this or similar treatment with cardiac stimulants and tonics. Trypanblau, so valuable in piroplasmosis, does not appear to exert any influence on *Anaplasma* infection.

TRYPANOSOMES AND TRYPANOSOMIASIS

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GENERAL REMARKS.

The scientific study of trypanosomiasis has become of importance within comparatively recent years, though in India and Africa the disease has been known and dreaded by natives for generations. To the veterinarian in the Tropics there are few diseases which offer so large a field for individual investigation; an enormous number of problems in treatment, transmission, and prevention are offered to the worker.

A perusal of the following pages will show how chaotic is our knowledge of the various trypanosomes, for, although the symptomatology of nearly all is very similar, the etiology of most leaves much to be observed, and carefully organised experiments—many of which do not require extensive laboratory facilities—will be of great benefit to future inquiry.

The present chapter is synoptic; an attempt has been made to cull the more important and practical points under each heading. The subject is too vast and at present too complicated to enter the various fields more fully, and time is not yet ripe for such dognatism as can be expected in the case of thoroughly studied diseases.

The literature on this subject has now reached extensive proportions, but those readers who desire to enter it more fully should obtain Nabarro's translation of Laveran and Mésnil's work, "Trypanosomes and Trypanosomiasis" (London, 1907, Baillière, Tindall and Cox), which contains a review of all work up to that date. For subsequent papers the *Bulletin of the Sleeping Sickness Bureau* should be consulted.

The genus *Trypanosoma* (τρύπανον, a borer; σωμα, a body) belongs to the class *Flagellata* of the *Protozoa*, and is characterised by the possession of an elongated fusiform body with a single flagellum, and is furnished with an undulating membrane.

As a protozoa this genus has affinities with the parasites of such diseases as redwater and East Coast fever of cattle, biliary fever of horses and dogs, and malaria of man, and these all agree in requiring the existence of an intermediary agent whereby to bring about transmission.

HISTORICAL.

Gruby (1843) receives the credit for the first description of this genus, made from a species found in the blood of a frog, though it is possible that Valentin (1841) had previously seen similar bodies in the blood of a trout (*Salmo fario*). Ray Lankester (1871) and Danielewsky (1885) are also among the early writers on trypanosomes in the blood of cold-blooded animals.

Lewis (1879) was the first to detect these flagellates in the blood of mammals; and the parasite found by him in the rat, *Trypanosoma lewisi*, is a common and apparently harmless parasite of these rodents throughout the tropical and parts of the temperate world.

In 1880, Griffith Evans, a veterinary officer stationed in the Punjab, discovered a trypanosome in the blood of horses and camels suffering from a disease locally called "surra." This is the first known pathogenic trypanosome.

A decade and a half later, Bruce (1895, 1896, 1897) published the results of his brilliant observations on the trypanosoma found in association with the disease "nagana," which affects all classes of domestic stock in Zululand (*T. brucei*). In 1894, Rouget detected a trypanosome (*T. equiperdum*) in a horse affected with dourine (mal du coit) in Algeria; 1901, 1902, and 1903, greeted the discoveries of Elmassian, Theiler, and Dutton and Todd, of the three trypanosomes now known as *T. equinum*, *T. theileri*, and *T. dimorphon*.

T. equinum is the cause of serious mortality amongst equines in parts of South America, notably Paraguay and Uruguay.

T. theileri, an organism considerably larger than any other mammalian form, was met with first in cattle of South Africa, and was considered as having some etiological connection with a disease known as gall-sickness. This connection now appears doubtful, and *T. theileri*, or closely allied species, is found in many parts of the world, including England and Germany.

T. dimorphon, originally found in horses in the Gambia Colony, is the first described of a series of African trypanosomes which in number and diversity are multiplying in the literature at an alarming rate.

In 1902 came the factor which has done more than any other to stimulate inquiry into trypanosomiasis—the diagnosis by Dutton of trypanosomiasis in man. A year later the same flagellate, *T. gambiense*, became recognised as the cause of human sleeping sickness.

Within recent years many new species of trypanosomes have been described, mainly from Africa, which are the cause of disease in livestock; but of greater importance are the results obtained by many workers as to the manner in which these organisms are transmitted from the sick to the healthy, a knowledge of which underlies all our attempts at prophylaxis.

Medicinal treatment has not yet yielded all that can be hoped for, but the work of many veterinarians, notably in India, shows that progress is being rapidly effected.

METHODS OF STUDY.

For the diagnosis of trypanosomiasis, and for the study of living trypanosomes, it is usual to prepare a "fresh preparation," consisting of a small drop of blood taken on the middle of a cover-glass (a ¼ inch square, No. 2, best serves the purpose for routine examination), and applied to a clean slide, so that the drop will run out and almost flow to the margin of the superimposed cover-glass. Too large a drop should be avoided, as if the blood exceeds the limit of the cover, currents will be set up, rendering diagnosis difficult. When continued examination over a prolonged period is desired, it is customary to seal the cover-glass to the slide by means of vaseline or other substance, so as to inhibit evaporation and drying.

Whilst blood is the fluid usually selected for routine examinations, there are certain trypanosomiases in which parasites are frequently very scanty in the blood. Human trypanosomiasis may be cited as an example, and also the trypanosomiasis sometimes encountered in cattle living in localities where the disease is endemic. In such instances a "gland puncture" is resorted to. A well-adjusted hypodermic syringe, preferably fitted with finger-rings, is employed, the attached needle being thrust into an enlarged superficial lymphatic gland (prescapular or precrural), and a few drops of the contained gland fluid aspirated. This is then deposited between slide and cover-glass, as is the case with blood.

Dry Preparations.—Whilst fresh preparations serve for the diagnosis of trypanosomiasis and for the examination of certain biological properties—*i.e.*, rapidity or otherwise of movement—detailed morphological study and specific diagnosis can only be attempted with dry preparations.

A film of blood, gland-juice, or of any organ, is made thinly and evenly on a perfectly clean slide, and is dried as quickly as possible by rapidly waving in the air. In damp weather it is desirable to free the slide of all moisture by passing it over the flame of a lamp or even a match immediately before attempting to make a film.

Such a slide is stained in one or other of the "protozoon" stains, of which Romanowsky's method and its many modifications are easily the most important. Unless a combined fixative and stain is employed, fixation must precede the staining. Heat, as used for the fixation of specimens intended for bacteriological examinations, must not be employed for the protozoa. Five minutes in absolute or methyl alcohol, or in the vapour of osmic acid, will complete fixation. Methylated spirit may also be employed, but the results on staining will usually be found less satisfactory.

For simplicity in field-work the combined fixative and stain have much to recommend them. In these the dye is dissolved in methyl alcohol, and is allowed to act for thirty to sixty seconds, to insure fixation, prior to dilution with water. Of this class, Leishman's method is the one most widely used by English workers. The stain may be obtained in powder from any dealer, or in the form of tablets (soloids) from Messrs. Burroughs and Wellcome, each soloid containing a sufficient quantity (0·015 gramme powder) to be added to 10 c.c. of methyl alcohol. Three or four drops of this solution are run on the slide (to prevent overflowing it is customary to draw a line with a grease pencil at each end of the portion to be stained) and allowed to act for about half a minute; then twice the number of drops of distilled or rain water are added, and the slide agitated to insure even mixing. This stain is left in contact for at least ten minutes; for most trypanosomes it is advisable to leave it for half an hour, evaporation being impeded by placing a saucer or Petrie dish over the slide. When staining is complete, wash in a good flow of water, and leave the water in contact for about half a minute to take out the excess of blue stain; rinse again in water, and dry with blotting-paper.

Giemsa's method has the advantage that the necessary solution can be carried in the Tropics with less fear of deterioration than can a solution made up with methyl alcohol alone; it has, however, the objection that fixation must be carried out independently. Giemsa's solution is made up of—

Azur. II.	0·8 gramme.
Azur. II.,-eosin	3·0 grammes.
Glycerine (chemically pure)	250 c.c.
Methyl alcohol	250 c.c.

It is advisable to purchase this solution already prepared from some well-known firm of reagent dealers, and preferably, for use in the Tropics, in small bottles of about 10 c.c. capacity. For use 1 drop of this solution is added to 1·0 c.c. of distilled or rain water. The fixed slide is supported film side downwards on two matches in a plate or dish, and the mixed stain (5 c.c. is the average amount necessary for a single slide) poured underneath. This inversion prevents much of the deposit settling on the film. The staining mixture must be prepared at the time of use, and its action is complete in about half an hour, being quicker with recently-made solutions, and being accelerated if the water at the time of dilution is warmed to body temperature, or if the staining process is carried out in an incubator or other warm place. When staining is complete, the slide is washed and dried.

For special study, methods which are difficult of execution outside the laboratory are occasionally employed—*e.g.*, Heidenhein's iron-hæmatoxylin, Breinl's method, etc.; but for field-work Leishman's and Giemsa's methods will be found very suitable.

For the study of animal reactions of a trypanosome, and for showing the morphological changes, if any, in the different susceptible animals, inoculation experiments must be undertaken.

Outside the United Kingdom this does not require any licence, and much may be done by observers who can obtain dogs, sheep, monkeys, or other animals, even although white rats and guinea-pigs are not available. A few drops of blood containing trypanosomes will usually suffice to bring about infection. This blood is prevented from clotting by being drawn into an equal amount of a 0·5 per cent. solution of sodium citrate, and in this solution, if sterile, the trypanosomes will remain virulent for twenty-four hours. The inoculation should, however, always be made as quickly as possible.

Subcutaneous and intraperitoneal injections are those most frequently performed, of which the latter is usually the more certain and reliable. For the subsequent examination of the inoculated animals the blood is taken by cutting a wafer off the tip of the ear (domestic animals, guinea-pigs, rabbits), and from the tip of the tail in rats.

Artificial culture will not be undertaken away from laboratory facilities. McNeal and Novy (1903), by employing a mixture of defibrinated rabbit blood and nutrient agar, were the first to obtain success. More recently this method has been somewhat simplified, but the results are uncertain and often difficult to obtain.

MORPHOLOGY OF A TRYPANOSOME.

The body of dead, stained trypanosomes is a more or less elongated, spindle-shaped mass of protoplasm, containing two nuclear bodies, and showing on one side a folded fin-like membrane, along the free border of which runs a thickened band, which is, in some cases, continued in advance of the body as a free flagellum.

The body consists of a *cytoplasm* surrounded by the limiting *periplast.* This former stains blue with Romanowsky's modifications, and may contain granules and more rarely a vacuole, which latter is commonly situated near to the smaller nuclear body. The smaller nuclear body is variously known as *blepharoplast, centrosome,* or *micronucleus,* and commonly occupies a position at the end, remote from the flagellum. By Romanowsky's method it stains a rather bright red.

The larger nuclear body is termed *nucleus, trophonucleus,* or *macronucleus,* and is situated about the middle of the cytoplasm. It is oval to round in shape, and stains a deep purple by the usual stains.

The rim which borders the undulating membrane arises close to the *blepharoplast.* The length and number of undulations are, of course, dependent upon the relative development of the undulating membrane, which in some trypanosomes (those commonly known as "tadpole" forms, *T. lewisi,* and others) is slight.

The flagellum is the anterior prolongation of this rim, and commonly moves in advance of the body when in motion. The length of the free portion is very variable; in the "tadpole" trypanosomes there is no free projection, and on account of the forward continuation of the body periplast, the actually "free" part even in many larger forms is small. In some examples, however, a clearly "free" flagellum can be distinguished. Both flagellum and the rim of the undulating membrane stain a deep brick red by Romanowsky.

The individuals of all trypanosome species vary somewhat in outline, and in the relative size of the different structures; in some the variations are not so marked —*e.g., T. lewisi, T. vivax, T. congolense*—as in others, notably *T. dimorphon* and *T. pecaudi.* These variations are most bewildering at times, and are apt to create an impression that more than one species is under observation. It is only by examination of slides taken at various stages of the disease and made from a series of subinoculated animals that any opinion should be expressed. As will be noted later, the factors, size and shape, are taken into consideration for the purposes of classification, but our knowledge of the genus is so imperfect that it is only within wide limits that they are of any value.

Trypanosomes multiply most frequently by longitudinal fission, which commences with the blepharoplast. From the vicinity of the daughter blepharoplast a new flagellum develops; the nucleus next divides, and finally the body separates. The division is unequal, the daughter cell being the smaller of the two.

All trypanosomes are active, free-swimming bodies, the motility of which varies considerably in different species. In a fresh cover-glass preparation the movement in most forms is detected primarily by the disturbance of the red blood-corpuscles amongst which they have become entangled. When observed in the open plasma, it will be seen that they commonly move with the flagellum in advance. In some of the smaller species movement is often sluggish, the parasite making no attempt to leave the field of vision. *T. evansi* and *T. brucei* are more active, but rarely cross the microscopic field, while *T. vivax* is so motile that it is difficult to detect more than a flicker created by its rapid transit through the corpuscles.

ANIMAL SPECIES AFFECTED WITH TRYPANOSOMIASIS.

Frog, reptile, fish, bird, and mammal, have their species of trypanosomes, all of which are apparently peculiar to the respective class. The inoculation of one order with the trypanosome of another is without result, the only exception of note being that certain mammalian forms have been able to live temporarily in the blood of some birds; it is doubtful, however, whether there is any true infection.

Among the mammalian forms, some—notably the so-called non-pathogenic organisms of rodents—are specific to their hosts, and are incapable of inoculation into other mammalian species. Examples of such are *T. lewisi* of rats, *T. cuniculi* of rabbits, *T. vespertilionis* of bats, and others of the squirrel, mouse, mole, etc. To this category also belongs *T. theileri*, a trypanosome of cattle, which has not yet been found naturally, or been induced by experimental inoculation, in the blood of any other domestic animal.

The disease-inducing trypanosomes of the domestic animals, on the other hand, are not only found in naturally affected animals of different species—horse, camel, ox, dog, etc.—but are, for the most part, capable of inoculation into all these as well as into the usual laboratory mammals, such as rats, guinea-pigs, rabbits, and monkeys. As will be seen later, two groups may be constituted from this disease-producing class, one of which is capable of positive inoculation into all the above-mentioned animals, while the other is incapable of infecting the smaller species, such as dogs, monkeys, rabbits, guinea-pigs, and rats, commonly used in the laboratory.

As far as is yet known, the human subject is not affected by any trypanosome found naturally in disease of domestic animals; on the other hand, *T. gambiense*, the cause of sleeping sickness in man, and also *T. rhodesiense*, should it prove to be a distinct species, can be successfully inoculated into domestic and laboratory animals.

Our attention will be directed here to a discussion of those trypanosomes found in the domestic animals, all of which, with the possible exception of *T. theileri*, are regarded as pathogenic, and which are the cause of severe mortality in the affected hosts.

TRANSMISSION OF TRYPANOSOMES.

Native rumour in the Punjab and North-Western India associated surra (*T. evansi*) with the large horse-fly, *Tabanus* species, and in Zululand the local population ascribed nagana (*T. brucei*) to the bite of a tsetse-fly. In regard to this latter, it was a debatable point as to whether the tsetse-fly itself was not poisonous, in the sense that it manufactured a substance toxic for the animal bitten, and it was not till Bruce conducted his experiments in Zululand in 1895 that the truth was ascertained. By a series of experiments this observer proved that the local species of tsetse—*Glossina pallidipes* and *G. morsitans*—could not *per se* give rise to the disease, and that they acted merely as transmitting agents for the trypanosome.

Rabinowitsch and Kempner (1899) showed that fleas parasitic upon rats infected with *T. lewisi* can give the infection to healthy rats; and Musgrave and Clegg (1903) have succeeded in transmitting *T. evansi* of surra from dog to dog by their agency.

Rogers (1901) proved that horse-flies (*Tabanus*) in India are capable of transmitting *T. evansi* within twenty-four hours of the infecting meal, and Musgrave and Clegg (1903) also record their successes with biting flies —species (?)—in the Philippines. During an epidemic due to *T. evansi*, which raged at Mauritius from 1902 to 1904, the genus *Stomoxys* was incriminated, though never actually proved to be a transmitting agent.

T. equinum, the cause of mal de caderas in South America, has been spread from the sick to the healthy by *Stomoxys* and *Tabanidæ* (Sivori and Lecler, 1902).

After the discovery of *T. gambiense* as the cause of sleeping sickness, inquiry soon elicited the information that its distribution and that of *G. palpalis* were very similar, and in 1903 Bruce and Nabarro proved that these tsetse-flies caught near Entebbe, Uganda, conveyed the trypanosome to a monkey.

The transmission of the avian and frog trypanosome is not well established, but some of the fish forms have been successfully transmitted by species of leeches.

The question as to whether transmission is purely mechanical—*i.e.*, such as might be affected by the needle of a hypodermic syringe—or whether the trypanosome undergoes a developmental cycle in the host fly, is one that has been much discussed. It is, of course, obvious that, granted desired conditions, such as proximity, mechanical transmission could be effected by any transitory blood-sucker; but it is an outstanding feature of protozoa that the life-cycle is complex, and that, as in piroplasma (*babesia*) and malaria, there is a special affinity between those parasites and certain species of ecto-parasites (ticks or mosquitoes). Further, the evidence in the case of the best-studied trypanosome (*T. gambiense*) demonstrates that it is almost exclusively in regions infected with *G. palpalis* that sleeping sickness occurs; consequently other biting insects appear to be of secondary importance.

In 1907 Klein proved that a cyclical development of *T. gambiense* does take place in *G. palpalis*. He showed that while mechanical transmission is possible within a few hours of the infecting meal, the fly becomes truly infective only after a minimum of about sixteen days, and that from this period trypanosomes are transmitted at all succeeding bites. This has been confirmed by Bruce and his co-workers in Uganda (1909), and it has since been demonstrated that cyclical development occurs also in the cases of *T. vivax* and *G. palpalis*, and of *T. lewisi* and a common rat flea in England.

It will require much time to prove this method of transmission for all the known mammalian trypanosomes, more especially since some flies are very difficult to rear artificially and even to keep alive in captivity; but the principle of cyclical development being established, it must be admitted that other methods of transmission—*i.e.*, mechanical—become of less importance, excepting under unusual conditions.

There remains to be decided the very important question as to whether any given trypanosome species can be conveyed by more than one genus of fly, such as *Tabanus*, *Stomoxys*, *Glossina*, or by more than one species of any genus, as, for example, *G. palpalis* and *G. morsitans*. We know that a single species of fly, *G. palpalis*, can transmit *T. gambiense*, *T. vivax*, *T. cazalboui*, and *T. dimorphon*, the two last named being flagellates which have been found to occur naturally in districts far removed from any known *G. palpalis*. The assumption would then be that it is possible for a trypanosome to be carried by, and probably to undergo a cyclical development in, more than one species of tsetse-fly, and, as a result, it behoves us to view with suspicion all such biting flies as a plague to domestic animals in localities where trypanosomiasis exists.

Recent work by Klein and his colleagues and by Roubaud upon the fly, and by workers of the Liverpool School of Tropical Medicine upon the mammalian host, has helped to demonstrate the developmental stages of trypanosomes, and to formulate some working view of the cycle undergone, but the question cannot yet be regarded as settled.

TREATMENT.

In the early years of our knowledge of trypanosomiasis in animals, and especially in the case of surra (*T. evansi*) in India, nearly all the drugs of the existent Pharmacopœia were tried. The results were negligible in all cases except with arsenic. Livingstone recommended this drug in the middle of the nineteenth century, and Lingard with surra, and Bruce with nagana, subsequently ascertained its beneficial influence. The action of arsenic has been recently studied by Holmes, whose method of treatment will be given in detail later.

Ehrlich and Shiga, working with mal de caderas, introduced the study of aniline dyes in the treatment of trypanosomiasis. Their initial work with trypanroth was followed by that of other writers on methylene green, crysoidine, etc., and on the group of benzidine dyes studied by Mésnil and Nicolle.

The effect of various sera from immune, recovered, or insusceptible animals has also been tried, but the results have never got beyond the experimental laboratory stage, and the animals successfully influenced were in most cases small, such as mice and rats.

Extracts of various organs, cultures of bacteria, and various forms of light rays (X, Finsen, and radium), have also been tried without tangible result, and the influence of heat or cold is also as yet negligible.

With preparations of various metals the results have been more hopeful; particularly is this the case when two have been tried in combination, the theory of action being that the one may expel the adult trypanosome from the blood, the second acting upon the resting forms which are believed to occur in certain internal organs.

One of the greatest difficulties in the path of the experiments with arsenic has been the toxic effect produced when doses sufficiently large to influence the trypanosomes have been administered. This to a great extent was overcome by Wolferstan Thomas, who introduced the use of the drug atoxyl, a meta-arsenic-analide, which is about forty times less toxic than Fowler's solution, and can be administered by subcutaneous or intravenous injection. The action of this drug destroys all or most of the adult trypanosomes in the peripheral blood within a few hours of administration, and in some cases apparent cures have been effected by it alone. In most successful instances, however, the preliminary treatment by atoxyl has been followed by the subsequent exhibition of an agent acting upon the "resting bodies" or possessed of a more prolonged action. Arsenic, perchloride of mercury, and trypan-red, have all been employed in this manner.

In an animal under treatment the trypanosomes often appear somewhat degenerated, and may show temporary morphological peculiarities. The most interesting feature with regard to atoxyl treatment, however, is that in unsuccessful cases the trypanosome acquires a great tolerance towards the drug, and this strain subsequently passed through a series of untreated animals will still retain the property of atoxyl resistance, an injection of atoxyl to such an affected animal being practically without action.

It is an interesting and important fact that while animals which have naturally recovered from a trypanosome infection appear to have considerable immunity against a reinfection by the same trypanosome, those cured by artificial treatment are as susceptible as they were prior to exhibition of the medicament.

Holmes has been particularly successful in his treatment of surra by an atoxyl-arsenic combination and with arsenic alone (see p. 1079).

PROPHYLAXIS.

Until quite recently, there had appeared no system of treatment applicable to domestic animals which is at all efficacious, and it will be many years before one can be perfected for success in practice, even should Holmes's method find support in diseases other than surra. It is equally unfortunate that our limited knowledge of the etiology of trypanosomiasis prevents us from adopting any precise rules for its prevention.

From the remarks on the subject of transmission, it will be obvious that for a continuation and spread of the disease three factors are necessary—(1) the original host of the trypanosome; (2) a new susceptible animal; and (3) the intermediary or transmitting agent. If it be possible to eliminate any of these factors, the chain of continuity is broken and spread of infection is impossible.

The Original Host of the Trypanosome.—We shall see that in the case of mal de caderas, a rodent, *Capybara*, is blamed for acting as reservoir for the trypanosome; in nagana, wild game, the buffalo, and various species of antelope, may act in this manner; and in surra, the camel and cattle harbour the trypanosome for varying periods, often several years. All infected animals, of course, fall into this category, but in many domestic species the duration of disease is short, and they are less dangerous than those in which the infection runs a chronic or subacute type. Any means which will reduce the risk of domestic animals or the transmitting agents coming into contact with the sick will reduce the risk of infection.

The Transmitting or Intermediary Agent.—At the present time all biting flies must be regarded with suspicion in this connection. In parts of Africa the genus *Glossina*, tsetse-flies, are undoubtedly to be considered the most serious offenders; and, as has already been noted, an actual multiplication of trypanosomes occurs in certain species, rendering them not only a transmitting agent, but also a true host from which infection may result for at least two months after feeding on a sick animal.

Until final proof of transmission be obtained with *Tabanidæ* and biting *Muscidæ*—*i.e.*, *Stomoxys*—and evidence regarding cyclical development be forthcoming, their importance in districts already infected by *Glossina* will remain secondary, although in India, America, and North Africa, and other zones free from tsetse, they are to be regarded as primary factors.

The distribution of *Glossina* is limited to zones or belts, which may be avoided if not of very large size (50 to 100 miles), as they frequently are; but *Tabanidæ* and *Stomoxys* are spread over a much wider area, and extend into towns and populous places near which tsetse do not, as a rule, live.

Much has been written on the relationship existing between the widely distributed *G. morsitans* (and with it *G. pallipides*) and big game, some writers holding that the former are dependent upon the latter for existence, and that destruction of the game will cause the death or withdrawal of the fly; others, again, regard the association as fortuitous, in so far that both have a predilection for the same type of country. Within localised areas it is possible to repel *Glossina* by destroying or altering the character of its haunts, but to apply the procedure of game destruction to the whole of the infested parts of Africa would be impossible.

The genus *Stomoxys* is of limited importance as an agent passing from wild game to domestic stock, for it is a species that lives particularly around houses and stables; but as an agent which may be instrumental in transmitting the disease from a sick to a healthy domestic animal it must be regarded with grave suspicion. Scrupulous cleanliness around horse and cattle sheds and the destruction of all manure, in which this fly breeds, will do much to control it numerically.

The New Susceptible Animal.—Absolute protection from the bites of flies is almost impossible, but the adoption of such means as avoiding known haunts of virulent species, marching by night when flies are usually dormant, smearing with fat and volatile oils, which repel the attack of insects, will often be possible. In places where *Stomoxys* are numerous, and the possibility of introducing infected animals cannot be excluded, stables should, where practicable, be made fly-proof by means of mosquito gauze, such as is done in districts where horse-sickness is rife.

CLASSIFICATION OF PATHOGENIC FORMS.

From what has been written, it will have been noted how closely connected, either in form or habits, or both, are all the pathogenic species which have been mentioned. Unlike many parasites of the metazoa, where classification by external or structural characteristics is possible, the protozoa in all sections present much difficulty, and this difficulty is particularly marked in the genus under review.

At the present time the methods available for classification are two—(1) A comparison of the morphological features, and (2) a comparison of the biological features, especially the "animal reactions," which demonstrate the susceptibilities of the various domestic or laboratory animals.

Further investigation will no doubt reveal specific points of difference either in morphology or biology, more particularly when the whole phenomena of transmission and the life-cycle through the fly host are known; but until this time is reached, doubt must remain regarding the identity of many species.

By adopting certain standards of animal reactions we can arrive at a diagnosis of *T. gambiense* and *T. rhodesiense*,* which, so far as is yet known, are the only forms pathogenic to man,† and those trypanosomes of the lower animals which are practically peculiar to their hosts can be eliminated. Examples of these latter are *T. lewisi* in rats, *T. cuniculi* in rabbits, and the trypanosomes of frogs, fish, etc.

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* It has been suggested that *T. rhodesiense* is a form of *T. brucei*, to which it is related morphologically, which has acquired pathogenic properties towards man.

† Cruz has recently described a disease of man in Brazil in which flagellates resembling trypanosomata are found; the life-cycle of these forms warrants the creation of a new genus, *Schizo trypanosoma.*

On morphological grounds we can identify *T. theileri*, which is of very large size, and *T. equinum*, which possesses a uniquely small blepharoplast.

On clinical grounds it is possible to diagnose *T. equiperdum*, since the disease caused by it—dourine—shows characteristic symptoms and features which differ from all other trypanosomiases.

The remaining species of trypanosomes may be roughly classified into two groups, each possessing fairly distinctive attributes.

A. Trypanosomes Pathogenic towards Most Domestic Animals, and producing a Rapidly Fatal Infection in the Usual Laboratory Animals.

1. Monomorphic (*i.e.*, of *relatively* constant size).

   (*a*) Large, from 20 μ to 35 μ in length, and carrying a distinct free flagellum. with a well-developed undulating membrane.

   Type : *T. evansi.*

   (*b*) Small (tadpole forms), from 9 μ to 16 μ in length, carrying no, or practically no, free flagellum ; undulating membrane poorly developed.

   Type : *T. congolense.*

2. Di- or poly-morphic trypanosomes, which may occur both as short forms, devoid of a free flagellum, and as long forms, resembling the *evansi* group.

   Type : *T. dimorphon.*

B. Trypanosomes Pathogenic towards the Domestic Animals but without Apparent Effect when inoculated into the Usual Laboratory Animals.

Type : *T. nanum.*

The following species will receive notice in the succeeding pages :

T. equiperdum.
T. equinum.
T. evansi.
T. brucei.
T. sudanense.
T. venezuelense.
T. hippicum.
T. congolense.
T. montgomeri.
T. dimorphon.
T. pecaudi.
T. nanum.
T. vivax.
T. cazalboui.
T. uniforme.
T. theileri.
T. gambiense
T. rhodesiense.

TRYPANOSOMA EQUIPERDUM (Doflein, 1901).

The causal agent of DOURINE. This trypanosomiasis is described in detail (see p. 1067)

TRYPANOSOMA EQUINUM (Voges, 1901).

Synonym.—*T. elmassiani* (Lignières).

The causal agent of MAL DE CADERAS in South America (see p. 1061).

GROUP TRYPANOSOMA EVANSI.

The trypanosomes of this group present morphological affinities, and in stained preparations all closely resemble *T. gambiense*. The classification is artificial, and is based largely on the geographical distribution or on the supposed transmitting agent. The group includes *T. evansi* of India, where no *Glossinæ* occur, and *T. brucei* of Africa, which is met with chiefly in *Glossinæ*-infected zones; *T. venezuelense* of Venezuela and *T. hippicum* of the Panama Canal zone. *T. soudanense* is constituted as a specific entity, since animals believed to be immunised to—*i.e.*, recovered from—*T. evansi* infection are still susceptible.

Trypanosoma Evansi (Steel, 1885).

Discovered by Griffith Evans in 1880.

This trypanosome and the disease it causes (SURRA) are fully described elsewhere (see p. 1081).

Trypanosoma Brucei (Plimmer and Bradford, 1899).

The cause of NAGANA in Zululand (see p. 1063).

Trypanosoma Soudanense (Laveran, 1907).

The cause of a disease mainly affecting horses and camels in Northern Africa (French Soudan) and Algeria, known locally as "MAL DE ZOUSFANA" and "EL DEBAB."

Historical.—In 1903 Rennes described under the former name the clinical picture presented by infected animals, and differentiated the disease from dourine (also a North African trypanosomiasis). Two years later the brothers Sergent published their observations on the disease known in Algeria as "el debab." In 1907 Laveran, who had made use of the trypanosomes obtained from these two diseases, regarded them as one and the same, and very closely related to, if not identical with, a trypanosome he had obtained from camels in the Upper Niger region of French territory, and to which he gave the name *T. soudanense*.

Morphology.—The trypanosome is indistinguishable from *T. evansi*. Laveran based the distinction of the species upon his immunising experiments, in which he found that animals immune to surra were susceptible to this trypanosome.

Clinical Study.—The disease presents the symptoms common to most trypanosomiases—anæmia, progressive wasting, œdemata, and lesions of the eyes. The duration in horses is from four to six months. Camels may live for several years.

Animals Affected.—Cattle, equines, sheep, goats, dogs, and laboratory animals, are susceptible to inoculation, the duration being similar to, or slightly longer than, that due to *T. brucei*.

Transmission.—Ed. and Et. Sergent showed that *Tabanidæ* could transmit infection by interrupted feeding, and on one occasion by an intermittent feed twenty-two hours after biting the infected animal. They did not succeed with *Stomoxys*.

Native opinion blames the *Tabanidæ* the vernacular for which is "Debab." There are no tsetse in the infected areas.

Trypanosoma Venezuelense (Mésnil, 1910).

In 1905 R. Rangel described a trypanosomiasis in Venezuela, which was considered as mal de caderas (*T. equinum*).

The symptoms were those of a progressive pernicious anæmia, with œdemata and sometimes paraplegia.

Recently Mésnil has examined some blood-films sent from there, and finds the trypanosome to resemble *T. evansi*, and to differ from *T. equinum* in possessing a well-defined blepharoplast.

Trypanosoma Hippicum (Darling, 1901).

Darling has studied an epidemic which occurred amongst the mules working on the Panama Canal in 1909. The symptoms were those of progressive anæmia, œdemata, and irregular fever, and the duration was one to two months or less.

Morphology.—The author describes two types of trypanosome, the one 28 μ in length and the other 16 μ to 18 μ; the flagellum is well developed.

Animals Susceptible.—All the laboratory animals are susceptible; of the domestic animals experimented upon, a calf did not acquire infection.

Trypanosoma Congolense (Broden, 1904).

Historical.—Broden describes several trypanosomes in the Congo Free State, to one of which he gives this name. Dutton, Todd, and Kinghorn, apparently refound the same trypanosome in the Congo, but associated it, as did Mésnil at first, with *T. dimorphon.* In 1908 Laveran, by his method of cross-inoculation, showed that animals believed immune to Broden's trypanosome were susceptible to *T. dimorphon* (Paris strain), and admitted the species. Kinghorn and the present writer found this trypanosome in Northern Rhodesia.

Morphology.—*T. congolense* is small and of the " tadpole " type, measuring only 10 μ to 16 μ in length, and *is devoid of free flagellum.* The undulating membrane is very poorly developed. It therefore differs essentially from the *evansi* group, in which the undulating membrane and the free flagellum are well defined, and from the *dimorphon* group, in which both " tadpole " and " long " forms occur (*vide infra*).

Species Affected.—*T. congolense* has not yet been found in countries where Equidæ are numerous. Bovines, sheep, and goats, are susceptible, the disease being usually somewhat prolonged, and lasting four to six months or more. All the laboratory animals are susceptible, though, according to Laveran, guinea-pigs have at times a marked resistance, and in mice the duration is very variable. The symptoms manifested show nothing which differs from those seen in other subacute trypanosomiases.

Transmission.—This trypanosome occurs naturally in districts invaded by *G. palpalis*, and in others where the only representative of the tsetse-flies is *G. morsitans.* Should the trypanosome of Theiler in Portuguese East Africa, and of Edington in Zanzibar, prove to be *T. congolense,* it will then be known to occur also in localities where no *Glossina* are found, and where *Stomoxys* and *Tabanidæ* probably act as carriers.

Theiler found a trypanosome on the Limpopo River and on the Zambesi River which is morphologically similar to *T. congolense*, but which appears non-pathogenic for the guinea-pig. It may be noted that the trypanosome found by Bruce and his colleagues in Uganda (1909), and described as *T. pecorum,* is also non-pathogenic for this animal.

The trypanosome found by Egington in Zanzibar appears identical with *T. congolense.*

In 1908 Kinghorn and the present writer met with a trypanosome in a cow near Lake Tanganyika which in its general morphology and pathogenicity resembled *T. congolense.* The average width of the parasite was, however, considerably greater, and this morphological peculiarity was retained in subsequent passage. Laveran has suggested the name *T. montgomeri* for this form, but its individuality is not yet established.

GROUP OF TRYPANOSOMA DIMORPHON.

Dutton and Todd described a trypanosomiasis of horses in the Gambia Colony in 1902, and being one of the first African trypanosomes to be studied after *T. brucei,* the parasite they described has received considerable attention. It has proved a convenient species to which to assign any animal trypanosomes that do not approximate to *T. brucei*, the result being that much confusion exists. If, however, the description of the original authors be borne in mind, this group now presents less difficulties.

Bruce has created the name *T. pecorum* to include all trypanosomes of the "tadpole" class. This grouping together of what will probably be shown to be distinct species is apt to increase rather than diminish confusion.

Trypanosoma Dimorphon (Dutton and Todd, 1904).

Dutton and Todd found ten horses to be naturally infected in the Gambia, and studied the trypanosomes in these animals and in inoculated laboratory animals. One naturally infected case was sent to England, and this has furnished a strain to various laboratories. The animal on which the work of Dutton and Todd was carrried out died in the Gambia, and its strain has not reached Europe. As the description furnished by Dutton and Todd differs materially from those of workers in Europe, this fact should be remembered, since it is probable that two distinct species were involved. Our remarks here apply to the original strain studied in the Gambia; notes on that sent to Europe are under the heading "*T. dimorphon, sensu* Laveran and Mésnil."

Morphology.—*T. dimorphon* may occur in any of three forms—"tadpole," "stumpy," and "long." Frequently one or other form predominates at a single examination, but all may be seen together.

"Tadpole" forms measure 10 μ to 15 μ in length; there is no free flagellum, and the undulating membrane is at most represented by a single fold. These forms resemble *T. congolense*. "Stumpy" forms measure about 16 μ to 20 μ; the parasite is broader, and has occasionally a short flagellum. "Long" forms are from 25 μ to 32 μ in length, carrying a well-defined free flagellum and an undulating membrane resembling that of the *evansi* group.

Dutton and Todd found that "tadpole" forms prevailed early in the infection, and "long" forms were most numerous towards death.

As this strain was not sent to England, the original has died out; but Kinghorn and the writer found a trypanosome in Northern Rhodesia which in morphology and animal reactions responded to the original description, and we can therefore oppose the suggestion that Dutton and Todd were dealing with a mixed infection of one animal by two trypanosomes, one resembling *T. congolense*, the other *T. evansi*.

Animals Affected.—In horses the infection appears rather prolonged, and trypanosomes are scanty until towards death. The same appears to hold good for cattle. In sheep and goats Montgomery and Kinghorn noted a marked periodicity in temperatures and in the occurrence of the parasites in the blood.

All laboratory animals are susceptible, the disease in them being rather acute.

Transmission.—We only know of two centres from which *T. dimorphon* (Dutton and Todd) has been obtained—Gambia and North-Western Rhodesia. In the former *G. palpalis* is the common tsetse-fly, while in the latter only *G. morsitans* was found.

Certain evidence of a circumstantial nature was adduced in Rhodesia indicating that *Stomoxys* could carry infection in a cattle kraal containing both sick and healthy animals, but no experimental proof was obtained, and the experience in Southern Rhodesia, to which infected cattle have been introduced, would appear to negative the suggestion.

Trypanosoma Dimorphon (*sensu* Laveran and Mésnil).

Laveran and Mésnil and Thomas and Breinl, working with the strain sent to Europe from Gambia, found the trypanosome differed in essential morphological points from that described by Dutton and Todd. These workers were unable to recognise "long" forms having a free flagellum, and in their descriptions only a "tadpole" and a longer form, measuring up to 22 μ, but without a free flagellum, are mentioned.

The pathogenic powers of this trypanosome are similar to those of *T. dimorphon, sensu* Dutton and Todd, but there can be little doubt that the parasites are distinct species. The specific name, *T. confusum*, was suggested by Montgomery and Kinghorn for the strain in Europe, but the title is preoccupied for a bird trypanosome, and cannot therefore be employed.

TRYPANOSOMA PECAUDI (Laveran, 1907).

The cause of "BALERI" in French West Africa.

Historical.—Cazalbou, writing in 1904, described the trypanosomiases then known in the French Soudan, and referred to "MBORI," due to a variety of *T. evansi;* "SOUMAYA," due to *T. cazalboui;* and "BALERI," which is a disease of horses especially, and occurs in districts infected with *Glossinæ*. Cazalbou at that time noted the morphological peculiarities of the parasite, which, without doubt, has often been regarded as *T. dimorphon* by other observers. In 1907 Laveran showed that the morphological features of this trypanosome were sufficient to warrant the creation of a new species, which he called *T. pecaudi*. It appears possible that a trypanosomiasis of mules observed by Head and Balfour in the British Soudan is also due to this parasite, and not to *T. dimorphon*.

Morphology.—*T. pecaudi* occurs in the blood under two forms—a long and a short; both are found together at all stages of the infection, thus differing from *T. dimorphon*, in which it is common for one or other form to be present at varying stages of the disease.

The "long thin" form measures from 25 μ to 35 μ in length, and is about 1·5 μ in width; the undulating membrane is narrow and well folded, and the flagellum is long and free. The "short broad" form measures about 14 μ to 20 μ long, and from 3 μ to 4 μ in width. The undulating membrane is broad, though not markedly folded, and there is no free flagellum.

Animals Affected.—In the horse and dog the affection is acute or subacute, death resulting in the former in two to four months, in the latter in fifteen to twenty-eight days. The symptoms shown are irregular temperature of a paroxysmal nature, with the coincident appearance of parasites in the blood, œdemata, conjunctivitis, and petechiæ on the membrana nictitans, and progressive emaciation. Some horses appear to recover.

In mules and donkeys the infection is sometimes very chronic. Cazalbou records the case of a donkey dying after 500 days.

Cattle, sheep, and goats do not seem to suffer much from the natural disease, which is chronic in them, and many recover, but they are susceptible to inoculation.

Laboratory animals are susceptible, and die of an acute infection; rabbits, and sometimes guinea-pigs, are the most resistant.

Transmission.—The evidence adduced by Cazalbou shows that natural infection is, chiefly at least, confined to the river-beds and tsetse-infected districts.

Roubard (1910) has demonstrated that *T. pecaudi* multiplies in *G. palpalis*, and produces what he terms a "permanent culture" in the fly, but he was unable to prove any infection in the proboscis itself.

Bouet and Roubard obtained one positive transmission result with *G. palpalis;* other experiments at the same time were negative.

GROUP OF TRYPANOSOMA NANUM.

The members of this group—*e.g., T. nanum, T. vivax*, and *T. cazalboui*—possess a striking characteristic, in that they are non-pathogenic for the laboratory animals, dogs, monkeys, rabbits, guinea-pigs, white rats, and mice. Though *T. nanum* differs markedly from *T. vivax* in morphology, this peculiarity in "animal reactions" is sufficient to permit of their being grouped together, with the former species as type, owing to its prior description.

Trypanosoma Nanum (Laveran, 1904).

In 1904 Balfour found a trypanosome in the blood of cattle in the Soudan which, on inoculation into a dog, two rabbits, and a monkey, produced no result. On account of this and of the morphology Laveran gave the specific name.

Wenyon apparently regained the trypanosome in the Soudan in 1908, and demonstrated that it might also assume a larger form. Montgomery and Kinghorn, in the same year, associated a trypanosomiasis of cattle in Northern Rhodesia with the species, and also noted the occurrence of long forms. Bruce and his colleagues encountered a similar trypanosome in Uganda.

Morphology.—The classical form is of the "tadpole" class, and measures from 10 μ to 15 μ in length, and 1·5 μ to 2·5 μ in width. The undulating membrane is practically absent, and there is no free flagellum.

The long forms seen by Wenyon and by the writer and Kinghorn measured from 20 μ to 25 μ in length; in appearance they differ slightly from each other, and further observations are required to show their connection with the "tadpole" forms.

Animals Affected.—Cattle, sheep, and goats have been found naturally infected. Dogs, guinea-pigs, and rats are resistant to the Rhodesian strain, which therefore conforms to the original description. Wenyon apparently succeeded in infecting a young dog and the local gerbils.

There is no positive evidence as to transmission, though certain indications point to flies other than tsetse being responsible in the Soudan and Uganda. In Rhodesia *G. morsitans* was numerous.

Trypanosoma Vivax (Ziemann, 1905).

Historical.—This trypanosome was first found by Ziemann in the German Cameroons in 1904, and was so named on account of its extraordinary motility in fresh preparations. In 1906 Laveran came to the conclusion that the trypanosome associated with the disease "souma" or "soumaya" was a distinct species, to which he gave the name *T. cazalboui;* these two trypanosomes are very closely related, if not identical. Subsequent observers (Montgomery and Kinghorn in Northern Rhodesia, and Bruce and his colleagues in Uganda) have associated their forms with Ziemann's species, as it has priority, although *T. cazalboui* has been better studied. A very similar trypanosome has been found in Italian Somaliland.

Morphology.—The parasite measures on an average from 22 μ to 28 μ in length, including the flagellum, which is almost 4 μ to 5 μ. There is very little tendency to dimorphism, all parasites at an examination being nearly of the same size and shape. The posterior (aflagellar) end is usually rounded, and shows a well-defined round blepharoplast; the anterior portion tapers evenly towards the flagellum. The undulating membrane is narrow, and is but slightly folded. When blood containing *T. vivax* is examined in a fresh preparation, the parasites can only be detected by the slight disturbance of the corpuscles, for the trypanosomes pass across the field of vision "like an arrow." It is only in preparations which have been made for some time, and in which parasites have become sluggish, that the outline of the trypanosome can be seen.

Montgomery and Kinghorn noted that when the trypanosomes were held up by massed corpuscles or by fibrin, they appeared to make a fixed point at the posterior extremity, and, by lashing around on this, to throw out the corpuscles like the rays of a catherine-wheel.

Animals Affected.—Ziemann, in his original paper, stated that he had infected wild rats; but it is possible that the inoculations were made with blood not containing *T. vivax* alone, for no subsequent writer has succeeded in infecting rats, rabbits, guinea-pigs, dogs, or monkeys. The larger animals are susceptible.

Laveran's species, *T. cazalboui*, is associated with a disease of cattle in the French Soudan known as "soumaya" or "souma," which causes serious mortality in some of the localities where the natives congregate their cattle for grazing or for sale.

The onset of the disease is insidious, and the animal may live for twelve months after infection. The symptoms are irregular fever, with anæmia, emaciation, and sometimes œdemata. This disease has also been seen in equines, the horse, which dies in about fifty days, being the most susceptible. Sheep and goats are susceptible; the disease in them is rather chronic.

Bruce, Hammerton, Bateman, and Mackie, found *T. vivax* to occur in Uganda, and discovered a bush-buck (*Tragelaphus scriptus*) naturally infected; they also found that *G. palpalis,* caught in Nature on the shore of Lake Victoria, were infected.

In Northern Rhodesia *T. vivax* was found in cattle, in some of which the unthrifty debilitated appearance of a "fly-struck" or trypanosome-affected animal was noticeable.

Transmission.—Cazalbou and Pecaud regard *Tabanus* as the natural carrier of the disease, which occurs in districts where tsetse-flies are not found. Bouffard has succeeded in transmitting infection by means of *Stomoxys* (interrupted feeding).

The Sleeping Sickness Commission (1909) in Uganda showed that *G. palpalis,* caught on Lake Victoria were infective. Bouffard, in Upper Senegal, at the same time, demonstrated the existence of a cyclical development in *G. palpalis,* the non-infective period in which is only about a week; the flies remained infected for at least two and a half months.

Roubard has shown that, whereas in *T. gambiense* and others the flagellates multiply in the gut of the *Glossina, T. cazalboui* multiplies only in the proboscis; and other writers have verified this finding for *T. vivax.*

In Northern Rhodesia *G. palpalis* did not occur in localities in which *T. vivax* was met; but *G. morsitans* was found there, and may be shown later to be a carrier, although some evidence indicated that *Stomoxys* should be incriminated.

In Italian Somaliland no tsetse are known to be present in the locality where the trypanosome was detected.

TRYPANOSOMA UNIFORME (Bruce, Hammerton, Bateman, and Mackie, 1910).

Historical.—This name was given by the Sleeping Sickness Commission of the Royal Society (1909) to a trypanosome found in cattle of Uganda which, like *T. vivax* and *T. nanum,* is non-pathogenic to laboratory animals.

Morphology.—*T. uniforme* measures from 14 μ to 18 μ in length, with an average of about 16 μ, and from 1·5 to 2·5 μ in breadth. The undulating membrane is poorly developed, and the free flagellum is from 2 μ to 5 μ long. The general shape is similar to that of *T. vivax,* from which it differs in being smaller, and is less active in fresh preparations.

Transmission.—There is no evidence in Uganda to show which fly acts as a transmitting agent.

TRYPANOSOMA THEILERI (Laveran, 1902).

Historical.—Theiler published his observations on this trypanosome in 1903, and associated it with gall-sickness in cattle, a disease common throughout South Africa, and one for which many causes have been assigned. In blood preparations sent by Theiler to Laveran, the latter found smaller trypanosomes, presenting different morphological features, to which he gave the name *T. transvaalense.* It has since been shown that these are immature forms of *T. theileri.*

T. theileri has been found in South Africa, Northern Rhodesia, Congo Free State, Uganda, German East Africa, West Africa, Uruguay, Transcaucasia, India, and morphologically similar forms have been detected in England (Stockman), Germany, and Russia.

Morphology.—*T. theileri,* on account of its great size, is easily distinguished from the other blood forms of mammals. It measures from 60 μ to 90 μ in length, of which the free flagellum is about 10 μ to 20 μ, and is from 4 μ to 6 μ in width. The undulating membrane is well developed.

The smaller forms (*T. transvaalense*) range from 25 μ to 40 μ, and in them it is common for the blepharoplast to lie close to the nucleus. This is a frequent finding in multiplication forms.

Animals Affected.—Dutton and Todd and Bruce and his colleagues found similar parasites in bush buck, but of the domestic animals, cattle alone appear susceptible. According to Theiler, the inoculated disease is one of anæmia, from which a large number recover. The distribution of the parasite in Nature leaves no room for doubt that its pathogenic powers are slight and very limited. It frequently occurs in the blood in the course of other diseases, and in many animals it does not appear to produce any clinical disturbance.

Transmission.—Theiler incriminated the genus *Hippobosca*, and produced experimental proof by interrupted feeding. The natural transmitting agent in Europe is not known.

Diagnosis.—*T. theileri* retains its vitality and motility for several days when removed from the body.

Knuth, Rauchbaar, and Morganstein, adopted a method for the detection of this trypanosome when present in such small numbers as to escape observation on direct examination.

They add a small quantity of sterile defibrinated blood from the susceptible animal to a tube of nutrient broth, and keep the tube at room temperature in the dark. After a few days numerous clumps of trypanosomes and young multiplication forms can be found on microscopical examination.

TRYPANOSOMA GAMBIENSE (Dutton, 1902).

Synonyms.—*T. ugandense* (Castellani).
T. castelleni (Kruse).

HUMAN TRYPANOSOMIASIS.—*Synonym:* Sleeping sickness in man.

Historical.—Dutton was the first to clearly recognise as a trypanosome a body seen in the blood of man in West Africa. A year later Castellani, a member of the Royal Society's Commission which was despatched to Uganda to investigate sleeping sickness, found trypanosomes present in many cases examined; it has since been shown to be the same as that found by Dutton, and to be the cause of the disease which has ravaged Uganda, parts of the Belgian Congo, and has now spread into British territory south of Lake Tanganyika.

Geographical Distribution.—*T. gambiense* is limited to Africa. On the west side of the continent it appears to be endemic, the natives not suffering to nearly the same extent as they did in Uganda, where it is supposed to have been introduced by Emin Pasha's followers. Between 10° north and 10° south it occurs sporadically, and often in epidemic form, on all the lakes and large rivers, and although introduced into distant countries, as Europe and the West Indies, it does not appear to have spread. The introduction of the disease into new areas has been largely due to the rapid opening up of this part of Africa.

Morphology.—*T. gambiense* measures between 17 μ and 30 μ in length—more commonly between 22 μ and 28 μ—and is from 1·5 μ to 2 μ wide. The undulating membrane is well developed, and a free flagellum is usually present, which measures about 6 μ to 10 μ of the total length. Ordinary microscopical examination could hardly differentiate this trypanosome from *T. evansi* or the large form of *T. dimorphon.*

Biology.—Man is the predominant naturally affected species. It is possible that dogs, cattle, and certain antelopes, may acquire the natural disease, and so act as reservoirs for the virus (Bruce). The duration of the infection in man is usually long—one or two years—but in some instances it assumes an acute type; secondary complications are frequent. The trypanosomes are always scanty in the blood, but in the early stages of the disease are more numerous in the juice of lymphatic glands and in the cerebro-spinal fluid obtained by gland or spinal puncture.

All the domestic and laboratory animals are susceptible to inoculation, but, with the exception of rats, mice, and most monkeys, the disease is rather chronic, and trypanosomes are scanty in the blood. Many artificially infected domestic animals show no outward symptoms, and do not succumb to the infection.

Transmission.—The Sleeping Sickness Commission in Uganda showed the epidemiological relationship existing between this trypanosomiasis and a tsetse-fly (*G. palpalis*), and in 1903 Bruce and Nabarro proved that a monkey upon which these tsetse-flies caught at Entebbe had been fed contracted the infection. During the following six years it was recognised that *G. palpalis* was the chief transmitting agent, though here and there opinions were expressed that other causes—*i.e.*, coitus and mosquitoes —might operate under certain circumstances. It was always felt that mere mechanical transmission would not account for the rapid spread of the disease, but until 1909 the actual proof of cyclical transmission was not obtained. In that year Klein, working in German East Africa, carried out experiments, first with tsetse (*G. palpalis*) caught in Nature, and afterwards with flies hatched from pupæ bred in the laboratory. He fed these flies first on animals and men infected with *T. gambiense*, and then daily on new, clean. susceptible animals. The animal of the eighteenth day and all succeeding ones became infected. Bruce and his colleagues in Uganda immediately confirmed this, and showed that infection could commence on the sixteenth day after the infecting meal and continue for at least seventy-five days, at which time the experiment terminated.

Taute* experimented with *G. morsitans*, and apparently obtained positive results. Klein also endeavoured to show whether *G. morsitans* can act as a cyclical host to this trypanosome, but his results have so far been negative.

In 1909 and 1910 several cases of human trypanosomiasis were detected in the Loangwa Valley of Northern Rhodesia, a district in which *G. palpalis* does not appear to exist, but where *G. morsitans* abounds at certain seasons of the year, and where *G. fusca* also occurs. This would indicate that other species of *Glossina* could convey human trypanosomiasis, but the question has arisen as to whether the trypanosome found there is not distinct from *T. gambiense*, and a new species, *T. rhodesiense*, has been tentatively created.

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* *Vide Sleeping Sickness Bulletin*, 1909, No. 7, p. 250.

TRYPANOSOMA RHODESIENSE (Stephens and Fantham, 1910).

Historical.—Cases of human trypanosomiasis were detected in 1909 in the Loangwa Valley of Northern Rhodesia, and Bevan and McGregor were struck by the great virulence of the parasite compared with that of *T. gambiense*, which, as already noted, produces in most animals a slow disease, with trypanosomes scanty in the blood.

Stephens and Fantham obtained the strain from a European (the same who furnished the strain to Bevan and McGregor) who returned from Northern Rhodesia to Liverpool, and the results of their observations led them to believe that the peculiarities of this trypanosome warrant the creation of a new species.

Morphology.—The peculiarity of the strain in question is that short and "stumpy" forms are encountered in which the blepharoplast occurs anterior to the nucleus. *T. rhodesiense* is more virulent to laboratory animals than is *T. gambiense*, and it is also resistant to atoxyl. It has been known for some time that frequent injections of atoxyl cause the trypanosome to acquire a tolerance for the drug and to resist its action. The strain in question would appear to have possessed this property without previous administration of the drug.

The trypanosome obtained from a second European who contracted the infection in the same locality does not show the same morphological or drug-resisting peculiarities, so that the specific nature of *T. rhodesiense* must remain an open question.

Transmission.—A Commission has recently gone to the Loangwa Valley to inquire into the exact mode of transmission; but it must be accepted that *G. palpalis* is not culpable. *G. morsitans* and *G. fusca* are at present incriminated, though C. S. Neave inclines to a belief that other ecto-parasites, such as bed bugs and house parasites of this nature, may be responsible.

I. MAL DE CADERAS.

By R. EUSTACE MONTGOMERY, M.R.C.V.S.

Synonym.—Cadeiras Disease.

Historical.—It is stated that this disease was first introduced to the mainland of America from the Island of Marajo, near the mouth of the River Amazon. It now occurs in parts of Brazil, Bolivia, and the Argentine, but is most serious in Paraguay. Of late years the mortality has been very severe, and in some districts equines have been practically wiped out.

Morphology.—In length the *Trypanosoma equinum* measures from 22 μ to 25 μ, and is about 1·5 μ wide, with a free flagellum of about 5 μ. The undulating membrane is well developed.

This trypanosome is peculiar amongst those described up to this time in that it possesses a uniquely small belpharoplast. Instead of arising at the well-defined and deeply staining coccus-like body, which is situated at the aflagellar extremity of the organism, the rim of the undulating membrane shows, in *T. equinum*, only a very slight thickening. Diagnosis of this trypanosome is therefore easy in a well-stained preparation.

Biology.—Equines are almost exclusively affected in Nature, but all domestic animals are susceptible to a greater or lesser degree on artificial inoculation. Natural cases of the disease have been detected in dogs. All the usual laboratory animals are capable of infection. The capybara (*Hydrochœrus capybara*), a common rodent in Paraguay, appears to suffer naturally, and possibly plays a rôle as reservoir, much as do camels and bovines to surra in India.

Transmission.—Sivori and Lecler regard *Tabanidæ* and *Stomoxys* as transmitting agents, and state that thay have succeeded in effecting transmission by their agency. Other writers, while admitting this possibility, point out that in stables where the sick and healthy animals have been herded together no case of infection among the latter has been noted, and this despite the prevalence of these pests. Epidemiological facts point to the importance of the capybara, a waterside rodent, which is often found dead in numbers prior to the onset of an epidemic of mal de caderas among the horses. The go-between from capybara to horse is not definitely known, but Elmassian and Lignières suggest mosquitoes and fleas.

We have no knowledge of any cyclical transmission such as obtains in *G. palpalis* and *T. gambiense.*

Clinical Study.—The onset of the disease in horses is usually insidious, the first indication being a weakness in the animal when being mounted. This is associated with a progressive anæmia, and slight temporary œdematous swelling may appear on the body. The temperature is irregular and paroxysmal, being elevated (103° to 105° F.) for two or three days, and succeeded by a normal or subnormal period.

As the disease progresses, the weakness in the hind-quarters becomes more pronounced; conjunctivitis and keratitis frequently appear, and there is often an eczematous eruption on the shoulders, neck, and loins.

Blood examination during the febrile paroxsym will usually show the trypanosomes, which are absent in the afebrile periods.

The duration of the disease in horses is from one to four months, and it always ends fatally. Mules and donkeys live longer, and show less pronounced symptoms, but the infection is always fatal.

Cattle, sheep, and goats will acquire infection, but rarely show any symptoms of the disease. Their blood is, however, infective to other animals, though trypanosomes may be too scanty for detection by ordinary microscopic examinations, and these animals may therefore constitute reservoirs of the virus.

In dogs the symptoms are similar to those of Equidæ: progressive wasting, febrile paroxysms, and conjunctivitis, frequently followed by keratitis and iritis. Trypanosomes are numerous during the periods of fever.

Pathological Changes.—The lesions present on autopsy vary in accordance with the duration of the infection. In acute cases enlargement of the spleen and hæmorrhagic œdema of the lymphatic glands will predominate; while in chronic instances lesions of anæmia, with paleness and anasarca of the muscles, fluid in the body cavities, petechiæ on the serous and conjunctival membranes, may be marked.

Treatment.—Medicinal treatment appears without avail. It was for the treatment of this trypanosome that Ehrlich and Shiga introduced the drug trypanroth, which is sometimes efficacious in laboratory animals.

Prophylaxis.—General principles (see p. 1048) only can be employed so long as the details of reservoir and transmitting agent are unknown. As it occurs mainly in marshy districts, these should be avoided where possible, and all affected animals slaughtered or safely isolated (fly-proof houses).

II. NAGANA.

By R. EUSTACE MONTGOMERY, M.R.C.V.S.

Synonym.—Tsetse-Fly Disease.

Historical.—In 1895 Bruce was sent to investigate the so-called tsetse-fly disease or nagana of Zululand, and by a series of experiments demonstrated—(1) a trypanosome as the cause, (2) that a species of tsetse-fly (*G. morsitans* and *G. pallipides*) acted as a transmitting agent, and (3) that certain species of big game acted as a reservoir for the virus.

The destructive influence of the tsetse upon domestic animals had long been known to the natives living near their haunts, and every book on sport and travel in South and Central Africa contains reference to the losses they engender in live-stock.

Bruce sent an infected dog to England, and the strain from this animal has passed into many laboratories, where the parasite was studied on exact lines. In Africa, however, until very recent years, it has been the custom to diagnose as nagana, and consequently as *T. brucei*, all cases in which the trypanosomes have been found. The result is that great confusion occurs, and that reports on the geographical distribution, clinical studies, and notes on possible transmitting agents, are often misleading, a totally different trypanosomiasis having in reality been studied.

More precise methods of study are now eliminating forms morphologically distinct, and even among those presenting similar microscopical appearances it has been possible to show that some—*i.e.*, *T. soudanense*—should be allied to *T. evansi* rather than *T. brucei.*

McFadyean has been able to separate *T. evansi* from *T. brucei* on morphological grounds, but the close similarity between the two renders a positive diagnosis very difficult, except, perhaps, when the two are available for comparison, and where numerous experimental animals have been observed.

Morphology.—*T. brucei* averages between 25 μ and 32 μ in length, of which about 6 μ represent the flagellum, and is about 1·5 μ to 2 μ in width. The blepharoplast, undulating membrane, and free flagellum, are prominent. Recently Bruce has published more exact measurements of the forms found in Zululand and Uganda, in which he gives the range in length as between 15 μ and 34 μ. He also shows that forms possessing but a very short flagellum may be present.

Animals Affected.—*T. brucei* is capable of being inoculated into all domestic and laboratory animals. In Nature wild ruminants appear to possess a great resistance to the disease, though experimentally certain species (zebra and some antelopes) have been shown susceptible. Bruce proved that in Zululand the blood of some wild ruminants, including the buffalo and also a hyena, contained the trypanosome, although in such small numbers that direct microscopical examination did not reveal them, and it was necessary to inoculate susceptible animals to prove their presence.

Among the domestic animals, dogs, horses, mules, and donkeys, are the most susceptible. Cattle are more resistant, and in sheep and goats the disease usually runs a chronic course. Of laboratory animals, the rabbit is the most resistant to this as to the majority of other trypanosomes.

Clinical Study.—The incubative period elapsing between the bite of a fly (or inoculation) and the appearance of trypanosomes is about seven days in domestic animals. Two or three weeks later visible symptoms of disease are noticeable in horses ; in dogs the period is rather less.

A staring coat, slight lachrymation, and some emaciation, are the first noticeable symptoms ; later the emaciation becomes more pronounced, fluctuating œdematous swellings appear on the legs and genitalia, and slight opacity of the cornea is often present. At the time of death the animal is in an extremely weak condition, with pallid membranes and a hide-bound coat, from which the hair may have come out in patches.

The appetite remains good, and even voracious, to the end. The temperature is commonly elevated at the first appearance of trypanosomes, and it continues elevated (102° to 104° F.) or shows paroxysmal remissions, during which time the parasites are found to be more numerous in the blood.

In cattle the course of the disease is more irregular, some animals dying within a few weeks, others living on for six months or more. French cows, inoculated at Paris by Nocard, showed only a rise in temperature about five days after inoculation. Later no trypanosomes could be seen in the blood, although it remained infective on subinoculation into susceptible animals for several months. These cows all recovered. The symptoms in susceptible cattle are in the main similar to those in equines and dogs—*i.e.*, emaciation, anæmia, changes in the eyes, and œdema of the dewlap. The temperature does not show so great a variation as in equines, and trypanosomes are less numerous.

In sheep and goats the disease is usually chronic, and the symptoms are those of anæmia and cachexia. Trypanosomes are very scanty in the blood.

Pathological Changes.—Beyond evidence of anæmia more or less profound, and sometimes enlargement of the spleen and lymphatic glands, no constant lesions are present on autopsy.

Transmission.—In 1895 Bruce showed that horses taken into "fly" country, if only for a few hours, contracted the disease, although they were not permitted to eat or drink when there. He also proved that tsetse (probably *G. pallipides,* although throughout the report *G. morsitans* is mentioned), caught in Nature and fed upon animals living away from infection, caused the disease to be produced. Bruce further showed that wild flies fed upon an infected animal transmitted the trypanosome to a healthy one forty-eight hours after the meal, and he found living parasites in the proboscis up to forty-six hours after feeding. This evidence did not disprove mere mechanical transmission from the sick animals or from big game acting as reservoirs, and it was not until 1908 that positive proof was obtained showing that a cyclical infection of the fly occurred, and it is this latter mode of infection which undoubtedly obtains in the majority of instances in Nature.

Klein, working in German East Africa, obtained a strain of trypanosome resembling *T. brucei,* and fed *G. palpalis* upon an infected animal for three days. These flies were fed on each subsequent day for seventeen days upon a new susceptible animal. From the eighteenth to twenty-fourth days they fed daily on one animal, and from the twenty-fifth to thirty-ninth on another. Both these latter became infected, while the animals used from the fourth to the seventeenth days remained healthy. Between the fortieth and fiftieth day the same flies fed on two goats, two calves, and two sheep, all of which became infected.

Klein commenced the experiments with fifty flies; on the sixty-sixth day after the infecting meal twenty remained. In order to ascertain how many of the flies were carrying infection, each of these was placed on a new susceptible animal, and it was shown that only two were still infective.

Minchin, Grey, and Tulloch, and recently Schuberg and Kuhn, have shown that *Stomoxys* can transmit *T. brucei* by interrupted feeding (*i.e.*, the removal of the fly during the process of feeding and the placing on a new animal), and it is conceivable that any other biting fly which will feed in captivity might do the same.

It must be concluded that the cyclical method of transmission is more in keeping with the life-history of a member of the protozoa, which, as in malaria and redwater, require the active agency of an ecto-parasite. The knowledge that it has been shown to occur also in other trypanosomes—*e.g.*, *T. gambiense* and *T. cazalboui*—indicates that it is the natural method, and that mechanical transmission must be largely accidental.

Flies other than *Glossinæ* are more difficult to rear and to feed in a laboratory (*Tabanidæ* are particularly so); and it may be for this reason that cyclical transmission of such species as *T. evansi*, which are certainly not spread by tsetse, has not yet been established. In the case of those for which a cyclical transmission has been made out in *Glossinæ*, as, for example, *T. brucei*, it has still to be shown that other biting flies are incapable of acting as hosts in the same way.

Whatever be the form of transmission, it demands the existence of a host or a reservoir from which the trypanosome can be taken. In many parts of Africa domestic animals are wanting, yet the tsetse of the locality is capable of infecting them when introduced. In some cases the domestic animals themselves may act as reservoirs, but in localities such as here indicated other animals must be incriminated, and Bruce showed that buffalo, wildebeeste, koodoo, bush buck, and hyena, could harbour the trypanosomes in their blood, and these, together with other species of game must be held responsible.

It is a much discussed question whether the tsetse, and especially *G. morsitans* and *G. pallipides*, can exist in the absence of game, a popular contention being that the destruction of this latter would kill off or drive away the fly. That such a procedure would remove the principal reservoir is undoubted, but its impracticability, except in localised areas, is also obvious. It may also be added that avian and reptilian blood affords nourishment for these flies, although it is not believed that they could acquire infection from this diet.

Treatment.—Livingstone recommended arsenic as an agent beneficial in tsetse disease, and Bruce undertook experiments to ascertain its value. The results were doubtful, but for the most part the drug as then administered was useless. The method advocated by Holmes for the treatment of *T. evansi* is worthy of trial (see p. 1079).

III. DOURINE.

By Major F. S. H. BALDREY, F.R.C.V.S., D.V.H., I.C.V.D.

Nomenclature (Synonyms).—A multitude of terms have been used to designate this disease for an indefinite period. Its actual cause not being suspected until comparatively recently, the names given were those that appeared most descriptive to various observers. They are—(1) Common venereal diseases of the horse; (2) Horse syphilis; (3) Maladie or mal du coït (French, and for some time the cognomen in England); (4) the French also used the term Exanthème coital; (5) Genital glanders; (6) Breeding paralysis; (7) Covering disease; (8) Epizoötic paraplegia (a most misleading title); (9) Schleichende Nervenkrankheit (German), (on account of the nervous complications, this name was given in 1833); (10) Hertwig gives the names Beshalkrankheit der Pferdo for the first stage, and Blashenausschlag for the second stage; (11) Dourine. The last term is the one now universally accepted. The name is not descriptive, as are some of the older ones, but it is distinctive and short. Pease[1] thinks the word is derived from the Arabic, and means "dirt"; but it may be from the running or discharge being called a "dour," which would be a play on the Hindustani word, which means "to run." Or it may refer to the gelatinous or syrupy exudate noticeable at the post-mortem, which resembles the syrup from the *Anthropogon sorghum*, commonly called "dour." However the name originated, it is the best yet suggested.

Definition.—Dourine is a progressive wasting disease complicated with nervous degeneration, manifested primarily in the genital organs, and due to direct infection by a trypanosome.

GENERAL REMARKS.

Short History and Geographical Distribution.

The disease appears to have been known in Algeria since early history, but was only first recorded in Europe in 1816, and then only in Southern Europe and Southern Russia. It is recognised in Northern Africa, including Egypt, in the whole of Spain, and Southern France, Austria, Hungary, and Italy. It is at present the only known pathogenic trypanosome indigenous to Europe. The early home of the disease would appear to be Syria and Asia Minor. Persia is affected, and probably parts of Arabia. It was first recorded in the United States of America, in the De Wet county of Illinois, in 1889, and was said to be imported from France. Nocard,[2] however, doubts this; but Law[3] traces the disease to an imported French stallion. From the States it was conveyed into Canada, where stringent measures by the Veterinary Department have stamped it out.[4] Chili has been credited with a similar disease, but this has not been authenticated. Ammon mentions the disease as occurring in Prussia in 1796, and Hertwig records instances in 1807. Lautour records it in France in 1830. Friedberger and Fröhner[5] mention it as occurring in 1840, at which time special laws were made for its control in Silesia and Poland.

For some time the disease was considered identical with human syphilis, until Hausmann and Knauert demonstrated the fallacy of this in 1837.

A French army veterinary surgeon named Signol described the disease in 1847 as occurring amongst Arabs in Algeria, and causing great loss. The Arabs themselves said the disease was old-established, but only recurred at long intervals.

St. Cyr[6] and Trasbot[7] reported at some length on the disease as existent in Algeria; their experiments were made at Alfort and Lyons. Nocard[8] demonstrated its inoculability to the dog in 1892, but it was not until 1896 that Rouget[9] first demonstrated the presence of a trypanosome in the lesions, and advocated this organism as a cause. Schneider and Buffard[10] confirmed this view in 1899.

The disease has no doubt been in existence in India for many years, but it was only in 1903 that Pease[1] demonstrated the actual cause. An old Punjab veterinary annual report mentions a case of maladie du coït, and I have myself seen cases in the Bombay Presidency, in Baluchistan, and in the United Provinces horse-breeding establishments as early as 1899, which, in the knowledge of subsequent events, were undoubtedly dourine. These cases were treated under various names, such as asthenia, sprained penis, ulceration, cachexia, or paralysis.

SUSCEPTIBILITY.

Natural infection appears to be entirely confined to equines. The disease is most common amongst horses, but the ass acquires it in a less virulent form. Dogs are susceptible, especially young animals; but Pease[1, 4, 5] has noted that the Indian pariah is practically immune, except when very young, and this fact has established an easy method of differential diagnosis between dourine and surra, as the pariah is easily susceptible to the latter.

Cattle are immune to the infection, and even after the inoculation of a very large number of organisms it is difficult to demonstrate their presence in the blood for more than a few hours. In this, again, it differs from surra, as cattle may carry the parasite, and the blood may be infective from this latter for an almost indefinite period. Rabbits, rats, mice, and European dogs, are easily susceptible to experimental inoculation, and may subsequently carry on the disease by coitus.

The passage of the organism through dogs and rabbits does not affect its power of infectivity. Its virulence appears to be in no way altered to equines by such passage. Infection may occur naturally from other means than coitus, as, for instance, by the medium of flies, but this is an almost negligible possibility. Cats, frogs, and birds appear to be refractory.

IMMUNITY.

No actual immunity can be said to exist naturally, except in a modified form. It has been noted that mares carry the infection for a very long time without showing any marked symptom. There may be nothing but a very slight leucorrhœa, but it is important to notice that sterility is concomitant with this; and the same may be said of stallions, who show a scarcely perceptible discharge. This condition can hardly be called an immunity: it is a chronic form. Such cases in course of time develop acute symptoms or spontaneously recover. It is doubtful if such recoveries ever regain virility. Nocard[11] mentions that several dogs recovered, and subsequently failed to react to large doses of infective blood. Pease[1] obtained the same results in the buffalo, and Mésnil and Rouget[12] immunised a kid. No experiments with serum inoculation appear to have been successful in producing immunity.

Very early castration will prevent the disease if the operation is performed while the affection is entirely local. I cannot explain this, unless the organism does not extend further than the testicle in the initial stage, but I have successfully operated on several occasions.

Immunity in mares must essentially be comparative. An infected stallion may cover twenty mares, and only infect two of them. This does not point to immunity in the mare, but to an absence or very few organisms in the stallion at the time of covering.

PERIOD OF INCUBATION.

From artificial infection this may be from seven to ten days. By coitus it is much more uncertain, but is probably about fifteen to twenty days. Lingard[13] suggests that a careful watch should be kept in suspected cases up to the fiftieth day, but such a time is exceptional. It is sometimes difficult to note the primary clinical symptoms in the mare, even more so than in the stallion, especially in the initial stage, and it is for this reason that the incubation has sometimes been thought to be so long.

ETIOLOGY.

The cause is the *Trypanosoma equiperdum*. Trypanosomes as a general rule are blood parasites, but the *T. equiperdum* is not only a blood parasite, but causes local lesions, in which it remains for a more or less prolonged period. Pease[1] has found that the parasite may remain in the vaginal mucous membrane for more than a year. It differs from other trypanosomes in that contagion is conveyed by direct inoculation from the diseased to the healthy animal during coitus, so that there is no necessity for an intermediate carrier—in fact, as far as is at present known, no species of biting insect is concerned in its transmission. Artificial inoculation is easily accomplished by the injection of infected blood, or by smearing infected material upon a mucous surface or even upon excoriated skin. We must, therefore, not eliminate entirely the possibility of infection through the medium of biting insects or infected material. The organism is not resistant to exposure to air and sunlight, so that the possibility of infection by contaminated bedding, etc., is not great. The parasite is found in the seminal and vaginal fluids of infected animals, also in the testicles, preputial glands, sheath, and in external ulcerations of the genital organs. It is as a rule difficult to find in the peripheral circulation, except in the localised eruptions of the second stage in very acute cases, and is even then seldom numerous. It is interesting to note the length of time which the organism is capable of living in the vaginal mucus, occasionally not showing any symptoms. Animals so affected are particularly dangerous, as contagion is easy and efficient diagnosis difficult.

BACTERIOLOGY.

The organism is 18 to 26 μ in length, and 2 to 2·5 μ in breadth, and is therefore somewhat smaller than the trypanosome of surra, which it otherwise nearly resembles. It is much smaller than *T. Lewisi*, and has not the chromatin granulations of that parasite. The macronucleus is situated in the centre of the body, and is very distinct, and even by the Giemsa method of staining a distinct centresome (or nucleolus, as it used to be called) is plainly discernible. The blepharoplast (or centrosome, to use the old expression) is situated at the posterior extremity, and its centrosome can only be demonstrated by the method of Heidenhein. From the blepharoplast arises a vigorous undulating membrane, which is terminated at the anterior extremity of the body by a flagellum. The length of this flagellum is from 12 to 18 per cent. of the length of the parasite by the method described by Lingard.[13] The organism is not so active as that of surra, and is never numerous. The protoplasm is finely granular, and that of the nucleus very loosely granular. The posterior extremity is very distinctly beak-shaped, but is not so sharp as that of the organism of surra. Fresh specimens will remain alive and active for some days on a moist slide at room temperatures, and for seven days on ice.[15]

Multiplication takes place by longitudinal division only, commencing at the blepharoplast, which approaches the nucleus during this stage. Division then rapidly develops by mytosis of the nucleus, the formation of another undulating membrane, and a flagellum. Rosette forms have been described by Buffard, but this was probably agglutination. Such phenomena are practically impossible in natural cases on account of the fewness of the organisms, but they can be observed in experimental laboratory animals.

Thomas and Breinl have had slight success in the cultivation of this organism in a media which was a modification of Novy and McNeil's. They found that the culture was still pathogenic after seventeen days.

In taking smears for examination from blood, care should be taken to accelerate drying as much as possible, as the organism appears to be particularly sensible to coagulation, so that if the blood at all coagulates, the demonstration of the organism is absolutely impossible. It stains well by Giemsa or Leishman, the latter being very easy to use. Where fixation is necessary, methyl alcohol is preferable. Any excess of methylene blue by the Giemsa method may be removed by passing quickly through methylated spirit, and then rapidly washing in water. To demonstrate clearly the nuclear outline, with its centrosome and chromatin network, as well as the differentiation of the blepharoplast, it is necessary to use the somewhat tedious method of Heidenhein, or Rosenbusch's modification of Moore or Heidenhein's methods. This is as follows:

1. Make the smears rapidly, either from blood or mucous membrane, and fix

while still wet in a hot saturated solution of mercuric chloride in alcohol or water, which contains 2 per cent. of acetic acid.

2. Transfer at once to a cold saturated solution of mercuric chloride for a few minutes only.
3. Wash thoroughly in running water.
4. Place in a 3·5 per cent. solution of iron alum for three-quarters of an hour, or longer.
5. Wash thoroughly in running water.
6. Place in a 1 per cent. hæmatoxylin solution in 96 per cent. alcohol for half an hour. The above solution should be kept for some weeks before use, to mature, in a brown bottle with a cotton-wool plug, and enough lithium carbonate (saturated solution) is added at the time of staining to make the stain a mahogany-brown.
7. Wash thoroughly in running water.
8. Pass rapidly through a 3 or 3·5 per cent. iron alum solution, and again wash.

To obtain a proper degree of differentiation, a very weak solution of iron alum (1 c.c. of a 5 per cent. solution in 10 c.c. of water) should be placed on the smear, and this covered with a cover-glass, and the specimen observed under the microscope. When the nuclei of the leucocytes are clearly defined and the surrounding protoplasm shows as a clear zone, differentiation is complete.

9. Wash thoroughly in running water for ten minutes.
10. Pass through alcohol and zylol, and mount in Canada balsam.

Great care must be taken that the film is never allowed to dry. There is no evidence of conjugation or of any other means of propagation except by simple division.

On account of the difficulty of cultivation, it is impossible to demonstrate an extracellular toxin, but, taking into consideration the small number of parasites in a naturally infected case, and the active changes which take place in the blood, it may be presumed that a considerable amount of toxin is produced.

MORBID ANATOMY.

The early stages of the disease reveal but little change beyond the local ulcerations and œdema, together with progressive atrophy of the testicle. Nervous irritation is evidenced by localised erythema, and subsequently by the presence of "plaques." The pathology of these plaques is somewhat obscure. Lingard[13] thinks that the plaque is a circumscribed œdema of the skin, due to a paralytic dilatation of the arterioles, followed by an exudation of serum and migration of leucocytes, the variations being due to the different depths to which the infiltration penetrates, and that the lesion is entirely dependent upon nerve disorder. Mott[16] does not think that the plaques are due to embolism by trypanosomes, but believes that the inflammatory irritation of the posterior spinal ganglia, which become affected by the causal agent, gives rise to the cutaneous plaques. Bayliss has shown that stimulation of the posterior roots produces vaso-dilatation. The trypanosomes find these plaques a suitable media for multiplication. The changes in the spinal cord are most marked in the lumbar and sacral regions, and there is marked degeneration of the posterior fibres of the cord, together with inflammation of the posterior spinal ganglia. The blood shows progressive anæmia, and Pease[1] has noted leucocytosis. The process of anæmia is apparently the result of destruction of the corpuscles by the enormous amount of oxygen used up by the trypanosome, and possibly by the very poisonous character of the toxin which it evolves.

The autopsy reveals a round-celled infiltration of the sweat glands, and a very marked yellow gelatinous exudation beneath the skin, particularly of the abdominal region. The system is denuded of fat, and the bone-marrow is reddish-brown in colour. There are the usual evidences of chronic disease, associated with enlarged spleen and kidney, and very often eroded surfaces of the joints. The characteristic post-mortem lesions are enlarged and softened lymphatic glands, which contain quantities of a yellowish blood-stained fluid, and the glands are spotted with petechiæ. In the male the testicles are very small, enclosed in a dense mass of fibrous tissue, and may even be replaced by it. The mucous membranes show hæmorrhagic patches. The muscular tissue, especially of the heart, shows fatty degeneration. The coverings of the brain are usually dropsical, and the fluid of the spinal cord is also increased. The spinal cord is degenerated in the posterior column, which is particularly noticeable in the cervical and lumbar regions. This degeneration is confined to the fibres of the posterior column and the sensory roots of the spinal nerves, the grey and the white matter remaining healthy. Owing to these changes, Marek[17] has termed the disease "polyneuritis infectiosa equorum." The cord, particularly in the lumbar region, is of a reddish colour, and considerably softened. These changes have not been observed in the brain substance.

The changes in the lymphatic glands are progressive, commencing in the posterior parts of the body, and in acute cases rapidly extending to the anterior.

SYMPTOMS.

The disease generally justifies its old name of syphilis, as the symptoms bear a very close resemblance to that disease in man; and since the discovery of the *Spirochæta pallida* the analogy is more pronounced. The symptoms may be divided into three very distinct stages, termed *primary*, *secondary*, and *tertiary*.

Primary.—As a result of infection, either by coitus or artificial inoculation, the first symptom noticeable in the female is a discharge from the vulva, and in the male from the urethra. The mucous membrane becomes inflamed, so that there may be some degree of eversion in either sex. Often these symptoms are so slight as to escape notice, unless the observer has the knowledge that the disease is already locally existent. This suppression of palpable symptoms in the early stages is especially dangerous, on account of the risk of further infection, as even at this stage the propagation of infection is easily accomplished. A stallion will show frequent erections, and the mare will exhibit irritation by simulating œstrum. It is more than probable that even at this stage both the mare and the stallion are sterile. Immediately following this, œdema of the penis and sheath will be observed, and probably phimosis and paraphimosis, together with swelling of the scrotum, extending forwards nearly to the sternum. These swellings are seldom inflammatory, and tend to persist, but may be metastatic. The testicles are tender, and the legs, particularly the hind ones, are swollen. Ulcerations and patches of erosions then appear upon the genital organs. At this time there are indications of urticaria and irritability of the skin. The mare will show similar symptoms, with probably swelling in the perineum. There may be a weakness of the loins, with a tendency to arch the back; but there are no symptoms of constitutional disturbance, the temperature being only very slightly raised, and the appetite remains good. As the disease progresses, pain may be evinced in micturition and in covering. If the pruritus has been acute, there may be some denuded patches of skin, particularly on the quarters and thighs.

Secondary.—Some four to six weeks after the initial onset the general condition will become more acute. Emaciation is evident, and the animal becomes listless and tires easily. The loins exhibit pain on pressure, and the swellings are more noticeable and exhibit some hardness on pressure, as a result of chronic infiltration. During this period the pathognomonic symptoms known as "plaques" appear. They first make their appearance about the fortieth day, and are characteristic. They are cutaneous eruptions, which occur irregularly on different parts of the body. They may only last for an hour or so, and seldom longer than a couple of days. They are rounded or oval in shape, varying in size from 1 inch to 4 inches in diameter, and Pease[1] describes them as showing an appearance as if a disc of metal had been slipped under the skin. They are sometimes so slight as to be only seen when looking at them sideways at an angle from the direct light. They are not at all hot or painful, but may exude a slight serosity. Their disappearance is usually spontaneous, no sign being left of their presence. They rarely become œdematous or persist. The eruption of plaques is sometimes preceded by an eruption of papules resembling a nettle-rash, which very quickly disappears. A drop of blood taken from a plaque and examined in a fresh state will almost invariably yield the trypanosome, but never in large numbers, and the presence of even one is sufficient for a positive diagnosis.

Progressive anæmia and generalised granular swellings are now evident. The glands are soft, but as the swelling is persistent they tend to harden; they are not painful, and rarely suppurate. Lassitude is more marked, and there is difficulty in rising. It is difficult to induce a trot, and in the stallion erection is feeble and incomplete. The discharge in mares becomes viscid and irritating, and excoriates the skin of the thighs. It is rare for such mares to conceive, but if they do abortion will follow.[1] The appearance of plaques in the mare, as well as the general symptoms, are the same as in the stallion.

Tertiary.—From about the tenth month of the disease all symptoms become more acute. Progressive anæmia is very marked, the appetite is variable, and the temperature may be slightly raised. The urine is high coloured, and defæcation irregular. The animal wastes and becomes very weak. Co-ordinated locomotion is impossible, and the horse sways while standing still. Partial paraplegia is evident, and may become acute. Indolent abscesses and ulcerations occur on the sheath and scrotum, and on other parts of the body. During this period death may occur at any time, but occasionally the animal lives long enough to suffer from complete paralysis of the hind-quarters.

COURSE.

It will be seen from the above symptoms that the progress of the disease is more or less chronic, and its actual duration is from ten to eighteen months.[18]

In the donkey the symptoms are very similar, but even of a more chronic nature, and it is said that complete recovery may occur in this animal.[2]

DIAGNOSIS.

On account of the insidious nature of the disease, diagnosis in the early stages is extremely difficult. An early recognition is of great importance, in order that active measures may be taken to prevent any further spread of the disease.

It has been shown that the parasite may live in the vaginal mucous membrane for a very long time without causing any marked symptoms. This is often the cause of unknowingly infecting a stallion, who in turn infects a large number of mares. A positive diagnosis can only be made by demonstrating the trypanosome. In any portion of country where there is the least indication of the presence of the disease, such as a discharge, or ulceration, or other suspicious symptom associated with the genital organs of either the male or female, some of the fresh material should immediately be submitted to a careful bacteriological examination. Failure to find the trypanosome in one examination will not constitute a negative opinion, and frequent examinations, strengthened by centrifugalisation of the suspected material, should be made. As a rule, however, a searching microscopical examination will show the trypanosomes. If any suspicion remains in a negative case, freedom from the disease cannot be certified until the period of plaques is past. In the event of these appearing, a positive result will no longer be difficult.

The parasites are always present, and generally in some numbers, at the centre of the œdematous swelling. The examination should be made under a cover-glass with fresh material taken from a recent eruption. The older the œdema, the less chance there is of finding the parasite. There are no marked constitutional disturbances in the early stages, and the temperature gives no aid to diagnosis. Recurrent crops of parasites, although causing the characteristic local lesions, have very little effect upon temperature. This is in marked difference to malaria and piroplasma. Uhlenhuth and Woïthe[19] remark upon the likeness of the disease in this respect to sleeping sickness in man. They found in experimental cases that, whenever any doubt existed as to the presence of trypanosomes, it was advisable to make test inoculations in mice. If six to eight mice were inoculated, about five would give a positive result, even when the blood used was from an animal in which no trypanosomes could be detected microscopically. Rabbits are also useful as a means of diagnosis. Pease[1] has found that test inoculations in the ear almost invariably give positive results, and cause a characteristic durable œdema of that organ.

The necessity for an early diagnosis has always been urgently recognised, and has led to some proposals for its accomplishment. Lingard suggested that sustained exertion, by inducing fatigue, and so lowering the animal's resistance, would allow of an increase in the number of parasites, and render the microscopical demonstration easier.

The inoculation of some harmless organism has also been suggested with the same object. A more scientific method is that of adapting the means employed by Wassermann in the diagnosis of syphilis.[27] This method is a modification of the Bordet-Gengou reaction, and is based on the fact that a specific antigene combines with its amboceptor in the presence of an indifferent complement. If union has taken place with the correct proportions of fluid, the complement will be all used up, and this latter can be demonstrated by the addition to the above substances of a specified quantity of hæmolysin and red corpuscles of the animal upon which the hæmolysin acts. If no hæmolysis results, it is evident that the complement has been utilised, so that the hæmolysin is unable to act specifically upon the red corpuscles. From this we would learn that the antigene has found a specific amboceptor, and the result would be a positive diagnosis.

The method of applying this phenomenon in trypanosomiasis has been demonstrated by Mantenfel and Woïthe.[28] The antigene is obtained in an extract of liver from an animal affected with dourine. This extract, in specified quantities, is mixed with a certain amount of serum from an animal suspected of the disease (the amount of extract to be used is ascertained by preliminary experiment). An indifferent complement is then added to the mixture, and it is kept at 37° C. for one hour. In order to demonstrate the union of antigene and the amboceptor in the suspected serum, rabbits' washed red corpuscles are added, together with a sufficiency of hæmolysin (rabbit)—*i.e.*, the exact amount of hæmolysin required to hæmolyse a given quantity of corpuscles is ascertained by preliminary experiment. (The object of using hæmolytic substances is to render demonstration easy to the naked eye.) The mixture is again placed in the incubator at 37° C., and changes carefully noted at frequent intervals. If the antigene and amboceptor have not combined, the complement will still be free, and will permit of the hæmolytic action. Such hæmolysis would demonstrate a negative diagnosis, showing that there was no specific amboceptor to combine with a specific antigene. On the other hand, if the complement has become fixed, owing to the union of antigene and amboceptor, there will be no hæmolysis, and this would constitute a positive diagnosis. The technique is somewhat difficult, but it is a practical demonstration of Ehrlich's theory. It is essential that a thorough comprehension of the significance of antigene, amboceptor, and complement be acquired.

Although this reaction has been undoubtedly accurate in diagnosing syphilis, it would seem that the antigene is not always specific in protozoan diseases. It has been shown that an antigene from one specified trypanosomiasis will combine with several other protozoan amboceptors, so that if the animal tested gives a positive reaction it would only prove that it was suffering from some form of trypanosomiasis or piroplasmosis. This difficulty may be got over in course of time. It explains the fact that atoxyl is equally good for all kinds of trypanosomiases because of their close relationship. In the case of malaria there is only one specific treatment—namely, quinine—and mercury has been found specific for the *Spirochæta pallida.*

The above method of diagnosis can, however, be made useful by a means of elimination. The horse suffers from surra, nagana, and mal de caderas, which can all be diagnosed. *Trypanosoma dimorphon* and the dwarf form described by Balfour of Khartoum appear to be confined to their own particular areas in Africa, and can by their clinical history be so eliminated. Piroplasmosis, even when it does not show the characteristic symptoms of hæmaturia and bilious fever, can also be determined by a few carefully stained specimens of blood. Malaria, as described by Theiler, may be considered in the same way. If all these protozoans be eliminated, then only dourine will remain.

The symptoms and course of the disease as a result of experimental inoculation are the same as those produced by natural infection. A direct intravenous inoculation will, however, cause a much more acute attack; and Uhlenhuth[19] found the duration of the disease to be only five months after such inoculation.

DIFFERENTIAL DIAGNOSIS.

The disease simulates glanders, but is at once differentiated by the mallein test. Surra differs in the temperature-chart. There are very high paroxysms of fever in surra, followed by corresponding low curves. The *T. Evansi* swarms during one of these paroxysms, and this condition never occurs in dourine. The same remark applies to nagana. The disease known as mal de caderas, prevalent in South America, has similar clinical symptoms, but the organism is entirely different when stained by Giemsa, Leishman, etc. It has been noticed that no centrosome (micronucleus) is demonstrable by these stains, although it can be shown by the Heidenhein process. In all other trypanosomiases of horses there are periodic accessions of fever and petechiæ on the visible mucous membranes, neither of which are associated with dourine. Moreover, the local lesions associated with the trypanosome are specific.

The history of the case will effectually differentiate it from purpura hæmorrhagica.

Pease[20] has drawn attention to the fact that two varieties of filaria are capable of producing plaques which simulate those of dourine. This may cause some confusion, but the absence of other local lesions and of the trypanosome, together with the presence of the filaria, is sufficient. Frequent examinations of blood are necessary to demonstrate the filaria, on account of the extreme periodicity of this parasite.

The same author has also pointed out[21] the insusceptibility of old dogs in India to dourine, although they are easily infected with surra. Morax[22] has noted the peculiar greyish opacity of the cornea which is distinctive in dogs experimentally inoculated with dourine. This is an interstitial keratitis, which is persistent. There is an invasion by mononuclear leucocytes and by trypanosomes, although the condition is not hæmorrhagic. Pease has also noted this in experimental rabbits.

Pease and Baldrey[23] have pointed out the complication caused by horsepox. The symptoms of horsepox are often evinced by an exanthematous eruption on the external genitals of the horse. This may sometimes resemble the papular eruption seen in dourine. It is not likely to be mistaken for dourine, as its course is rapid and acute, and goes through the ordinary variolous manifestations, and it is seldom that the eruption becomes confluent or causes ulceration. This latter never happens if the case is treated.

Diagnosis in the ass is in all cases more difficult, on account of the more chronic nature of the disease. It often is so little accompanied by symptoms in the stallion donkey that the first intimation of the disease is learned from the mares that he has covered. Steps for positive diagnosis should be made exactly as stated above.

PROGNOSIS.

It may safely be said that the result will be fatal if the affection is once established, and an animal so affected can only be looked upon as a dangerous focus from which disease may be spread. Statistics in Germany and Russia have shown a death-rate of only 50 per cent., but it has been noticed in India that of mares exposed to infection more than one-third contract the disease, and 80 per cent. die.

PROPHYLAXIS.

The rules laid down for prevention are as follows :

1. In any district in which the disease is prevalent every mare that is to be put to the horse is to be carefully examined. Any discharge from the vulva or any swellings other than those which ordinarily accompany œstrum should preclude the mare from being covered.
2. The stallion is to be carefully and frequently examined, and never used if there is the slightest suspicious symptom on the genital organs.
3. Breeders to be given every information concerning the disease.
4. Compulsory report of all suspicious cases.
5. All sales in infected areas are stopped during the prevalence of the disease.
6. Where the malady is widespread the service of stallions is prohibited.
7. Segregation of affected animals. Those deemed curable to be treated, and incurable to be destroyed.

8. Stallions attacked to be castrated, and also any which have transmitted the disease, although they may show no symptoms.

9. Mares that are apparently cured not to be covered for one year, and then they must have a veterinary surgeon's certificate of health. It is better that such mares should not be used again for breeding.

TREATMENT.

I have already said that treatment is unsatisfactory. In the very early stages castration appears to benefit the stallion. Uhlenhuth and Woïthe[19] have made a special investigation in treatment, and have found that, the horse being particularly intolerant to atoxyl, satisfactory results were not attained with this drug; and in any case, unless treatment can be commenced simultaneously with the beginning of infection, no good result can be obtained. Pease[24] found the injection of cacodylate of soda of no use. Breinl and Nierenstein[25] have had results which promise some success, by using alternately atoxyl and perchloride of mercury injections similarly to the way in which Lingard treated surra in 1894,[26] the suggestion being that the atoxyl causes the trypanosomes to become encysted as a protective measure, most commonly in the spleen; and while they are in this state the mercury attacks them, causing their death.

In animals, such as rats and mice, used experimentally in the treatment by atoxyl (an arsenical compound) better results have been obtained, and even cures, but such animals are very tolerant to the toxic effects of arsenic and its combinations. In order to obtain such results the treatment must be continuous and pushed to the extreme. Unfortunately, the equine cannot withstand this without succumbing to grave toxæmia from the arsenic. Such treatment is still more or less experimental.

No serum-therapy has been accompanied with any successful practical results.

The medicinal treatment for dourine has not been very successful, and this is not difficult to understand if the habit of the organism is considered. It remains chronically persistent in the mucous and deeper mucous layers of the vagina of the female and in the genital organs of the male. Drugs, such as arsenic, which may have a beneficial effect by acting on the parasites in the blood-stream will not have the same effect upon parasites protected in such mucous membranes, so that, though the blood may be cleared, there still remains the possibility of auto-infection from the local areas, although the animal may have improved under the drug treatment, as Uhlenhuth and Woïthe[19] found. Fröhner[29] has tried arsenate of potash, strychnine, tartar emetic, and atoxyl, with no lasting results.

The treatment adopted for surra by Holmes[30] might be tried. This is only a modification of the many systems of arsenical treatment adopted in trypanosomiases, and would probably be successful except for the habits of this organism mentioned above. This system, which is to push arsenic to subtoxic limits, at intervals, has been very successful in surra, and is applied in any of the three following ways:

System A.—(1) Atoxyl, 4 per cent. solution, 50 to 70 c.c. subcutaneously, first day; (2) arsenical solution (1 in 1,000), 600 c.c. *per os*, second day; (3) arsenic in ball, 1 gramme *per os*, third day. Four days' interval. Repeat, but slightly increase (2) and (3). Four days' interval. Repeat again, increasing (2) and (3). Eight days' interval. Repeat, increasing (2) to 1,000 or 1,200 c.c., and (3) to 3 or 3·5 grammes.

System B.—Atoxyl, 4 per cent. solution, 50 to 70 c.c.; arsenious oxide, 1 gramme. The atoxyl and arsenic are alternated on every second day. The former is not increased, but the latter is pushed to a subtoxic dose. This in small ponies would be about 2 grammes, and in big horses 3 to 3·5 grammes.

System C.—Atoxyl, 4 per cent. solution, 50 to 70 c.c., followed every second day by gradually increasing doses of arsenious oxide to subtoxic doses.

In all the above great care must be taken to observe all clinical symptoms, and treat accordingly. The reactions are carefully noted in the temperature charts, and the doses modified according to the reaction obtained. As a solution of arsenic is more quickly absorbed than that administered in bolus, it is considered safe to administer them as such on successive days, as in System A, the solution having become assimilated long before the absorption of the bolus has taken place. Atoxyl, being rapidly absorbed by subcutaneous administration, has also been disposed of before the absorption of the solution *per os* can be effected.

Big horses in the above mean animals weighing 800 to 900 pounds, and ponies in the above mean animals weighing 200 to 300 pounds.

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NOTE BY EDITOR.

An outbreak of dourine occurred in Taylor County, Iowa, U.S.A., in June, 1911. Four stallions and five mares were affected. No authentic information as to the origin of the outbreak was discovered, but it was suspected that the infection was brought by a Percheron stallion which was imported in 1909, and brought direct to Lenox, Iowa.

IV. SURRA.

By Colonel H. T. PEASE, M.R.C.V.S., I.C.V.D.

Definition.—Surra is that form of trypanosomiasis due to the entrance into the blood of a specific hæmoflagellate named the *Trypanosoma Evansi.*

Nomenclature.—Surra is the name which was given to this disease by the natives of Dera Ismail Khan, where it was discovered. It means "rotten," "dried up," etc. The disease is known in various parts of India by different names, having either a similar meaning to the above, or having, in the case of some animals, reference to certain of the symptoms, such as *Zahrbad,* meaning "œdema," or to the length of time it lasts, as *Tibarsa,* signifying "lasting three years." It will be readily understood that in India, with its great diversity of languages and dialects, the variations in nomenclature are enormous, and no good purpose would be attained in quoting them here.

HISTORY.

To Griffith Evans belongs the credit of the discovery of the first pathogenic trypanosome, the *T. Evansi.* He recorded it in a report of November, 1880, to the Government of the Punjab in connection with a disease occurring amongst the horses and camels belonging to the 4th Punjab Cavalry at Dera Ghazi Khan, on the North-West Frontier of India.

Steel, five years later, reported on an outbreak in British Burma. Gunn next found it, in 1886, at Kohat, on the North-West Frontier. Nuttall saw it in the United Provinces in 1886-87.

Practically no work was done in connection with it until the advent of Lingard, whose first exhaustive report on the subject was published in 1893, to be followed by numerous others.

Since 1893 our knowledge of its distribution and importance has been greatly enlarged, and it is now certainly recognised to be the most important and widespread malady affecting solipeds and camels which we are called upon to deal with in India and the neighbouring countries in the East.

GEOGRAPHICAL DISTRIBUTION.

Considerable confusion has arisen regarding the geographical distribution of surra, owing to the fact that the trypanosome was the first pathogenic one to be discovered. As new ones were found, it was often concluded that they belonged to the same species. An examination of the subject shows that many of the supposed centres must now be eliminated, as the forms of trypanosomiasis found in them are due to other species of parasites. The subject is, however, to a great extent veiled in uncertainty, owing to the difficulties in definitely deciding as to the identity or otherwise of various of the trypanosomes, which very nearly resemble each other morphologically.

The countries in which trypanosomiasis is admitted to be due to infection with *T. Evansi* are roughly mentioned below.

Recent investigations in India show that surra is extremely widespread over the country, and that wherever the conditions are favourable for its spread it exists. It is naturally more prevalent along the course of rivers, in marshy ground, in the low-lying rice-lands, in marshy places in the low hills which are jungle-covered, etc.

In Burma, Manipur, and Assam the same is true. It has been noticed in Persia by Haig. Blanchard recognised it in Tonkin in 1886, and the disease is now known to be enzoötic in practically all the provinces of French Indo-China, becoming epidemic from time to time, the regions most affected being Upper Tonkin, Annam, and Laos. The disease also occurs on the borders of Cochin China and Cambodia. It appeared in the Dutch East Indies in Java in 1900 in horses and buffaloes, and has now become enzoötic there. In Sumatra, Vrijburg, the veterinary officer at Deli, found it; and Leicester investigated an outbreak in Perak, where it had probably been imported from Sumatra. In 1901 Smith and Kinyoun reported the disease from the Philippines, and a history of the epidemic in that country has been published by Musgrave and Williams. Since its introduction it has spread throughout the Archipelago.

In the Mauritius the disease was discovered in 1902, and from July to October of that year, 1,882 solipeds and 1,681 cattle are reported to have died there. It is said that so many animals perished that planting and gathering of crops became impossible, the mortality being 100 per cent. in horses, and from 25 to 30 per cent. in cattle.

Gibson, in 1903, discovered the disease at Hong-Kong. In the Federated Malay States it was discovered in 1903 by Ford, and is now recognised to be enzoötic there. It is said to exist in some parts of Africa.

GENERAL REMARKS.

Surra is a specific febrile disease occurring in many mammals, and capable of being inoculated from one animal to another. It is characterised by an intermittent or remittent, and sometimes by a relapsing, type of fever, which varies in severity, and lasts for a period varying from a few days to some months in the different species of animals, and generally presents definite symptoms, following each other in regular sequence.

The number of the parasites in the peripheral blood varies considerably from time to time during the attack. There are periods when they are *apparently* absent, and cannot be demonstrated by ordinary microscopical examination. But a sub-inoculation of the blood in sufficient quantity at such times into a susceptible animal reproduces the disease, and parasites appear in the inoculated animal after the usual incubation period, thus proving the presence of the mature parasite in small numbers, or of some unrecognised developmental form of it.

At other times the parasites are more numerous, and occasionally the blood simply swarms with them. The behaviour of the parasite in this respect varies in the different species of animals, and also in animals of the same species. In some cases the parasites appear in crops, and there are well-marked periods of paroxysms when the parasites are numerous, and intermissions when they are few or apparently absent. This phenomenon may be very striking in some instances, but not so in others. They seem to increase and decrease in numbers at uncertain intervals, being in the majority of cases most numerous when the fever is high, and they are not generally demonstrable by microscopical examination when the temperature is close to normal, excepting towards the termination of a case. This, however, does not invariably occur.

The symptoms and course of the disease induced by the parasite vary very considerably, not only in the different species of animals attacked, but also in individual cases in the same species. Thus, in the horse infected from another horse or from a bullock the disease runs a comparatively rapid course, and is accompanied by well-marked symptoms, which are quickly evolved. There is intermittent fever, marked by paroxysms and intermissions, and the presence of parasites in the blood in great numbers during the paroxysms. There is rapid wasting of the tissues, in spite of the maintenance of a good appetite. Œdematous swellings of the limbs, sheath, and under the abdomen, soon make their appearance, and paralysis and death occur generally in about one or two months. In a horse infected from a camel similar attacks of intermittent fever occur, but the general symptoms are more slowly evolved. The animal maintains condition for a considerable time before beginning to lose flesh, and death may be delayed for six months or more in some cases. In the camel the disease may run either a rapid or prolonged course. The same general symptoms are noted: very marked intermittent fever, loss of condition, but less œdema. Death may result in a couple of months, or be delayed for four years, whilst some recoveries occur.

In cattle the same variation of virulence is to be noted, those living in countries where the disease is enzoötic and widespread being endued with great tolerance of the parasite, and generally showing little evidence of its presence in the system. Indian cattle and buffaloes are of this description, and although the parasite is widespread amongst them, the number present in the blood is generally few, and the symptoms of its presence are seldom shown, some of those in the best of apparent health and condition carrying the parasites in their blood. The case is, however, very different where the cattle from countries in which the disease has been previously unknown become infected with the parasite. This has been well illustrated in the case of the introduction of the disease from India to Mauritius, where severe symptoms were present, and the mortality amongst cattle reached 25 or 30 per cent.

The disease is influenced in some cases also by frequent passages through susceptible animals of the same or of different species. Thus, passing the parasites from the camel through the ox to the dog increases its virulence. Continued passages through the horse or mouse have the same effect ordinarily for the horse. Terry recently proved that, as a result of repeated passages through guinea-pigs, the virulence of the surra of Mauritius for mice became greatly attenuated; and while the virulence of the parasites decreased for mice, it increased for guinea-pigs. But by cultivating the parasites in mice the virulence for these animals was restored after four to eight passages.

The chief symptoms are the occurrence of petechiæ or blood extravasations on the mucous membranes, chiefly those covering the *membrana nictitans*; œdematous swellings on the limbs, sheath, and inferior aspect of the abdomen and chest, depending on exudation of a yellow semi-gelatinous material into the subcutaneous and other connective tissues. The animal rapidly loses condition and strength, although in the great majority of cases the appetite remains good. Throughout the entire course there is progressive anæmia, which finally terminates in death.

ANIMALS SUSCEPTIBLE.

Most mammals are susceptible to inoculation, and so far as can at present be ascertained all the more important ones may become naturally infected. Birds may be inoculated, but the parasite seems to remain in their blood for a short time only. In India, where the disease is enzoötic, none of the mammals appear to escape infection.

Horses, mules, asses, oxen, buffaloes, camels, elephants, goats, sheep, dogs, pigs, and deer, may be attacked.

The laboratory animals—viz., rabbits, rats, guinea-pigs, monkeys, and mice of all kinds—are readily infected. The disease can be transmitted to the goose, but causes no apparent disturbance, and the parasite rapidly dies out. Man seems to be resistant.

ETIOLOGY.

Surra is a wound infection. The cause of the disease is the presence of the *T. Evansi* in the blood. Slowly but surely the opinion of the natives regarding the spread of surra by means of the biting flies, "Tabanus" and "Hæmatopota," which suck the blood of the domesticated animals, has gained ground, and the more we investigate the matter, the more evidence is accumulated that the only important means of propagating the disease is by means of the bites of insects.

Griffith Evans remarked in 1880, in connection with the "Baradhang," which is a "Tabanus," and which the natives informed him was the cause: "It is not improbable, however, that those flies, so excessively troublesome at Quetta and other outposts, which bite horses so that blood streams down the legs as if it had been squirted thereon, do convey the disease from one horse to another by inoculation. It is a fact that the disease does spread mostly at those posts where the horses are closely packed and flies are in the greatest numbers."

So far as the history of the malady in India is concerned, the manner in which outbreaks occur, the localities in which it spreads, the season of the year when the disease appears, and the freedom from epidemics at other seasons, all indicate that some special cause must be at work at the particular season of the year.

Surra occurs exactly in those situations which are most favourable to the breeding of, and suitable as a habitat for, biting flies.

The surra belts are low-lying, marshy lands, subject to inundation, and subsequent partial drying up, and covered with rough grass and brush jungle, and it is such areas that are best adapted to the "Tabanidæ."

Surra appears in such belts immediately after the break of the rains, and continues during the rainy season, and it is at this time of the year that the flies breed out and are present in great numbers. It is only during the time that, and so long as, the biting flies remain prevalent that surra continues to spread amongst horses and camels.

It is a matter of common knowledge that these animals can live in such zones at other times of the year, when no biting flies are present; yet, place the same animals in them during the time that flies are present, and surra will spread, provided that a reservoir of the virus is also present. Moreover, it has been definitely proved by carefully controlled experiment that susceptible animals placed in fly-proof stalls may live through a surra season in such a locality, being fed and cared for in exactly the same manner as controls not protected from flies, and which consequently died from surra.

We have a considerable mass of evidence now available to prove that "Tabanus," "Hæmatopota," and even "Stomoxys," are capable of carrying the trypanosome from a diseased to a healthy animal by acting as simple vectors. Rogers first proved this in India. Musgrave and Clegg have transferred the infection to monkeys by means of biting flies in experiments so guarded as to make the results absolutely conclusive. These experiments have been repeated, and the transmission of the disease to the horse, dog, rat, and guinea-pig by biting flies has been confirmed, the work being conducted with the greatest care and in all cases controlled.

Leese's recent experiments in India prove that the disease may be transmitted from sick to healthy animals by means of "Tabanus," "Hæmatopota," and "Stomoxys" in this way—readily by the two former, and with difficulty by the latter. His experiments also proved beyond a doubt that susceptible animals kept in fly-proof stables in a surra zone during the surra season escaped infection, whilst those kept in the same place in open sheds became infected and died.

In more recent experiments in Malaya, Fraser and Symonds have also proved that the disease is readily transmitted directly by "Tabanus" and "Hæmatopota," but they failed with "Stomoxys." It is doubtful whether other biting insects have much influence in spreading the disease in the larger mammals. Mosquitoes have been suspected, but nothing has been proved against them. It has, however, been proved beyond doubt that fleas commonly spread surra in the dog, and that consequently it is not strictly confined to the surra season in packs of these animals.

A careful examination of all the other methods of spread which were put forward before the fly theory had been properly tested must now be abandoned, as they have not stood the test of experiment, nor are they justified by observation.

Surra, being a wound infection, can be spread by any means which might transfer the parasite to a wound, however minute, whether of the skin or of mucous membrane; so feeding on diseased carcasses, drenching with blood containing the parasite, flies going from a wound on a diseased to one on a healthy animal, crows pecking first a diseased and then a healthy wound, and so on, may spread it.

But there is no reason to suppose that tainted food or water could transmit it, nor need we seriously consider the excrement of rats as a cause. Rats may, however, carry infection. Musgrave and Clegg, whose observations agree with our own, summarise the available evidence as follows: "It has been repeatedly shown that biting flies can transmit the infection mechanically. It has been proved that animals protected from insects do not contract the disease in places where it is prevalent, but that when protected from every other source of infection but this one, trypanosomiasis occurs with the usual regularity. Finally, no other suspected method of transmission explains why they are confined in zones where the disease is endemic."

The manner in which the disease spreads through packs of hounds when they are not exposed to the bites of flies may appear to be evidence against the "biting-insect" method of infection. It has, however, been convincingly proved that fleas transmit surra from diseased to healthy dogs in packs, and that once a reservoir of the virus has been introduced—generally by eating the diseased carcass of a carrier of the infection, such as a bullock or camel—the disease may run through a large pack in this way. Fleas can transmit surra from dog to dog, from rat to dog, and from rat to rat.

The question whether the fly invariably acts simply the part of a vector in transmission, or whether in some instances the parasite may not go through some form of developmental cycle in a fly, is a much-debated one. Repeated observation has shown that the latter is, if at all, only exceptionally the case, nor is it at all necessary for the indefinite existence of the disease, with maintenance of virulence of the trypanosome. It seems improbable, too, that any particular species of fly acts the part of a definitive host.

It has, however, long been considered possible that a developmental cycle can occur in an invertebrate host, but, unfortunately, there is not much reliable information recorded on this subject in regard to *T. Evansi.* A good deal of evidence has recently been accumulated in regard to other trypanosomes—*T. Gambiense* and *T. Lewisi,* for example. Prowazek, Baldrey, and others, have traced the development of *T. Lewisi* in the rat, louse, and flea. More recently Klein and others have established distant transmission of *T. Brucei* in *Glossina palpalis* up to sixty-six days after the infective bite. Bruce has confirmed this observation as regards *T. Gambiense* and a parasite of the *dimorphon* group. Klein and Taute have traced the development of *T. Gambiense* in *G. palpalis.*

We are, in the absence of experiments, not in a position, therefore, to deny the possibility that some particular fly may act occasionally as a real intermediary host, sucking blood from a reservoir of the virus, the contained trypanosomes multiplying and undergoing changes in the fly's body, and being transmitted to a healthy animal by a consecutive bite after the lapse of some days. If this does occur, it would explain some points which are obscure at present. It does not follow, of course, that this is the usual method of spread, nor does it annul the fact that the fly may also act as a mere mechanical carrier.

All the experiments available seem to point strongly to the fact that in surra no one species of fly is specially concerned in the spread of the disease once a fresh outbreak is established, whatever may occur in regard to the first case in the outbreak. Almost any vector seems capable of spreading the disease mechanically once the trypanosomes are numerous in the peripheral blood of the infected animal. "Tabanus," "Hæmatopota," and "Stomoxys," as well as fleas, may transmit it.

The available information regarding surra is to the effect that these flies, etc., are only capable of spreading the disease for a comparatively short period after an infective meal. Forty-eight hours is put down as the ordinary limit of time for which a fly remains infective after biting a diseased animal. Unfortunately, however, it has been shown by Klein and Bruce in regard to other trypanosomes and flies that *Glossina morsitans* fed on infected sheep (*T. Gambiense*) proved infective for fresh animals on the first three days, then non-infective from the fourth to the tenth day, and infective again from the eleventh to the forty-fourth day. Only a small percentage of certain flies give these results. The experiments which have been carried out with flies on surra are, so far as my memory serves me, incomplete, as, although they show that mechanical transmission is very easy, they have not been carried on for a sufficiently long period of time to eliminate the possibility of a non-infective interval and distant transmission.

While, therefore, admitting the possibility of an insect intermediary host, the bulk of the evidence at our disposal appears to indicate that this is not the most serious factor in the spread of the disease from animal to animal in the course of an outbreak, but that it is mechanical transmission which plays the more important part. There is no doubt whatever that no particular species of fly can act as the sole agent in the spread of the disease. Yet even here it is difficult to explain why the bites of many flies prove non-infective after an infective meal, and there remains a suspicion of doubt whether the evidence is so reliable as to entirely exclude a developmental cycle in the fly. The question is, then, at present in an unsatisfactory state.

Whatever may occur in Nature, there is no doubt that the disease may be carried on for many years artificially by a simple wound infection, the slightest prick with a soiled needle being sufficient in many cases to transmit it to a healthy susceptible animal.

Perhaps the safest conclusion in regard to the rôle of the fly in transmission is that a developmental cycle occurs in certain flies, and that outbreaks may in this way arise, the fly taking the parasite from the blood of a "reservoir of the virus," in which it may be very scanty, multiplication and development occurring in its body, and the reinforced parasite being passed on by a subsequent bite to a susceptible animal. Once the outbreak is induced, the disease can be spread by the mechanical intervention of biting flies, etc.

For the spread of surra in the larger mammals, then, three things appear to be necessary:

1. A reservoir of the virus—*i.e.*, an animal carrying the parasite in the blood. Such animals are plentiful in places where surra is enzoötic. Cattle, buffaloes, and camels may all carry the parasite for long periods of time, and furnish the necessary reservoir. But it is when the disease has once declared itself in animals like equines that it is most rapidly

spread to other animals, as the parasites exist in such great numbers in the peripheral blood, which is only free from them for short periods during the course of the disease. Camels are also very dangerous. Rats were proved to have been important factors in Manila.

2. A means of transmitting the parasite from the reservoir or the diseased animal's blood to that of a susceptible animal. This, as has already been said, exists in the form of biting flies and fleas.

3. The presence of susceptible animals.

THE PATHOGENIC AGENT.

The causal agent of surra—*T. Evansi*—closely resembles the *T. Brucei*, the parasite which causes nagana in Africa; *T. equiperdum*, the cause of dourine; and *T. Elmassiani*, the cause of "mal de Caderas"; and it is really indistinguishable from them morphologically. It appears in the fresh blood as a small, actively lashing, eel-like body. By its movements it gives to the corpuscles a peculiar quivering movement, which is very striking, and, once seen, is not likely to be forgotten. Its activity in the fresh preparation becomes more sluggish after a time, and we are better able to make out more of the details of its structure. It will then be seen that it is a spindle-shaped, more or less elongated protoplasmic body, possessing a folded membrane, along the thickened border of which runs a flagellum which has a free portion of variable length.

To make out further details regarding the structure of the parasite, it must be stained by one of the classical methods, the chief of which is that effected by a mixture of eosin and methylene blue in definite proportions (Romanowsky's method). Laveran recommends the use of Borrel blue, 1 c.c.; eosin solution (1 per cent.), 4 c.c.; distilled water, 6 c.c., in which the blood-film is left from five to twenty minutes, well washed, and then treated with tannin solution for several minutes; again well washed, and dried. Giemsa's method is also very useful, and gives excellent results in smear preparations. Very handy, also, are the powders prepared by Jenner, Leishman, J. H. Wright, etc. Any of these methods suffice for ordinary purposes, especially Leishman's and Giemsa's. For studying the cytology of the parasite, however, it is said that smears prepared by the method of Salvin, Moore, and Breinl are calculated to give a far truer conception of the form and structure of the parasite.

The method of Salvin, Moore, and Breinl, or some modification of it, certainly seems the most rational for the study of the cytology of trypanosomes, and we quite agree with them that the study of the structure of these organisms cannot suitably be made upon material after it has been heated and dried—a method which, to say the least of it, may be shown, so far as the finer details of any cell structure are concerned, to be particularly barbarous.

Staining for Examination by Breinl's Method.—Place a very thin layer of albumin-glycerine on a clean slide. The best method is, perhaps, to put a drop the size of a large pin's-head on the slide, and to spread this out with a clean duster over the slide. On top of this layer spread a drop of blood in the usual way, and dip the slide, while wet, into the fixing solution (Fleming's strong solution is usually used). Leave it for about five or ten minutes, wash immediately in water, and pass the slide through alcohols in consecutive order, increasing by 10 per cent. at a time to absolute alcohol; then back from absolute in 80 per cent. alcohol in which is contained iodine and iodide of potassium (make up a concentrated solution of potassium iodide in water, and iodine in alcohol, mix them together, and add them to some 80 per cent. alcohol until the mixture becomes a dark brown colour). Leave the slide in this for five or ten minutes, and then bring it into 30 per cent. alcohol. Use for staining either aniline safranine (Babes) or the following solution: Prepare a concentrated watery solution of safranine (Grubler) and a concentrated alcoholic solution of safranine, mix them in equal parts, and then add pure aniline oil. Shake from time to time, and leave the solution to ripen for three to six months. Stain in this solution for from half to two hours. Wash off the safranine, and stain afterwards with polychrome methylene blue (1 gramme methylene blue *purissimum medicinale* (Höchst), 100 c.c. distilled water, and 0·5 gramme sodium carbonate, placed in an incubator to ripen; the older the solution the better the staining properties). Wash off the methylene blue, and differentiate with Unna's orange tannin as long as the blue stain comes out. Bring the slide up through alcohols, as above, into absolute alcohol. The film has thence a reddish colour. Now dip the slide into aniline oil until the reddish colour changes to a purple-blue tinge; the aniline oil takes out at the same time the excess of blue stain left by the orange tannin. Clear in xylol, mount in Canada balsam under a cover-slip.

The staining may be done by the modification of Heidenhain's hæmatoxylin method, by fixing and treating the film, clearing the slide from the alcohol containing iodine and iodide of potassium, and passing it through successive alcohols, as above described, into water. It is then stained in a 3½ per cent. solution of iron alum for one hour, washed, and put into the following solution: 0·5 gramme hæmatoxylin dissolved in 100 c.c. distilled water, to which, after the hæmatoxylin has dissolved, a few drops of concentrated watery solution of lithium carbonate is added. Stain for half an hour, and then differentiate in the usual way with iron alum. When suitably stained, the parasites will be found to consist of an elongated spindle-shaped cell, in which an anterior and a posterior end are arbitrarily defined.

When suitably stained by Leishman, Giemsa, etc., it will be seen that they consist of a body the protoplasm of which stains blue, and it will at times, according to Laveran, be seen to contain a few chromatic granules which stain a deep violet colour, and are far more scanty in *T. Evansi* than in *T. Brucei;* but the number varies so in the two parasites that it is impossible to confirm the statement.

There is a difference of opinion as to whether the body has a definite investing membrane or not. Salvin, Moore, and Breinl consider that the exterior protoplasm is differentiated into a thin outer layer or periplast. This appears so by the use of their stain, but is not seen in parasites stained by other methods.

Situated in the middle of the body of the parasite is an oval nucleus, also named macronucleus and trophonucleus, varying considerably in size and shape. Its length varies from 1·5 μ to 5 μ, the average being 3 μ. It appears to be composed of a number of chromatic granules which stain deeply of a purple colour. Some authors divide the cell into a central area which stains a purple colour, and the whole of which is named the "nucleus." Within the nucleus they find a clearly defined body which stains under the same conditions red, and this they call the "intranuclear centrosome."

Near the broad and commonly called posterior extremity is a small deeply stained chromatic body which is usually named the "centrosome," or by some authors the "extranuclear centrosome." It is a small homogeneous mass of chromatin which stains a deep violet.

Extending from one side of the body of the parasite along a part of its length is a very thin and folded membrane which stains lighter than the body protoplasm, and to which the name "undulating membrane" is given. Its hyaline appearance sharply demarcates it from the cytoplasm.

From the centrosome springs a lash-like structure which runs along the free edge of the undulating membrane, and is then prolonged beyond the end of the body into a free part. This we name the "flagellum."

There is a considerable range in the size of the parasites as recorded by various authors. Fraser records that the smallest specimen found by him in the liver measured only 13 μ in length, inclusive of the free portion of the flagellum, and 1·5 μ at its broadest part. In the peripheral blood, however, such small forms have not been recorded, and the smallest may be taken to be from 18 μ to 20 μ long. The general consensus of opinion appears to be that the small forms are 20 μ on an average, and the largest 35 μ long. There is also a difference of opinion regarding breadth. Lingard found the mean of eighty parasites to be 1·64 μ broad. Laveran and Mesnil say the average is 1·5 μ; but larger forms measuring 2 μ to 2·5 μ wide are not uncommon. In the Mauritius outbreak the parasite is said to have measured 33 μ long, and the width, not including the undulating membrane, was 1·5 μ to 1·7 μ.

In Malaya Fraser found the breadth to be 3 μ to 3·4 μ, but this probably included the undulating membrane.

The question of the behaviour of the parasites in the blood has long been a much debated one. The most widely accepted theory has been that the parasites multiply in the blood solely by longitudinal division during the paroxysm up to the point when agglutination and trypanolysis occurs, when most of them perish, only some of the more resistant surviving and awaiting favourable conditions for multiplying. There is, however, a growing tendency to believe that a definite cycle of development occurs in the blood of a susceptible animal, and that, at trypanolysis, some of the parasites form latent, resistant, non-flagellate bodies, which form the nucleus for the new crop when conditions become again favourable. The resistant forms were described by Moore and Breinl. Fantham has recently confirmed their observations. He says that the bodies are formed by the anterior end of the trypanosome disintegrating and being cast off, while the blepharoplast gradually migrates nearer the nucleus, and then the non-flagellar or posterior end of the original trypanosome is cast off, together with the remains of the flagellum. The rounded body, consisting of chromatin, with a thin layer of cytoplasm, has then surrounded itself with a definite, very thin capsule (cyst). This change is said to be most active in the lungs, heart, and spleen, but the oval forms can be seen also in the peripheral blood. Fantham describes the metamorphosis of these latent bodies into trypanosomes. The bodies grow larger and send out a pseudopodium-like process, which lengthens, and a flagellum is formed from an area close to the blepharoplast. The parasite forms at its drawn-out anterior end an undulating membrane, along the edge of which the flagellum lies, and the organism somewhat resembles a Crithidia. The organism grows, and the blepharoplast passes posterior to the nucleus, and the trypaniform stage is assumed. Fantham claims to have reproduced trypanosomiasis by the injection of these latent bodies only, but admits the difficulty of definitely excluding trypanosomes from the material employed. His experiments, however, give very strong support to the theory that there is a life-cycle of trypanosomes in vertebrate hosts, comparable to those of Crithidia and Herpetomonas in the alimentary tracts of various invertebrates. The latent stages of trypanosomes occurring in vertebrates are separate from and in addition to stages of the parasite which may occur in the invertebrate carrier.

Fantham's researches seem to clear up many obscure points, and to explain the significance of the various developmental forms which have from time to time been described by numerous observers.

When the trypaniform stage is reached, the parasites reproduce themselves more or less rapidly, in accordance with the susceptibility of the animal and other causes, by longitudinal division.

When about to divide in the blood, the parasite becomes longer and larger; the centrosome elongates and divides into two, the daughters separate some distance from each other in the long axis of the parasite, and from each centrosome a flagellum appears to take its origin more or less directly. I think that the flagellum actually divides. There is, however, a difference of opinion about this. The nucleus also becomes elongated, the chromatin collects at the extremities, and division takes place, when the protoplasm also separates between the two divided nuclei, and after a short time the divided parts separate, forming two distinct parasites. Sometimes further division occurs before the original protoplasm divides, and in this way parasites containing four centrosomes and four nuclei are produced.

Separation occurs from the anterior part first, and the parasites swing apart usually into one long line, remaining connected for a time by their posterior extremities; finally detachment is complete. The newly divided forms are not small, the two separate parasites being generally of equal size, and measuring from 20 μ to 30 μ long, and not exceeding 2 μ in breadth.

The parasites are generally separate from each other in the blood, but towards the end of a paroxysm, and especially when they are very numerous, they become collected into masses or agglutinated previous to the occurrence of trypanolysis. In some rare cases rosette forms may be noted, but they are very uncommon, and have only been seen by me in chronic cases in the camel.

If at the time of death mature trypanosomes are plentiful in the blood, they will be found in every organ of the body if examined soon after death. If they are few, then they are not readily found in any organ. In the last stages, when parasites are very numerous in the blood, numerous degeneration forms are found, gradually passing down to the débris of the parasites themselves. Fraser notes the following distribution of the parasite in the body:

"In the lungs parasites are found, but in rather less abundance than in the heart's blood. In the liver they were as numerous as in the heart-blood, occasionally more so. In the spleen trypanosomes tend to disappear very rapidly after death. In the kidney parasites were found, although not usually in abundance. In the glands parasites were invariably absent or scanty. In brain squashes they were usually scanty, although on one occasion a cerebral capillary was found to be occluded by them. In bone-marrow, trypanosomes could be found, but there was no evidence to incriminate this as a site of their evolution. Animals have been killed when the trypanosomes have been just beginning to appear in the peripheral blood and when they were just about to disappear; careful examination has shown that no organ was at either of these times a special seat of the parasites."

Trypanosoma Evansi outside the Body.—Outside the body the parasite soon dies, presenting in blood films various degeneration forms.

It quickly dies under all tried environment outside the body of some living creature, and no evidence of its existence in water, grass, or similar places, can be advanced. It may be kept alive in a sealed specimen of blood for about forty-eight hours. Laveran and Mesnil state that blood containing the parasites, if defibrinated or mixed with citrated salt solution, and kept at room temperature or in the ice-chest, does not preserve the parasites, for what remain after three days are avirulent. In the cadaver they have usually perished in about forty-eight hours.

There is no evidence to show that the parasite is capable of existing outside the body, except in the body of an invertebrate, in any form whatever, either in water, on grass, or in any other known medium, and all attempts to infect water and pastures have failed so far. It is very susceptible to heat.

Culture.—Novy, McNeal, and Hare apparently succeeded in cultivating the surra trypanosome, using a medium composed as follows:

Extractive of 125 grammes chopped beef in distilled water	1,000 c.c.
Agar	20 grammes.
Peptone	20 grammes.
Common salt	10 c.c.

The tubes were allowed to stand until there were drops of water of condensation in the tube, and were then inoculated with 2 drops of blood containing numerous trypanosomes of surra. They obtained a culture which differed markedly from that of nagana in that the trypanosomes were larger (25 μ to 35 μ average). The flagella were very long; they were very active, motile, and the protoplasm of the anterior end contained a large number of small granules or globules about 0·3 μ to 0·5 μ in diameter, and of a yellowish or greenish tint, and there was an entire absence of rosettes and colonies. Subcultures were not obtained, and attempts to infect animals failed.

Thomas and Breinl also apparently succeeded in obtaining cultures.

Laveran succeeded only once in six experiments.

Neither we nor, so far as I am aware, any other Indian observer has met with success, and we are inclined to think that all sources of error have not been eliminated in the previously recorded successes. The discovery of trypanosomes other than those of surra by culture from the blood of cattle lends colour to the suspicion that this may have been mistaken for the cultural form of surra.

MORBID ANATOMY.

The pathological lesions found in animals dead of surra are very varied in character, but there are practically no constant changes which can be said to be diagnostic beyond those which may be noted during life.

When the carcass of an animal dead of surra is examined soon after death, the ante-mortem lesions will still be present. We find that the body is greatly emaciated, and that the skin and hair are harsh, the latter being sparse. There is in the horse a considerable amount of œdema of the legs and under the belly. This may be present in other animals, but is generally not so well marked. In the camel the hump is very small.

The mucous membranes have generally a pale yellowish colour. The skin is difficult to remove, the subcutaneous tissue being infiltrated with a yellow gelatinous material at the seat of œdema, and being dry, hard, and colourless in other situations. A yellow gelatinous material will be found in those parts of the body where areolar tissue and fat are normally present. All the organs are anæmic.

In the horse we find numerous subserous hæmorrhages, particularly over the right side of the heart and under the pleuræ.

The lymphatic glands are enlarged, and the lymphatics themselves to some degree. The heart muscle shows parenchymatous changes corresponding to the duration of the disease. Its chambers usually contain large clots, graphically compared by Musgrave and Clegg to chicken-fat.

The one organ which shows the most constant changes in acute cases, such as occurs in equines, is the spleen, which is always enlarged to a greater or less degree, and in some instances assumes very abnormal proportions. Musgrave and Clegg consider that there is some relation between the enlargement of this organ and the number of parasites present in the blood at the time of death. Lingard notes that it assumes abnormal proportions in the case of horses dying after the hæmatozoön has been swarming in the blood for some days; when, however, the parasite has been absent for any time, the enlargement may be slight. In animals in which the parasites are not usually present in great numbers or for any long period at a time, the splenic enlargement is slight or absent. This is generally the case in oxen and buffaloes, as well as in camels and animals which have been inoculated from them. When enlargement is present in the horse, the organ is softer and more friable than normal. The surface is uneven, due to enormously swollen corpuscles, which stand out prominently. The capsule will often be seen to be dotted over with irregular-shaped ecchymoses of a brick-red colour. The dotted appearance is generally present, even when the enlargement is slight.

On the state of the liver in post-mortem examinations of horses in India no reliance can be placed, as most horses show old signs of perihepatitis, and few have not got some enlargement of the organ.

PERIOD OF INCUBATION.

This is generally regarded as averaging about five days, but in some cases it may be as long as a month.

SURRA IN THE HORSE.

Naturally Contracted Surra.—The disease in the horse is characterised by repeated attacks of fever, and the presence of the parasite in great numbers in the blood usually only during the paroxysms of the febrile attack. Severe progressive anæmia occurs, with its attendant wasting, œdematous swellings, and cardiac palpitation.

The red blood-corpuscles are very considerably reduced in number (as much as 50 per cent. in some cases) following the increase of the trypanosomes in the blood. The hæmoglobin is similarly reduced. Surra is insidious in its attack, and its invasion is not marked by any diagnostic symptoms, unless the blood be examined microscopically. Cases occurring in practice are generally not brought for treatment until the disease has made considerable progress, and the symptoms then present are sufficiently suggestive. The horse is in wretched condition, the coat is rough, and the hair falls out in places; the skin is dry, harsh, and scrawny; and œdema is marked on the limbs, sheath, and under the belly.

The mucous membranes are pale, and on the conjunctival membrane are found dark-coloured petechiæ. Slight catarrhal symptoms are also sometimes noted. The history of the case is that the animal has had a few attacks of fever at intervals, and has rapidly lost condition, although maintaining a fairly good appetite. A horse brought in this condition for treatment in the surra season in a country where the disease is enzoötic may be at once suspected of surra, and such cases present no difficulties in diagnosis if a microscopical examination of the blood be made when the fever is present. At this period the *T. Evansi* can be generally detected. We cannot give a better account of the symptoms and course of a case in which the disease has been diagnosed early than that given by Lingard, as follows:

The invasion of this disease is usually marked by symptoms of a trivial character: the animal is dull, the skin hot and dry, the appetite lessened, and the temperature is elevated. In some cases an urticarial eruption may appear on the skin. If the blood be examined microscopically during the febrile stage, it may be found normal in appearance; but in the majority of cases a number of small, rapidly moving organisms may be observed lashing about and imparting to the blood, as they pass amongst the corpuscles, a peculiar vibrating movement, which, if once observed, will not be readily forgotten. These parasites are not constantly found in the blood during the whole course of the disease, but generally occur in considerable numbers during the febrile period, and are not easily found in the apyretic periods.

This condition does not last for more than a few days, during which the fever continues; but the temperature oscillates, and finally falls to the normal limit, the horse becoming apparently healthy, and presenting no symptom whatever of disease. A second attack of a similar nature, however, soon supervenes, generally in the course of a few days. The animal again becomes dull, the temperature runs up, and the usual symptoms of fever appear. During the second attack other symptoms generally manifest themselves, such as a paleness of the visible mucous membranes, with dark-coloured petechiæ on the *membrana nictitans*. The animal will be observed to be rapidly losing weight and condition, although in the intermissions the appetite is ravenous. The heart-beats become somewhat irritable, and œdema is commencing to appear—as a rule about the hind fetlocks. If the blood be examined microscopically during the second period of fever, parasites will be found in it in varying numbers.

After the fever and accompanying symptoms have, for the second time, been present for some days (one to six), the temperature again drops, and an intermission occurs, during which there is considerable remission of all the symptoms, which leads the owner to think that recovery is taking place. But, unfortunately, this condition does not continue for any length of time, for another attack of fever and a general exacerbation of all the above-mentioned symptoms is noticed. The symptoms shown during the third period are usually as follows: The animal is dull, off his feed, very thin, the muscles much wasted, and the bones showing markedly through the skin. The respirations are quickened, and a considerable amount of œdema is present on the hind-legs, sheath, and under the belly. Anæmic palpitation of the heart may be noted. The pulse shows an irritable heart; it is quick and, at times, intermittent. The visible mucous membranes are yellowish, the conjunctival membranes covered with petechiæ, and a discharge may be present from them.

In the blood a repetition of the previous events takes place.

These periods, alternating with and without fever, may go on for some time longer, the other symptoms noted gradually increasing in severity, until at last, towards the termination of the disease, the animal has the appearance of a living skeleton, with much œdema of legs, etc. When the disease has fully developed, the emaciated animal presents a dull, listless, semi-somnolent appearance; it is disinclined to move, but when made to do so staggers, and there is evident weakness of the hind-quarters. From now onwards the progress of the disease is rapid; the animal becomes exceedingly weak, and exhibits manifest loss of power over the hind-quarters. Paralysis of the *sphincter ani* is often seen at this stage. Anæmic palpitation is very marked, the heart-beats being so violent as to shake the whole body at each beat. In some cases the animal suddenly drops dead. In others it becomes too weak to stand, falls to the ground, and in this position may live for a day or two, and, after some struggling, dies.

Analysis of Symptoms.

Skin.—Urticarial eruptions are occasionally noticed on the skin.

Temperature.—In inoculated cases the temperature curve is variable. In animals in which the disease is severe, however, we generally find that the entrance of the parasite into the blood is followed by relapsing fever, although in some cases it is remittent. The rise and fall of the temperature is concomitant with the presence or absence of the parasite, but when the disease has been running on for some time there comes a period, some days before death, when the temperature remains elevated until immediately before death, when it may become subnormal.

Pulse.—The pulse is at the commencement of the disease full and strong during the paroxysm of fever, but seems normal during the fever-free period. Soon, however, as the anæmia progresses, the pulse becomes gradually weak and thready, and in many cases dicrotic. It is naturally weak during the paroxysms of fever, and generally regains about the normal number of beats during the apyrexial periods. Towards the end of the disease the pulse is much disturbed, increased in frequency and altered in character.

The Blood.—Bruce noted that in nagana there is a diminution in the red corpuscles, from 5,260,000 to 1,800,000. Kanthack, Durham, and Blandford have also noted that anæmia occurs in some trypanosome infections, but not, in their opinion, sufficient to account for death. The evidence on this point in surra is not very extensive, but numerous authors have recorded the fact that severe anæmia occurs with decrease of the red corpuscles. Lanfranchi has recently shown that there is a considerable reduction of the red corpuscles following the increase of the trypanosomes in the blood. The hæmoglobin is correspondingly diminished. The lymphocytes are generally increased and the polymorphonuclears decreased.

The red corpuscles, as first pointed out by Lingard, exhibit a marked degree of agglutination, clumping together into masses, the blood having a laky appearance. There is, of course, a certain amount of auto-agglutinin in the normal blood of the horse. The condition found in normal animals may be accentuated by errors in cleanliness and technique, but if well-prepared cover-slip preparations are used it will never approach in intensity the well-marked clumping obtained in infected cases, as pointed out by Yorke, who has proved that auto- and iso-agglutinin are present in the blood.

Digestive Apparatus.—The appetite is usually persistent throughout, excepting at times when the fever is high. This is a marked peculiarity of surra, as in no case of other forms of fever do we find an animal maintaining the appetite which we see throughout the course of this disease. The tongue and mucous membrane of the mouth become first pale and later yellowish in colour. The bowels are, as a rule, constipated, and there is evidence of hepatic disturbance in the colour of the fæces and the fact of their being coated with mucus. Diarrhœa may come on towards the end of the disease, but it is not invariable.

Yellowness of the mucous membranes, probably due to the destruction of the red corpuscles, the liberation of the colouring matter of the blood, and the staining of the plasma, is a very common symptom at one period or another of the disease. It sometimes appears early in the first paroxysm, and may be evanescent in character, appearing with each paroxysm, and gradually fading during the intermission. In the majority of cases, however, the yellow colour of the membranes is marked and persistent.

Urinary System.—The urine changes in colour after the disease has somewhat progressed, becoming viscid and green in many cases. The reaction is usually alkaline until five or six days before the termination of the attack, when it becomes acid. Albuminuria appears after the disease has run a part of its course, the quantity of albumin passed being very considerable as the disease progresses. Microscopical examination of the urine generally reveals the presence of epithelial cells, blood and granular casts, signs of a desquamative nephritis. The evidences of renal disease other than those found on microscopical examination are usually not well marked.

Organs of Vision.—Ocular lesions are not so common in the horse, but are frequent in other animals. The form usually seen is a non-ulcerative interstitial keratitis, which is provoked by the multiplication of trypanosomes in the interlamellar spaces of the cornea. The proliferation of the parasite leads to an infiltration of leucocytes, then a vascular development. These lesions may cause complete disorganization of the cornea, or may disappear, leaving very slight traces after them.

The whole eye is generally affected. The anterior chamber may be filled with blood or with a fibrinous exudate, and the surface of the iris covered with a layer of trypanosomes, below which the tissue is infiltrated. The iris and even the vitreous humour may also be the seat of cellular infiltration.

Nasal Mucosa.—Ulceration inside the nostrils has been noted by Steel. There may be slight epistaxis. Petechiæ are constantly present on the Schneiderian membrane, which may be tumefied. During the second week after invasion there is generally a slight discharge of clear fluid from one or both nostrils.

Complications.

The most frequent complications noted in this disease are catarrhal nephritis, broncho-pneumonia, œdema and congestion of the lungs in the horse, and broncho-pneumonia or œdema of the lung in the camel and oxen. In the camel also are noted intractable sarcoptic mange and skin abscesses.

Differential Diagnosis.

The *sine quâ non* in the diagnosis of surra is the demonstration of the *T. Evansi* in the blood. This, although a comparatively simple matter in solipeds, when the parasites are at times swarming in the blood and the intermissions are of short duration, is far from being so in bovines and camels.

The periods during which the parasites remain sufficiently numerous in the blood to be readily demonstrated by a microscopical examination are extremely short in bovines as a rule, and they soon become too few to be thus discovered, and in camels at some stages the intermission may last for some months. It is thus obviously necessary, when dealing with animals from surra-infected countries, to adopt other methods, and the best of these is the inoculation of a large quantity of blood mixed with saline solution and centrifuged from the suspected into a susceptible animal. This is of great assistance, but is not certain in all cases. Inoculation of very large doses of blood from an infected animal, especially the camel, has not in all cases revealed the presence of the disease, although the organisms have been proved to be present in the blood by a subsequent successful inoculation. In these cases the parasites are extremely few in the peripheral blood, and, so far as my own experience goes, I consider that it is not possible in the present state of our knowledge to decide with absolute certainty in all cases whether a camel or an ox is free from the disease.

In the horse, however, the symptoms are well marked and the organisms numerous in the blood. As the periods of intermission in this animal vary generally within fourteen days, it is only necessary to await the return of fever and examine the blood, two or three slides being examined if necessary.

In testing by means of subinoculation, it is necessary to bear in mind that the period of incubation may be so long as a month in some cases.

Prognosis and Mortality.

The disease is severe and invariably fatal in the horse, ass, mule, and dog. It is highly fatal also in the camel, but some cases recover after long infection, lasting over three years. It is not fatal, as a rule, to bovines and buffaloes indigenous to countries where the disease is enzoötic, but at times it causes a high percentage of mortality—30 per cent. or more in animals from uninfected countries, as occurred in Mauritius. The buffalo and ox become tolerant, and carry the parasite for long periods in the blood without showing any symptoms of disease. They are thus very dangerous, forming reservoirs of the virus, and carrying the parasite over from one surra season to another. Elephants also suffer from naturally acquired surra, and, according to Evans, some animals certainly die from it; but the rate of mortality is so far unknown, and probably, as Laveran says, it is doubtful whether the elephant is to be included in the category of animals in which surra runs an invariably fatal course. The elephant takes the inoculated disease, but does not show any marked symptoms.

Goats are susceptible, as are also sheep. The disease usually runs a chronic course, many deaths eventually occurring after six or seven months. In some cases, however, death takes place within a month.

SURRA IN THE ASS.

The potter's ass is about the only soliped animal that the people are obliged to use in the surra zones in India. They know by experience that both horses and asses die during the surra season, and there are some villages where not a single ass can survive through the surra season, a new supply having to be obtained each year. The disease runs at times a more prolonged course than in the horse. In some cases death occurs within a month, but four to six months may elapse before it takes place. The local symptoms are little marked.

SURRA IN CATTLE.

The symptoms of surra in cattle are by no means constant or well marked. It is quite an ordinary occurrence to find animals carrying the parasite in the blood without their evincing any symptom of disease. As to the number of cattle which succumb to the disease it is not possible in the present state of our knowledge to give any accurate information. It is believed, however, that the mortality amongst cattle in India, if any, is extremely small. All the inoculated cases so far seen have been unaccompanied by any serious symptoms. In some cases, however, emaciation is marked, and if the temperature be taken we may find considerable variations. Usually, however, it does not go much above 104° F. Parasites are few and intermittently present in the blood. They may be absent, apparently, for long periods, and can then only be demonstrated by subinoculations of blood into susceptible animals. The parasite, however, can live for a very considerable period of time in the blood of Indian cattle, which appear to have acquired great tolerance to it. Cattle from surra zones are therefore a source of great danger, as they form reservoirs of the disease. Specially selected cattle from India, in the best of condition, have been found to be the carriers of trypanosomes in their blood after they have travelled to America. It is impossible to say what percentage of Indian cattle do carry the parasites in their blood, but the American figures show 30 per cent.

Cattle in other countries also show great variation in susceptibility. Thus it is recorded that Nocard and Vàllée, at Alfort, inoculated several bovines, one of the animals dying from the diesase.

During the epizoötic in Mauritius the mortality amongst cattle was comparatively high, reaching 25 to 30 per cent.; but if they had to do hard work it reached 75 to 80 per cent. The only noticeable symptom was a periodical febrile paroxysm, which passed unnoticed unless the animals' temperatures were taken. In the Philippines, Musgrave and Clegg found but few cases of natural infection. They observed, however, that animals were susceptible, and the course of the disease, as well as its symptomatology, corresponds to the descriptions given by the best authors in other countries. The incubation period is about the same as in the horse, and the other symptoms begin in very much the same manner. The temperature, as a rule, is less intermittent than in the horse, and does not run so high. Trypanosomes in sufficient numbers to be detected by the usual microscopic examination are intermittently present in the blood, but the latter, as in other animals, is constantly infective by animal experiment. The appetite usually remains good, but there are transient periods of anorexia in almost all cases, and some animals refuse food for days at a time.

Emaciation is rapid, and, as a rule, begins earlier than in the horse. Œdema is decidedly less marked than in the horse, and in some animals is scarcely perceptible; while in others it appears particularly in the dewlap, and less so on the abdomen and hind-legs. The hair becomes rough, and in places falls out. Urticarial eruptions are quite frequent. The course of the disease is generally chronic, and the mortality low.

Fraser, who has written on the disease as it occurs in Malaya, says that the course of the affection in cattle is apparently dependent on the condition of the animal at the time of infection. Animals in good health may show no interference with their condition apart from an occasional rise of temperature. On the expiry of the incubation period parasites are found in the peripheral blood. They may remain present for some days, and continue to be found at intervals, but apparently disappear entirely after several months, when the inoculation of the blood, even in large quantities, into susceptible animals fails to produce trypanosomiasis.

When cattle in poor condition are attacked, the disease usually runs a more acute course. The incubation period ranges from four to seven days, apparently coincident with the occurrence of parasites; the temperature rises, and its subsequent course varies considerably in different cases. As a rule, febrile intervals of a few days' duration alternate with afebrile ones, which show great variation in their extent; even in well-advanced cases a normal temperature may exist for several weeks in succession. In other cases the temperature is irregular. In naturally infected animals which succumb, exacerbations are more frequent towards the end. In animals which apparently recover the temperature is irregular for about six months after the last occasion on which the parasites are discovered microscopically. Cattle may be attacked by an intercurrent disease, or become run down as the result of overwork, and surra may then reassert itself, and death occur.

When the blood ceases to be infective the temperature becomes normal. Clinical signs are indefinite. When there is obvious interference with health, progressive emaciation of the affected animal is the rule, so that it becomes less and less fit for work. Œdema has never been observed. The coat loses its glossy appearance, and the hair falls out in patches. Eczematous eruptions on the head, neck, forequarter, and along the back, may be noticed, the parts affected being denuded of hair. Lachrymation and a mucoid nasal discharge are at times noted. The visible mucous membranes ultimately become markedly anæmic.

As the end approaches the emaciated animal spends its time lying down, and ultimately becomes too weak to rise, and after a few days death quietly ensues.

The parasites in healthy cattle may appear on several occasions, but later tend to remain absent for long periods—for even months—and may reappear for a short time.

At the beginning and end of fatal cases there is usually some relationship between the presence of the parasites and the temperature, but in the intermediate stages this is not so.

SURRA IN THE MULE.

The mule is just as susceptible to experimental inoculation as is the horse, and the mortality is as high. It has been stated by some observers that mules are not so frequently attacked by natural infection as are other animals. This, according to Musgrave and Clegg, may be partly owing to the fact that flies disturb them less. Outbreaks amongst mules occur in India when the animals are placed in conditions favourable to the production of the disease in other animals. They were also found by Steel to be readily infected naturally in Burma. The symptoms in the mule closely resemble those seen in the horse, but they appear to vary somewhat in different countries and in different cases in the same country.

It has been found in India and Burma that the attacks may vary in acuteness and urgency, in some cases evolving slowly and lasting for some months, at others quickly and killing off the mule in a few weeks.

The period of incubation is about the same as in the horse, in the majority of cases averaging about five days. This is followed by the first actual febrile attack, which evolves as in the horse, being followed in a few days by an intermission, after which a relapse occurs. Musgrave and Clegg have noted in mules in the Philippines that the temperature is less remittent and more rarely intermittent, and they have not seen a single case in which the fever was of a relapsing type. This observation differs from that of Steel in Burma, who named the affection "relapsing fever," and records a course of the disease closely in accord with that seen in the horse. In India and Burma the disease may run a fairly rapid course in the mule, but many cases are undoubtedly more prolonged than in the horse, seventeen or more relapses having been recorded. This coincides with the observations made in the Philippines. The more acute cases are evolved as in the horse, whilst in the chronic ones the symptoms appear more slowly. Œdema of the legs is not such a constant accompaniment to surra in the mule as in the horse, but it may occur, as may also anasarca.

SURRA IN THE SHEEP AND GOAT.

Neither the goat nor the sheep appears to be very susceptible to natural infection, but our knowledge of the subject is not very accurate. Sheep and goats, however, do not usually inhabit the sort of country which is likely to be a surra zone. We are not acquainted either with the species of biting flies, if any, which bite the sheep and goat as well as other animals.

Both sheep and goats are susceptible to the inoculated disease, which usually—in Eastern goats and sheep, at any rate—runs a more chronic course than in the horse, and is not marked by very characteristic symptoms. Goats become listless and sluggish in their movements, and lie in the shade a great deal. Œdema is seldom seen, and the animal, although it gets thin, does not become so anæmic nor so emaciated as do other species. The temperature shows occasional rises, but is atypical. The trypanosome appears for a short time only, and is very scanty in the blood, which, however, remains constantly infective for other animals for a considerable time. Death is the common result, though recoveries have been recorded. Edington and Coutts inoculated five goats, all of which died in 96, 55, 43, 33, and 52 days. I have seen them last from four to six and a half months. The same is true of the sheep.

Fraser's goats, inoculated from the bullock, showed parasites on the 6th and 8th days, and died on the 70th and 47th days respectively. Laveran says that they recover in France.

SURRA IN THE DOG.

Until lately the naturally contracted disease has not been found to be of common occurrence in the dog, but in recent years it has been recorded from several countries in the East, and doubtless it is very common. I found it in certain bad surra zones in India. Lingard recorded an outbreak amongst the Bombay foxhounds in 1893. Last year Smith and Gaiger saw the Lahore foxhounds wiped out by it. Spontaneous cases have also been recorded from Indo-China, Hong-Kong, Mauritius, the Philippines, and Malay. Considering that the dog is so highly susceptible to the disease, and that he is readily bitten by the "Tabanidæ," also that he shares the carcasses of animals dead of the disease with the jackals and vultures, it would be a wonderful thing if he escaped infection in surra zones during an epidemic. The disease is doubtless far more common than is generally supposed, and escapes detection because pariahs are seldom examined.

Dogs are very susceptible to surra by inoculation, the slightest scratch being easily infected, and the disease runs a more or less acute course in them, proving invariably fatal. The parasites generally appear in the blood in about 3 to 7 days after inoculation, but may be delayed to 15 days. The number of parasites in the blood varies, at some times being fewer than at others; but it generally increases towards the end of the disease, and at death they are generally numerous. The temperature rises when the parasites appear, and is thenceforward elevated, and the fever is generally of a remittent character. It may be either very high or subnormal just prior to death. There is anorexia, anæmia and rapid loss of condition, œdema of the head and face, occasionally also of the legs and under the belly. The hair becomes rough and falls out.

Catarrhal symptoms, accompanied by watery discharges which become muco-purulent and acrid, are often noticed early, and may become severe. Ocular lesions are common, and vary in intensity from a cloudiness of the aqueous humour to corneal ulceration and total blindness. Towards the end the dog becomes sleepy and stupid, and occasionally paralysis sets in previous to death. The disease, when produced from the horse or ox, runs a more or less acute course. In India the average duration is about 27 days, the longest being 47. Laveran and Mesnil found the average duration 28 days, while Edington and Coutts, in Mauritius surra, ascertained it to be 23·6 days.

The course of the disease induced by the camel trypanosome differs considerably from that due to the horse trypanosome. The incubation period is about the same; it may be up to 20 days, but the disease takes on a far more chronic course. The presence of the parasites is more markedly periodic and the fever more intermittent. The dog at first improves in condition, but later gets gradually thin and weak. He eventually often gets diarrhœa, and becomes very emaciated, the other symptoms of horse surra also developing at times. Death may not occur for some months; in some cases noted by me it has lasted 63, 191, and 288 days.

SURRA IN THE ELEPHANT.

The disease in this animal was brought to our notice by G. H. Evans, I.C.V.D., in 1889-1890, and it is a common affection in certain parts of Burma. To those who are not in constant attendance on and well acquainted with the habits of elephants, symptoms of disease are not easy to interpret, and as during the early stages of surra there are no marked symptoms it doubtless often escapes detection unless systematic blood examinations are carried out.

Evans gives the following as the symptoms: Elephants during the early stages do not exhibit marked symptoms. They become dull, listless, and sleepy-looking. They are disinclined to move about, and, if made to do so, the movements are sluggish. The eyes appear dull and dim, and there is sometimes a discharge from them. The mucous membranes are, as a rule, very pale, and later may take on a yellowish tinge, and may show mulberry-coloured spots, but these are not constant. The bowels are generally normal, but there may be constipation and the dung is covered with mucus. The urine is scanty, gradually becomes viscid, and is often markedly greenish in colour. Gradual but progressive loss of condition and strength is usually present, but is not easily appreciable in these huge animals until it becomes pronounced.

During paroxysms the pulse is quick and strong, and during the intermissions normal, but as the strength fails it becomes feeble. The respiration is not much altered. The appetite remains good. Accompanying the debility and prostration of strength, though not constant, there may be dropsical swellings, which may appear between the jaws, on the lower part of the abdomen and chest, or the feet and limbs may be swollen.

When daily observations of the temperature and microscopical examinations of the blood can be made, it will be found that even with little rise in temperature trypanosomes may be swarming in the blood. The temperature rises to 102° F., but during intermissions it may occasionally be subnormal. The animal is dull and heavy during the paroxysms. The information regarding the course and termination of the disease is not yet definite, but it appears certain that some cases prove fatal, while others recover. In Lingard's two elephants which were inoculated with blood containing horse surra parasites there was apparently recovery after a short illness, during which no symptoms were noticed.

SURRA IN THE CAMEL.

Evans found surra in the camel at the same time and place as he discovered it in the horse. It is generally considered to be due to the same trypanosome as that causing surra in equines, but this has not yet been satisfactorily proved. Whilst there is a great similarity between cases occurring in the two animals, and while they can be readily inoculated from one animal to the other, there are no doubt great differences in virulence between the parasites from the two animals. As, however, virulence is very variable in different species of animals, and, indeed, in animals of the same species, not much importance can be attached to this. We have noted the difference between the virulence of the disease as it attacks the horse, in which it is fatal, and the Indian ox, in which it is seldom fatal. But there is a further difference in the virulence of the camel parasite in animals to which it is subinoculated, the disease produced in such susceptible animals as horses and dogs being of a comparatively very chronic nature, and running a very long course. The camel is readily susceptible to surra either by natural or artificial inoculation. In India it is the most important and fatal disease attacking the animal, and it has been estimated that 25 per cent. of the camels in the Punjab are suffering from it.

The course of surra in the camel is extremely variable. It may take on either an acute or chronic form. In the former the attacks are sharp, rapidly ending in death; whilst in others, although the paroxysms may at first be frequent (three or more a month), the later ones become less so (one in a month or more), and the disease drags on for a prolonged course, with occasional attacks of fever and presence of parasites, which may continue for over three years, and still prove fatal eventually.

In the acute form the symptoms are more marked, and the disease runs sensibly the same course as it does in the horse, death occurring in from six weeks to a few months. The animal is dull, off its feed, more inclined to lie down, lazy at work. The eye has lost its lustre, and there may be a muco-purulent discharge from it. The temperature* is elevated, and parasites are present in the blood for nearly the whole course of the disease. There is rapid emaciation, œdema at times, the coat is rough and the skin hide-bound, whilst the hump rapidly disappears.

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* In dealing with surra in the camel it is well to know that the normal range of temperature is 5° F., and in the early morning 94° and 95° F. is common, whereas it may be 101° F. in the evening. A temperature of over 99° F. in the early morning often indicates fever, which may rise to 103° or 104° F. later in the day.

In the chronic form, the camel being a very phlegmatic animal, the symptoms are not at all well marked. To the ordinary observer he may appear quite healthy, even when in a paroxysm of fever. The camel-man, however, notices dulness, loss of appetite, and lack of lustre in the eye. The condition may be fair, but not prime. The disease is marked by paroxysms and intermissions, the latter of which lasts for a few days to a month or more, and during this time the animal at first appears healthy and well. Sooner or later, however, the camel begins to steadily lose condition, the hump decreases, and eventually disappears. The animal is hide-bound, and its neck and quarters look scraggy. Later on weakness is evinced by difficulty in rising, accompanied by trembling of the muscles of the hind-quarters.

Œdema is not a constant symptom. In some cases it is marked, being present on the legs, sheath, scrotum, mammary gland, or at the side of the chest. The disease progresses, with at times improvement and again falling off, some cases ending in recovery, but the majority being carried off by pneumonia or œdema of the lungs. Diagnosis is not easy, but in a surra country the blood must be examined in all cases of fever or illness. In surra the parasite will be discovered in the blood during the fever stages, and may also be found at other times. Even by a blood examination the diagnosis is uncertain unless carried out for a considerable time, as in the older cases the intervals between the paroxysms become very long—as much as two months occasionally—and failure to find the parasites on several successive days is no proof that the animal is free. Subinoculation of susceptible test animals with large quantities of blood is easier, but also not absolutely certain.

The course of the disease is influenced greatly by the conditions under which the animals are kept. If hard worked and badly fed, they don't live long, but if carefully treated they may live for a considerable time.

SURRA IN THE RABBIT.

The rabbit is susceptible to the disease by inoculation. The incubation period is not easy to determine accurately, for the reason that there is generally not much rise in temperature, and the parasites are at first scanty and not easy to find in the blood in some cases. The course of the disease is somewhat chronic, but it is invariably fatal. At the beginning of the malady the animal may eat well and put on condition. There may be a slight rise of temperature early in the disease, but exacerbations and remissions are rare, although Fraser's cases had elevated and irregular temperatures. The parasites are seldom numerous in the blood. They increase somewhat after their first appearance, and then gradually disappear, to reappear after an interval of from three or four days. These waves continue throughout the disease; long intermissions lasting from a few days to weeks are noted. Anæmia and emaciation come on rather slowly, but develop to an extreme degree before death. Watery discharges from the nose and eyes appear early, gradually becoming muco-purulent and tenacious, and collecting round the eyes and nose. The hair falls out. Œdema occurs round the base of the ears, the nose, and the abdomen, involving especially the scrotum, which may ulcerate and suppurate. Lameness of the hind-legs is common. Laveran and Mesnil say that the disease usually averages a month, Musgrave and Clegg 15 days to 3 months, Fraser 5 days to 49 days. The disease inoculated from the camel has about the same incubation period. There was occasional high temperature, but this did not coincide with the presence of the organisms in the blood. The organism was at times present in the peripheral blood, but at others could not be found. There was gradual loss of condition, shedding of hair from the ears and around the eyes, œdema of the limbs, ears, and scrotum, and purulent conjunctivitis, with opacity of the cornea. Death occurred in one case on the 154th day, in two others on the 146th day, and in one on the 150th day.

SURRA IN THE GUINEA-PIG.

The guinea-pig is constantly susceptible to the inoculated disease, which is invariably fatal. After a period of incubation lasting from two to fifteen days or more, there is usually a rise of temperature, and the organisms appear in the blood. The incubation period seems generally to be shorter in animals infected with parasites of horse origin. In the parasite transmitted from the camel the incubation period may be considerable, fifteen, nineteen, and twenty days being not uncommon. In India the organisms remain in varying but often considerable numbers in the peripheral blood for long periods at a time, seldom completely disappearing, and then only for a short time, and the guinea-pig is, therefore, a very useful test animal. The general symptoms are slowly evolved. The animal at first appears to gain flesh, but after a time it gradually and progressively becomes thin and anæmic, and is merely skin and bone at death as a rule. Œdema is not a constant symptom, but does occur at times on the genital organs, which may become ulcerated. The fever is atypical and irregular. The disease is invariably fatal, the course varying from 40 days to 4 months. The average period appears to be near 80 days for India, 39 days for Mauritius surra, and in Malaya 79 days.

TREATMENT.

The fight against trypanosomiasis is one of the most interesting problems affecting colonial and imperial expansion. Surra is now recognised to be one of the most serious diseases of the horse and camel. It is spread by an invertebrate host, and the ideal means of combating it would be by destroying this host. With regard to the "Tabanidæ," what we know of the tracts they inhabit and the method of their reproduction leaves little hope of success in attacking them during their larval stage. As to the mature flies, how are we to destroy them?

In a country like India, for instance, where these invertebrate hosts are innumerable, and where reservoirs of infection are plentiful, we fully realise the difficulties of protecting susceptible animals from the bites of these dangerous insects, and this is the case in all surra zones.

Efforts have been made to render animals refractory by means of protective inoculation, and also to render affected animals more or less harmless to others by destroying the parasites in their blood by means of drugs. Rost speaks highly of injections of goat serum in the treatment of surra, but it has been shown that goat serum has no special action on the trypanosomes.

Human serum injected into infected animals has a marked effect upon the course of the disease. The trypanosomes disappear from the blood, sometimes permanently, but generally only for a time. The progress of the disease is delayed, and sometimes even complete cure may result. The serum of adults is best. It retains its properties for some time when collected aseptically and dried.

The serum of none of the lower animals has any such effect.

All attempts at producing an immunising serum have failed, nor has it so far been found possible to attenuate the virulence of parasites. Koch and Schilling have attempted this by passing them through different species of animals, and they claim to have obtained certain favourable results.

None of the methods so far experimented with have met with much success.

Serotherapy has so far given no practical results, and we have found it necessary to have recourse to chemical substances. The previous observations concerning the action of arsenic have formed the basis of all recent work in the direction of prophylaxis, and such progress has been made in the search for a suitable curative medication that many animals have been cured of what was formerly an absolutely fatal disease in the horse.

Although a cure effected by drugs does not leave the animal any immunity against a future attack of the disease, still—in certain parts, at any rate—we are in a very favourable position for the application of curative treatment, because the disease, being transmitted by "Tabanidæ," is of generally seasonal occurrence, and the surra zones where horses and camels become infected are, in many places, limited in extent. Once we can pull a horse through the dangerous time we have achieved a great deal. In many cases, too, we can remove the horse to a healthy locality, and thus avoid reinfection. We appear to be in sight of a curative treatment which may succeed in a certain percentage of cases in certain outbreaks, and will be cheap enough to be practicable. There seem to be some grounds for hoping that we may soon be in possession of a method of prevention of the spread of the disease founded on the principle of destroying the parasites in the blood of the infected animal, and so allowing the latter to be taken through a surra zone at the dangerous time of the year without spreading the disease. It may be hoped also that the application of drugs which manifest a very marked preventive power may render it safe to take susceptible animals through a surra zone during the season when "Tabanidæ" are present. Curative treatment in the horse has made immense strides in recent years.

The great difficulty to be contended against is that the virulence of the trypanosomes in various and unknown conditions is so varied. We are not in a position to decide the causes of this great variation in virulence. There are, in fact, an infinite number of races of the trypanosome, and, so far as we know at present, these do not possess the same sensibility to any one medicament. This well-known fact obviously complicates the question, and explains the difference in the results obtained with the same drug in the various recorded observations of the same disease. There is another point which is to be remembered, and that is the fact that the trypanosomes rapidly become resistant to various drugs, and especially to arsenical preparations.

Two points are therefore of paramount importance in attempts at treatment: one to hit hard, so as to destroy, if possible, the trypanosomes before they can become accustomed and resistant to the drug, and the other to associate various known trypanocides in the treatment.

In 1899 Lingard tried a great number of chemical and other substances for the treatment of surra in horses, but the only one with which he had any success at all was arsenic, with which he succeeded in curing one horse. The method, however, is not a practicable one, and only very occasionally successful, as it does not hit hard enough, and the trypanosomes become resistant. Later, Bruce showed the favourable results obtained by the arsenical treatment of horses infected with surra and nagana; and Laveran and Mesnil subsequently entered upon the study of the action of arsenical preparations. They observed that by repeated injections it was possible to considerably prolong the life of animals, but that they could not save their lives. During the epizoöty at Mauritius a great number of medicaments were used without success—quinine, arsenious acid, Fowler's solution, cacodylate of soda, arrhenal, and intravenous injection of corrosive sublimate. Ehrlich and Shiga made a great stride in the treatment of surra by the discovery of trypanroth, but it was not, unfortunately, efficacious in the larger animals. Methyl and ethyl green was recommended by Wendelstadt and Felmer, but was useless. One of the greatest discoveries so far was made by Thomas, who introduced atoxyl, an arsenical derivative forty times less poisonous than arsenic, and containing 24 per cent. of it, into the treatment. By repeated administration it has saved a certain proportion of animals, but it is not satisfactory alone. The impossibility of obtaining certain cure by any of the medicaments used singly, owing to the parasites acquiring immunity to the drug, could but lead to the combinations of two or more of them. Laveran did this a short time after the discovery of trypanroth by injecting this agent the day after arsenic, and repeating more or less frequently the administration of the two active drugs, and he cured some animals in this way.

Thomas later substituted atoxyl for arsenic with good result, and Mesnil and Nicolle tried benzidine colours with some success.

Atoxyl and perchloride of mercury were also recommended, but did not prove suitable in the treatment of surra.

Laveran and Thiroux obtained very promising results in some forms of trypanosomiasis by the use of atoxyl and precipitated orpiment. This has, however, proved ineffective in surra of the horse. Gaiger has, however, obtained cures by the use of atoxyl and ordinary orpiment, which contains a high percentage of free arsenic. Holmes at Muktesar cured four ponies out of seven by the latter method, giving the drug alternately, with an interval of one day between each dose.

The association of atoxyl and tartar emetic has been advocated, as well as that of atoxyl and a mercurial salt.

Löffler and Ruch advise the use of atoxyl with their arsenious acid solution. Holmes has tested the value of atoxyl, arsenious acid, soamin, mercury and antimony salts, alone and in various combinations, as curative agents in the treatment of surra in horses.

The best results which have so far been obtained in India have been due to a combination of atoxyl with inorganic arsenic in some form. A dose of atoxyl has been first employed to clear the blood of trypanosomes, and then the largest subtoxic doses of arsenic, in solid or fluid form, are given alternately. More recently Holmes, I.C.V.D., has secured excellent results at the Muktesar Laboratory by the use of atoxyl and arsenic in various forms, as well as by the use of arsenic alone in large doses. The following is the method he recommends :

1. A dose of atoxyl is first given subcutaneously. The atoxyl by itself has no curative effect, but exercises a rapid action in clearing the circulation of mature trypanosomes. It is, therefore, used only when trypanosomes are present in the circulation. The use is indicated at the commencement of treatment. If the first dose does not result in the complete disappearance of trypanosomes from the blood on the following day, a second injection of atoxyl should be given twenty-four hours after the first dose. Atoxyl should be kept in the dark. The atoxyl and arsenic should be given after feeding.

2. The injection of atoxyl has to be followed by ten subtonic doses of arsenious acid in ball. Care must be taken in making up the balls and in mixing the drug with other ingredients.

The doses have been determined for horses according to their weight (see p. 1079).

An interval of one day is allowed between each dose.

The amount of arsenious acid is gradually increased. If after any dose the animal is dull or off feed, the next dose must be suspended till the symptoms have passed off. If after any dose the animal is off feed and uneasy, and showing symptoms of colic, it shows that he cannot tolerate the amount of arsenious acid in that dose, and treatment must be suspended till he has recovered, and the next lower dose used, and no further increase in the following doses is to be made. Animals showing symptoms of colic are to be treated with opium, chlorodyne, or other sedative.

Should trypanosomes reappear in the blood during treatment with arsenic, atoxyl should again be used to clear the circulation of the parasites, after which the arsenic is to be continued.

3. Daily examination of the blood should be made, and the result recorded with the daily temperature in the observation charts. After the completion of the treatment the animal should be kept under observation, the blood being examined daily (or at least twice a week) for a period of two months.

If a relapse occurs after the first course of treatment, a second similar course should be given, with slightly increased doses of arsenic.

4. Throughout the treatment the animal should receive a liberal diet and walking exercise.

This treatment in Holmes' hands has given 75 per cent. of recoveries.

5. If the animal is in a very advanced stage of surra and in weak condition, it should be treated with atoxyl injection alone at intervals of three to four days, and receive care and full diet until it has sufficiently recovered to stand the doses of arsenic.

This treatment is worth a trial in various parts, but, for the reasons already given, it cannot be expected that it will invariably give as good results as Holmes obtained. It has not been found very successful in Mauritius, for example, nor in some of the plains stations in India. I am personally in favour of a more mixed treatment. In regard to the treatment of other animals, the results so far obtained with the camel and dog are negative. The elephant apparently recovers with care and arsenical treatment. The ox generally recovers spontaneously in enzoötic areas, and the other animals do not pay for the cost of treatment.

When considering the advisability of adopting curative treatment in this disease, it must be remembered that, unless proper means of preventing its spread by means of flies from the sick to healthy animals are available, it is far better to destroy the sick animal at once and dispose of the carcass. If dark or fly-proof stables are available, there is little danger in keeping the sick animal alive. If it can be removed from the area in which the flies are prevalent, or if the fly season be past, then there is no danger.

PROPHYLAXIS.

In view of the cause of the spread of this disease, it will be evident that arrangements for its prevention in known surra areas during the fly season, if possible, would be impracticable on account of cost. We cannot hope to destroy the flies, and to prevent them biting is also next to impossible.

Spraying animals which have to pass through known zones would, no doubt, be fairly efficient.

The removal of the reservoir of the virus would doubtless have a good effect if it were possible. We can at any rate destroy all animals bearing the trypanosome in some countries, although in others—India, for instance—this is out of the question.

The only safe thing is to weed out so far as possible all reservoirs of the virus, and to ascertain and avoid known surra zones in the dangerous season; and if it be necessary to pass through them, to do so at night, or to spray animals before doing so. Grazing animals in such areas should, if possible, be done at night. Horses and mules, etc., should be protected so far as possible by burning smoky wood fires, and all known reservoirs of the virus should be kept at a distance from susceptible animals. Horses and mules should not graze or be stabled near buffaloes, camels, and oxen. Government clean camels should not come into contact with privately owned ones, which may be infected.

Countries free from the scourge should prohibit importation from infected areas.

V. TRYPANOSOMIASIS OF BIRDS AND FISHES.

By H. GRAY, M.R.C.V.S.

TRYPANOSOMIASIS OF BIRDS.

Birds are infested with different varieties of trypanosomes, and since the researches of Danilewsky on this subject were published in 1888 a great deal of attention has been given to their trypanosomes, which have been found in nearly every species of bird.

The following table gives the species of trypanosome and its bird host:

Parasite.	Host.
Trypanosoma avium.	Red-winged blackbird (Agelæus phœniceus), Colaptus auratus; Roller (Coracias garrula), Blue jay (Cynocitta cristata), Baltimore oriole (Icterus galbula), Song sparrow (Melospiza fasciata); American robin (Merula migratoria), Common sparrow (Passer domesticus), Bluebird (Sialia sialis), Tawny owl (Syrnium aluco), Mourning-dove (Zenaidura macrura).
T. Hannæ.	Indian pigeon (Columba *sp.*).
T. Johnstoni.	St. Helena waxbill (Estrelda estrelda).
T. Laverani.	American goldfish (Spinus tristis).
T. Mathisi.	Swallow (Hirundo rustica).
T. Mesnili.	Red-shouldered hawk (Buteo lineatus).
T. noctuæ.	Little owl (Athene noctua).
T. paddæ.	Java sparrow (Padda oryzivora).
T. polyplectri.	Germain's peacock-pheasant (Polyplectron germaini).
Trypanosomes of various unnamed species have been found in—	Cameroon kingfisher (Alcyon *sp.*); Hawk (Asturinula mongrammaica), Indian owl (Athene brama), Trumpeter Hornbill (Bycanistes buccinator), House-martin (Cheledon urbica), Indian crow or raven (Corvus *sp.*), Crane, A "millet-eater" (Chrithagra *sp.*), Hairy woodpecker (Dryobates villosus), Egret, Yellow-hammer (Emberiza citrinella), Goldfinch (Carduelis carduelis), Chaffinch (Fringella cœlebs), Goatsucker, Brown thrasher (Harporhynchus rufus), Swallow (Hirundo rustica), African shrike (Laniarius cruentus), Redpoll (Acanthus rufescens), Blackbird (Merula merula), Indian kite (Milvus govinda), African vulture (Neophron percnopterus), Nicticorax gardenia, Rusty blackbird (Scolephagus carolinus), Blackcap warbler (Sylvia atricapilla), Tachyphormus ornata, Angola dove (Treron calva), Song-thrush (Turdus musicus), House-wren (Troglodytes aedon).

The *Trypanosoma* (*Trypanomorpha*) *noctuæ* of the little owl (*Athene noctua*) has its alternate host in a gnat (*Culex pipiens*), in which a complex part of the life-cycle takes place. This gnat can only transmit infection after a certain interval has elapsed since the meal when it became itself infected. Towards the end of digestion of the ingested blood, this parasite, after a period of multiplication, enters upon a resting phase. Following a second meal, another multiplicative period occurs, and the trypanosomes gradually collect in the anterior part of the stomach, where the nutriment remains longest unabsorbed. In this situation the parasites commence to agglomerate in large numbers, being able to penetrate the delicate surface of the layer of invaginated œsophageal epithelium in this region. Ultimately there is an enormous accumulation of the trypanosomes at this place, all arranged in rows and in a resting-place. The next inflow of blood drives this mass before it, in the form of a rolled-up ball, until it reaches the junction of the ileum and colon, the narrowest point of the intestine, at which point the wall is very thin and readily ruptured. Through this the majority of the trypanosomes pass, gain the vacular lacunæ, and are carried to the heart. Finally, the parasites become arrested in the sinus surrounding the pumping-organ of the pharynx, where they continue to multiply and collect again into clusters, which press on the wall of the pharynx in this region. By the time the third digestive period is reached, these clusters of trypanosomes have broken through, and partly block up the lumen; and in the next biting act they are forcibly injected into the blood of the owl. Thus, the parasites cannot leave the gnat until the fourth meal, including that which effected their entry, has taken place. Schaudinn, who worked out this life-cycle, found that the shortest time elapsing between entrance and exit was seven or eight days, when the insects were maintained at the optimum temperature for digestion. Some of the trypanosomes—usually females—after breaking through the wall of the colon, may pass into the ovarian follicles, penetrate into the young eggs, and so infect a succeeding generation—true hereditary infection (Woodcock).

In certain phases this trypanosome in the owl's circulation becomes attached to the red corpuscles, while at other times it penetrates into the corpuscle, and eventually destroys it.

Petrie at Elstree found trypanosomes in several species of British birds. Examination of the blood gave negative results, but the parasites were found in the bone-marrow. In a house-martin (*Chelidon urbica*) spirochætes were present in the blood.

Java sparrows are rarely free from the *Trypanosoma paddæ*. Young rollers, even when only three or four days old, contain numerous parasites (*T. avium* or *T. Danilewskyi*) in the bone-marrow. At times they are more numerous in the blood. In the St. Helena waxbill (*Estrelda estrelda*) *T. Johnstoni* has been found.

Infection from one Java sparrow to another is possible by subcutaneous, intramuscular, intravenous, or intraperitoneal inoculation of infective blood. The period of incubation by this last method varied from twelve hours to eighteen days. After subcutaneous inoculation it was never less than twelve days. In some of the birds infection remained latent for two or three months, trypanosomes being only very rarely seen in the blood. After severe infection of long duration the spleen is often enlarged considerably. In acute infection the number of parasites may equal that of the red corpuscles, in which case the bird usually succumbs.

Ravitch, Eisenman, and Purdey, of Kentucky, U.S.A., suggest that pellagra in man, and a pellagra-like disease in horses and cattle, might be due to a trypanosome introduced by birds migrating from South America. This disease in horses and cattle resembles in clinical appearance *mal de caderas*, due to *T. equinum*. In the incriminated birds, migrating from the South in the spring and leaving again in the autumn, Novy and McNeal have found trypanosomes. The chief species of bird are blackbirds, which are alleged to be the reservoirs of the virus. This view, however, is problematical.

There is no evidence that the presence of trypanosomes in birds that have become infected by natural means produces clinical symptoms, and brings about death. It is, however, quite possible for disease to be brought about, since experimental infection in certain cases ends in death. As the mortality of foreign birds when introduced into this country is very great, it would be well if the cause of such mortality were inquired into.

A piroplasm—*Piroplasma avicularis*—was described by Wenyon in 1909 as occurring in the bird.

TRYPANOSOMIASIS OF FISHES.

Fishes, as well as reptiles and amphibia and other vertebrates, are often the hosts of various species of trypanosomes. The trypanosomes of fishes belong to two genera—Trypanosoma and Trypanoplasma. This latter genus is, according to Laveran and Mesnil, characterised by the following:

Flagellates with elongated body, presenting laterally an undulating membrane, whose thickened edge is prolonged posteriorly as a flagellum, and turns back anteriorly to reach a mass (centrosome), which is as large as, and has, up to a certain point, the same structure as, the nucleus. An anterior free flagellum has its origin in the same mass. Multiplication is probably by binary equal longitudinal division.

GENUS TRYPANOSOMA.

Hæmoflagellate Parasite.	Host.
Trypanosoma abramis.	Abramis brama (Bream).
T. acerinæ.	Acerina cernua (Pope).
T. barbatulæ.	Cobitis barbatula (Loach).
T. barbi.	Barbus fluviatilis (Barbel).
T. bothi.	Bothus rhombus (Rhombus lævis) (Brill).
T. callionymi.	Callionymus dracunculus.
T. carassi.	Carassius vulgaris (Crucian carp).
T. clariæ.	Clarias (Silurus) clarias.
T. cobitis.	Cobitis fossilis.
T. cotti.	Cottus bubalis.
T. Danilewskyi.	Carassius auratus (Goldfish), Cyprinus carpio (Carp), Phoxinus lævis (Minnow).
T. delagei.	Blennius pholis (Blenny).
T. elegans.	Gobio fluviatilis (Gudgeon).
T. flesi.	Pleuronectes flesus (Flesus vulgaris) (Flounder).
T. gobii.	Gobio niger (Goby).
T. granulosum.	Anguilla vulgaris (Eel).
T. langeroni.	Cottus gobio (Bull-head).
T. laternæ.	Platophys laternæ.
T. leucisci.	Leuciscus *sp.*
T. limandæ.	Limanda platessoides.
T. macrodonis.	Macroden malabaricus.
T. percæ.	Perca fluviatilis (Perch).
T. phoxini.	Phoxinus lævis (Minnow).
T. platessæ.	Pleuronectes platessa (Platessu vulgaris) (Plaice).
T. raiæ.	Raia clavata, R. mucrorhynchus, R. mosaica, and R. punctata (Rays).
T. Remaki.	Esox lucius (Pike).
T. rhamdiæ.	Rhamdia queler.
T. saccobranchi.	Saccobranchus fossilis.
T. scardinii.	Scardinius (Leuciscus) erythrophthalmus (Rudd).
T. scyllii.	Scyllium canicula, S. stellare (Dogfish).
T. soleæ.	Solea vulgaris (Sole).
T. squalii.	Leuciscus (Squalius) cephalus (Chub).
T. tincæ.	Tinca tinca (Tench).
Various undetermined species of Trypanosomes have been found in—	Bageus bayard (Bagara), Barbus carnaticus, Clarias angolensis, Gobio giuris, Leuciscus idus, L. cephalus and Rutilus rutilus (Roach), Lota vulgaris, Macrones seenghala, M. tengara, M. cavasius, Mugil *sp.*, Ophiocephalus striatus, Polypterus *sp.*, Raia microcellata, Silurus glanis, Synodontis schal, Trichogaster, fasciatus, etc.

GENUS TRYPANOPLASMA.

Hæmoflagellate Parasite.	Host.
Trypanoplasma abramidis.	Abrama brama (Bream).
T. barbi.	Barbus fluviatilis (Barbel).
T. Borelli.	Leuciscus (Scardinius) erythrophthalmus (Rudd or red eye), Phoxinus lævis (Minnow).
T. cyprini.	Cyprinus carpio (Carp).
T. guernei.	Cottus gobio (Bull-head).
T.(?) intestinalis.	Box boops.
T. truttæ.	Salmo fario (Trout).
T. varium.	Cobitis barbatula (Loach).
T. (?) ventriculi.	Cyclopterus lumpus (Lump-fish).
Other Trypanoplasma of which the species yet remains undetermined are found in—	Acerina cernua, Esox lucius (Pike), Leuciscus idus, L. cephalus and Rutilus rutilus (Roach), Lota vulgaris, Perca fluviatilis (Perch), Tinca tinca (Tench), etc.

Trypanosomata and Trypanoplasmata are conveyed from one fish to another by leeches, which attach themselves either to the gills of the fish or to the surface of the body between the scales. They may also be conveyed from one fish to another of the same species by experimental inoculation. It is said that the trypanosomata of fresh-water fishes undergo their evolution in leeches of the *genus* Hemiclepsis ; and that of the trypanoplasmata according to the species of trypanoplasm in *Hemiclepsis,* or in *Piscicola.*

The following table gives the species of leech (*Hirudinea*) and the species of trypanosome or trypanoplasm which has its habitat in it :

Leech Host.	Parasite.
Calobdella punctata.	Trypanosoma cotti and T. soleæ.
Helobdella algira.	T. inspinatum.
Hemiclepsis marginata.	Trypanoplasma varium.
Hemiclepsis of undetermined species.	Trypanosoma abramis, T. acerniæ, T. barbi, T. Danilewskyi, T. granulosum, T. percæ, T. phoxini, T. Remaki T. squali, and probably also T. barbatulæ, T. langeroni, T. leucisci, T. scardinii, and Trypanoplasma abramidis.
Piscicola species.	T. barbutulæ, Trypanoplasma Borreli, T. barbi, T. Guernei, T. Truttæ.
Piscicola geometra.	Trypanoplasma Borreli, etc.
Pontobdella muricata.	Trypanosoma raiæ.
Pentobdella *sp.*	Trypanosoma scyllii.

Symptoms.—The most appreciable sign of intense multiplication of the parasites is severe swelling of the saddle region of the body backwards. After a time the gills become pale, the fish does not move, refuses all food, and finally dies. According to Hofer, *Trypanoplasma cyprini,* which has been obtained by Miss Marianne Plehn from the blood of carp coming from various places, caused severe enzoötics in carp in 1900-1902.

According to Laveran and Mesnil, the blood for examination can easily be obtained from a fish by incising two or three rays at the base of the caudal fin. An ordinary fresh preparation is made at once and examined for trypanosomes.

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SOME PROTOZOAL DISEASES OF BIRDS, FISHES, ETC.

By H. GRAY, M.R.C.V.S.

I. HÆMATOZIASIS OF BIRDS.

NUMEROUS species of birds, not only in Great Britain, but elsewhere, harbour in their blood trypanosomes. But as this subject has been mentioned (see p. 1113), the writer will not discuss it beyond saying here that another class of blood-parasite, *Hæmamœba*, is frequently associated with it in the same bird, which may also further be infested with blood filaria, so that three infections may coexist at the same time. Schaudinn, however, believes that the Hæmamœba is only a stage in the life-history of trypanosomes; but this question must remain in abeyance until further evidence is brought forward to prove or disprove Schaudinn's view.

Hæmamœba have been found in the jay, magpie, chough, crow, hawk, brown owl, common owl, pigeon, chaffinch, lark, Java sparrow, whistling teal, comb duck, little bittern, the Hungarian partridge, turkey, etc.

They have been described under various names, such as Halteridium, Proteosoma, Laverania, Plasmodium, Globidium, Hæmoproteidium, and Hæmamœba. According to Laveran, they should be included under the genus Hæmamœba.

The principal species are as follows: *Hæmamœba relicta* (*Plasmodium relictum, Proteosoma* of Labbé), *Hæmamœba Danilewskyi* (*Halteridium Danilewskyi, Plasmodium Danilewskyi*), *Hæmamœba Ziemanni* (*Plasmodium Ziemanni*), *Hæmamœba majoris*.

These parasites are generally found grouped on the surface, or included in the interior of the blood-corpuscles, and mostly appear as rounded, and occasionally as ovoid, bodies. By their presence the blood-corpuscle is deformed, and then gradually destroyed, when they are set free. In their adult stage they take on two distinct forms: (1) *In the form of finely granular elements,* coloured well by methylene blue, they show disseminated granules of pigment. By Laveran's modified method of Giemsa, their nucleus appears regularly circular or oval in outline, situated in the centre of the parasite, and containing a small karyosome. The nucleus appears violet and the karyosome darker, and are the *female elements.* (2) *In the form of hyaline elements,* containing large granules of pigment, extending to their extremities, not staining well by methylene blue, possessing a very large elongated nucleus having an irregular contour, and occupying the whole of the central part of the parasite. After these parasites have escaped from the blood-corpuscles they take on a rounded form, and become furnished with flagella, and constitute *the male elements.*

The flagella are four to six in number for each element, and show a swelling, which varies in position and form, and in the centre of which a mass of chromatin is found. When the parasites have become free, the flagella enter, penetrate, and fecundate the female elements.

The marguerite or segmented bodies are rarely encountered, and Laveran has never witnessed them in *Hæmamœba Danilewskyi.* In the *H. relicta,* which is a very common parasite in sparrows in the Roman Campagna, the marguerite body is found. This parasite undergoes its transformations in the *Culex pipiens,* which transmits by its punctures the parasite.

In the Partridge.

In the blood of the Hungarian grey partridge (*Perdix cinerea*) dead of some emaciating disease Laveran and Lucet found the *H. relicta* in the centre of the blood-corpuscles. Of 100 of these birds in 1905 introduced into France from Hungary, on arrival appearing in the best of health, shortly began to fall ill, and 97 succumbing within a few days to a week or two. The symptoms were emaciation, and the anal, or rather cloacal, feathers were matted with diarrhœa.

On post-mortem examination the spleen was found very much enlarged, and on microscopical examination of the blood the red corpuscles contained numerous hæmatozoa.

A very large number of Hungarian partridges for stocking purposes are every year introduced by landed proprietors into Great Britain; but it is not known if the mortality soon after arrival is extensive.

In the Turkey.

Lucet encountered a great mortality from *crise du rouge* (dysentery) in young turkeys bred and reared in Courtenay in the Loiret district. He found on post-mortem examination perityphylo-hepatitis. In scrapings from the liver, small oval, more or less elongated parasites, measuring from 14 to 25 μ in length by 5 to 8 μ in width were discovered. Their protoplasm contained fine granules of black pigment. They were found in the leucocyte, of which the nucleus was generally divided into two equal parts, placed on either side of the parasite, which had already been observed by Theobald Smith, who at the time had not recognised its pathogenic rôle. Laveran and Lucet gave it the name of *Hæmamœba Smithi,* but eventually Smith rechristened it *Amœba meleagrides* (*vide* Coccidiosis, or Black-head of the Turkey, p. 1181).

Although it is not usually recognised if the hæmatozoa of birds have any pathogenic action on birds, Danilewsky has observed that at certain times the birds fall ill, and even succumb. In this latter case the parasite is found in marguerite form in the blood.

From a scientific as well as a practical point of view, it would be interesting to learn if these parasites are responsible for the large mortality in foreign birds introduced into this country by dealers for the cage and aviary.

Quinine, although active against the hæmatozoa of human malaria, is without effect on the hæmatozoa of birds.

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II. THE MYXOSPORIDIA (PHÆNOCYSTES).

The Myxosporidia form a very large order of protozoal parasites infesting fishes, reptiles, amphibia. This order is included in the class Neosporidia of the subphylum Sporozoa.

The *Sporozoa* are parasitic protozoa without motile organs, but capable of moving from place to place by structural modifications of various kinds. Their reproduction is either simple or multiple, but mainly by spore formation, which is either asexual (schizogony) or sexual (sporogony).

The *Neosporidia* are that class of sporozoa in which the sporulation of the amœboid parasites takes place during the activity of the parent cell, and without interfering with the vegetative processes. These parasites are either cælozoic, histozoic, or cytozoic in their situation, and are found chiefly in fish, reptiles, amphibia, arthropods, etc.

The *Myxosporidia* are comparatively large Neosporidia, and reproduced by pansporoblast formation. Their spores are provided with polar capsules containing *more or less easily seen filaments.*

This order is divided into two suborders—the *Disporea* and the *Polysporea.*

The *Disporea* have one pansporoblast containing two spores, produced by each trophozoite. The spores are greater in width than length, and the trophozoites are found floating freely in the fluids of various organs of their hosts. They form one family, *Ceratomyxidæ*, which has all the characters of the suborder. The two genera of this family are *Ceratomyxa* and *Leptotheca*.

The *Ceratomyxa* have the two valves of the spore produced into long attenuated processes. The following table gives approximately the species, their habitat, and their fish host only:

Name of Sporozoön.	Habitat.	Species of Fish infested.
C. appendiculata.	Gall-bladder.	Lopheus budegassa.
C. arcuata.	Gall-bladder.	Crenilabrus melops, Pagellus centrodontus, Motella tricirrata.
C. arcuata, var. scorpænarum.	Gall-bladder.	Scorpæna porcus and S. scrofa.
C. arcuata, var. typica.	Gall-bladder.	Pagellus centrodontus.
C. inæqualis.	Gall-bladder.	Crenilabrus mediterraneus and C. pavo.
C. linosporo.	Gall-bladder.	Labrus turdus.
C. pallida.	Gall-bladder.	Box boops, B. salpa.
C. reticularis.	Gall-bladder.	Trachinus draco.
C. spærulosa.	Gall-bladder.	Galeus galeus, Mustelus canis.
C. truncata.	Gall-bladder.	Clupea pilchardus.

The *Leptotheca* have the valves of the spore not drawn out into long filaments. The sporoplasm completely fills the spore membranes. The species are approximately given in the following table:

Name of Sporozoön.	Habitat.	Fish Host.
L. agilis.	Gall-bladder.	Scorpæna *sp.* and Trygon pastinaca or T. vulgaris.
L. elongata.	Gall-bladder.	Merluccius merluccius or M. vulgaris.
L. hepseti.	Gall-bladder.	Atherina hepsetus.
L. parva.	Gall-bladder.	Scomber scombrus.
L. perlata.	Muscles.	Acerina cernua.
L. polymorpha.	Gall-bladder.	Phycis phycis or Merluccius vulgaris.
L. renicola.	Kidney tubules.	Scomber scombrus.

The suborder *Polysporea* has more than two spores, usually a great number, produced in each pansporoblast. The spores are greater in length than width. It is divided into three families—*Myxidiidæ*, *Chloromyxidæ*, and *Myxobolidæ*.

The family *Myxidiidæ* has the trophozoites typically free-living as parasites in the fluids of the internal organs of their hosts. The spore has two polar capsules. It includes six genera—*Spærosphora*, *Myxidium*, *Spæromyxa*, *Cystodiscus*, *Myxosoma*, and *Myxoproteus*.

The genus *Spærosphora* has spherical spores. The following are the species usually encountered:

Sporozoön.	Habitat.	Fish Host.
S. elegans.	Kidney tubules and connective tissue of ovary.	Gasterosteus aculeatus, G. pungitius, Lota vulgaris, and Phoxinus lævis.
S. divergens.	Kidney tubules.	Blennius pholis and Crenilabrus melops.
S. Masovici.	Gall-bladder.	Abramis brama.
S. nostrata.	Glomeruli of kidney.	Mugil *sp.*

The genus *Myxidium* has navicular spores, with polar capsules at each end. The following table gives most, if not all, of the known species:

Sporozoön.	Habitat.	Fish Host.
M. giganteum.	Gall-bladder.	Raia alba.
M. histophilum.	Kidney and ovary.	Leuciscus phoxinus.
M. incurvatum.	Gall-bladder.	Blennius pholis, Callionymus lyra, Nerophis æquoreus, Scurpæna scrofu, Syngnathus acus.
M. Lieberkühnii.	Urinary bladder.	Esox lucius, Lota lota or L. vulgaris.
M. sphæricum.	Gall-bladder.	Belone acus, and B. belone or B. vulgaris.

The genus *Spœromyxa* has navicular-shaped spores, with truncated ends, and a polar filament at each extremity. The polar filaments are short and thick, and somewhat conical in form.

Sporozoön.	Habitat.	Fish Host.
S. Balbianii.	Gall-bladder.	Cepola rubescens, Motella maculata.
S. incurvata.	Gall-bladder.	Blennius ocellatus.
S. Sabrazèsi.	Bile-ducts.	Hippocampus brevirostis.

The genus *Cystodiscus* has trophozoites, without amœboid movements or changes of form. The spores are symmetrical, with the sutural plane running obliquely from one extremity to the other, and with a polar capsule at the extremities of the oblique suture. As far as is known, there is only one species—*C. immersus*—and has been found inhabiting the gall-bladder of toads and Cystignathus in Brazil.

The genus *Myxosoma* has flattened spores, which are ovoid in form, and has polar capsules crowded together at the narrow extremity.

Sporozoön.	Habitat.	Fish Host.
M. Dujardini.	Gills.	Rutilus rutilus, Scardinius erythrophthalmus.

The genus *Myxoproteus* has somewhat pyramidal spores, with spinous processes from the base of the pyramid.

Sporozoön.	Habitat.	Fish Host.
M. ambiguus.	Urinary bladder.	Lophius piscatorius.

The family *Chloromyxidœ* is characterised by the spores having four capsules. It includes only one genus—*Chloromyxum*.

The genus *Chloromyxum* has all the characters of the family.

Sporozoön.	Habitat.	Fish Host.
C. cristatum.	Gall-bladder.	Tinca tinca or T. vulgaris.
C. fluviatile.	Gall-bladder.	Leuciscus cephalus.
C. Leydigi.	Gall-bladder.	Acanthius acanthius or A. vulgaris, Raia batis, R. clavata, R. undulata, Rhina squatina, Scyllium canicula, Spinax spinax or S. vulgaris, Torpedo narce, T. torpedo or T. marmorala, Trygon pastinacea or T. vulgaris.
C. quadratum.	Muscles.	Nerophis æquoreus, Callionymus lyra, Coris julis, Syngnathus acus, Trachurus trachurus.
C. truttæ.	Gall-bladder and bile-ducts.	Salmo *sp.*
C. mucronatum.	Urinary bladder.	Lota vulgaris.

The family *Myxobolidæ* are typical histozoic parasites rarely found in an amœboid form, but usually as cysts filled with spores. They are generally polysporous, and the spores have one or two polar capsules. The sporoplasm contains vacuoles, which are stained reddish-brown by iodine. It contains three genera—*Myxobolus, Henneguya,* and *Hoferella,* or *Hoferellus.*

The genus *Myxobolus* has ovoid spores, which, when flattened, form an ellipse, and have single or double polar capsules. The spore membrane is without a tail-like process.

Sporozoön.	Habitat.	Fish Host.
M. chrondrophagus (M. cerebralis).	Gills, ears, fins, and cartilage of skeleton.	Salmo irideus.
M. cycloides.	Operculum and pseudo-branch.	Rutilus rutilus.
M. cyprini.	Kidney tubules and gills.	Cyprinus carpio, Tinca vulgaris, Abramis brama.
M. dipleureus.	Kidney.	Lota lota or L. vulgaris.
M. dispar.	Kidney tubules, muscles, spleen, gills, skin, and connective tissue.	Cyprinus carpio, Scardinius erythrophthalmus, Alburnus lucidus.
M. ellipsoides.	Swim-bladder, gills, kidney, spleen, liver, cornea.	Tinca tinca or T. vulgaris.
M. exiguis.	Gills, stomach, pyloric cæca, spleen, kidney.	Abramis brama, Mugil auratus, M. capito, M. chelo.
M. globosus.	Gills.	Erimyzon succetta.
M. inæquales.	Gills.	Piramutana blochi.
M. Lintoni.	Subcutaneous connective tissue.	Cyprinodon variegatus.
M. merlucci.	—	Merluccius merluccius or M. vulgaris.
M. Mülleri.	Fins, gills, eye, ovary, and kidney.	Leuciscus cephalus, Barbus barbus or B. fluviatilis, Crenilabrus mediterraneus, C. pavo, Phoxinus lævis.

Sporozoön.	Habitat.	Fish Host.
M. obesus.	Fins.	Alburnus alburnus.
M. oblongus.	Skin.	Erismyzoii succetta.
M. oviformis.	Fins, gills, kidneys, spleen.	Alburnus alburnus, Gobio gobio or G. fluviatilis, Lota vulgaris.
M. Pfeifferi.	Spleen, kidney, intestine, ovary, neurilemma (?).	Barbus barbus, Thymallus thymallus or T. vulgaris.
M. piriformis.	Gills, spleen, kidneys.	Tinca tinca, Cobitis fossilis.
M. pisiformis.	Kidney.	Misgurnus fossilis.
M. sphæralis.	Gill epithelium.	Coregonus lavaretus or C. fera.
M. *sp.* (Gurley).	Body cavity.	Carassius carassius.
M. *sp.* (Müller, J.).	Gill epithelium.	Lucioperca lucioperca or L. sandra.
M. textus.	Gills.	Perca fluviatilis.
M. transversalis.	Scales.	Leuciscus funduloides.
M. unicapsulatus.	—	Labeo riloticus.
M. Zschokkei.	Intermuscular tissue.	Coregonus lavaretus.

The genus *Henneguya* has ovoid spores, with two polar capsules, the sporocyst being prolonged into two long caudal processes, which are not penetrated by the sporoplasm.

Sporozoön.	Habitat.	Fish Host.
H. brevis.	Kidney tubules and ovary.	Gasterosteus aculeatus and G. pungitius.
H. Creplini.	Muscles.	Acerina cernua.
H. Kowlesnikovi.	Intermuscular tissue.	Coregonus lavaretus or C. ferax.
H. linearis.	Gills, gill-chambers, and base of dorsal fins.	Amiurus melas, Pinnelodus sebæ, Platystoma fasciatuni.
H. macrura.	Connective tissue of lower jaw.	Hybognathus nuchalis.
H. media.	Kidney tubules and ovary.	Gasterosteus aculeatus and G. pungitius.
H. monura.	Muscles.	Aphredoderus sayanus.
H. psorospermica.	Gills, muscles, and eye.	Perca fluviatilis, Esox lucius.
H. psorospermica anura.	Gills.	Esox lucius.
H. psorospermica lobosa.	Gills.	Esox lucius.
H. psorospermica oviperda.	Eggs.	Esox lucius.
H. schizura.	Intracellular tissue of eye muscles, etc.	Esox lucius.
H. strongylura.	Head.	Synodontis schall.
H. *sp.* (Claparéde).	Gills.	Coregonus lavaretus or C. fera.
H. *sp.* (Borne).	Gills.	Rutilus rutilus.
H. tenuis.	Gut.	Acerina cernua.

The genus *Hoferellus, Hoferia,* or *Hoferella,* has broad and compressed spores, with two tail-like processes at the posterior end.

Sporozoön.	Habitat.	Fish Host.
Hoferellus cyprini (commonly associated with Myxobolus cyprini).	Kidney tubules and gills.	Cyprinus carpio.

Myxosporidiosis of Fishes.

Synonyms.—Psporospermosis of fishes; Myxoboliasis; Myxidiasis; Small-pox of fishes; Protozoal gid in rainbow trout; Protozoal jaundice in trout; Myxoboliasis tuberosa.

Definition.—Myxosporidiosis is a disease in which various organs of fishes become invaded and infested by protozoal parasites belonging to the *order* Myxosporidia of the *class* Sporozoa. These parasites, included in the *families* Myxidiidæ, Chloromyxididæ, and Myxobolidæ, often give rise to serious disturbance of health, and not rarely cause a terrible mortality in the affected fish. They may, however, like nearly all other parasitic infestations, be present without producing palpable disease. Amphibia, reptilia, etc., may also be affected by their presence.

History and Geographical Distribution.—The presence of Myxosporidia in fishes has for a long time attracted attention, but no serious study was given to it until 1838, when Gluge, of Brussels, published his observations. In 1841 Johannes Müller recorded several observations on the same subject. In 1842 Creplin published further researches. In 1843 P. Rayer, a distinguished comparative pathologist, reported his observations on the diseases of fishes caused by them. In 1845 F. Dujardin drew attention to them in his "Histoire Naturelle des Helminthes." In 1851 Franz Leydig gave much attention to the subject. In 1852 Remak published a paper on the subject; and since that year numerous studies have been made, but the more important are those of Lieberkühn, Balbiani, Bütschli, Pfeiffer, Henneguy, Thélohan, Perugia, Gurley, Doflein, Hofer, and Plehn.

Myxosporidia have appeared, and often occasioned great mortality, in certain rivers in Europe, Asia, and America. German rivers, especially the Weser, Elbe, Rhine Saar, and Mosel, have been especially afflicted. In France, where there has also been extensive mortality in crayfishes from Microsporidia, the Rivers Seule, Marne, Vilaine, and Doubs, have often been subject to scourges. In Italy, the River Mincio, and also the Mediterranean Sea, have on several occasions given fishes suffering from various species of these parasites. In Russia, the River Don has been the principal place of its activity.

In Asia, the Irtisch has been affected. In Africa, the Nile is also occasionally the seat of its ravages. It is very prevalent in various rivers of the United States of America and of South America.

In Great Britain and Ireland, the presence of these parasites, and the diseases caused by them, have not been observed. It is well to remark here that the serious study of the diseases of fish in this country only dates back to the great outbreak in salmon and other fish in various rivers from 1877 to 1883. The frightful mortality resulting from it seemed to have aroused the attention of those entrusted with the care of the fish industry; but, beyond a few departmental reports, not much has otherwise been done in this direction.

In some well-organised States, more especially in Germany, and to a lesser degree in France and in the United States of America, well-equipped laboratories have been established for the scientific study of diseases and the biology of fishes. In these favoured places eminent zoologists, bacteriologists, and pathologists, are employed in their investigation.

To show the importance of this study, it is well to mention that in one epizoötic alone occurring in Lake Mendota, U.S.A., in 1884, "it was estimated that fully 300 tons of fish had died up to that time. On August 7 the Madison Transcript reported that 200 tons had been hauled away by the city authorities during the four weeks preceding, and that the fishes were still dying" (Forbes). And one well knows the terrible losses of salmon and trout that occurred in our rivers from 1877 to 1883, and occasionally since. Such frightful losses must necessarily make the food fishes scarce, and in consequence very expensive. Salmon that could be bought for twopence a pound a little over a hundred years ago now ranges from one to three shillings, and is therefore beyond the reach of the majority of the population.

It is to be hoped that ere long the Board of Agriculture and Fisheries will copy other States, and set up a well-equipped fish station for the purpose of making scientific investigations with the object of preventing diseases among fishes and of increasing our fish-food supply.

Occurrence.—It usually occurs during the warmer months, notably those of spring, summer, and early autumn, of the year. Its comparative absence during the cold months indicates that cold is inimical to the development or spread of the spores.

Etiology.—Spores leaving the body by the natural channels of diseased fish, or by disintegration of cysts or abscesses on the surface of the skin, fall to the bottom of the water, and are swallowed by the new host with its food, and pass into its alimentary canal, and then commence to undergo germination. Infection does not result from direct inoculation by means of intermediate hosts or in other ways.

When the spore has reached the alimentary canal, the digestive juice acts on it, and extrudes the filaments of the polar capsules, which apparently enable it to anchor itself to the alimentary epithelium. The two valves of the spore-membrane slip along the suture, and allow the contained sporozoite to escape in the form of a minute amœbula (*Myxosporidium* of Gurley), which then enters the wall of the alimentary canal.

In some instances, it appears, the amœbula actively travels on foot to its destination or seat of predilection, boring its way through the tissues. In other cases it may be passively conveyed by the blood or lymph-stream. There is no evidence that the blood-corpuscles are attacked. Probably the parasites are merely suspended in the blood-plasma.

When the amœbula reaches its seat of election it invades the tissues, and begins to take in nourishment and to multiply endogenously with great rapidity. There is no doubt that Myxosporidia reproduce themselves endogenously within the host (*multiplicative reproduction*), as well as exogenously by means of spores (*propagative reproduction*).

Multiplicative reproduction may take place in the full-grown trophozoite by simple binary fission, or by the formation of buds from the protoplasmic body. Or the multiplicative reproduction may take place in the youngest stages of the parasite in the amœbula, which has just reached its seat of predilection. The nucleus of this amœbula splits up into numerous daughter nuclei by a process of "multiple mitosis." Afterwards the protoplasm becomes divided in the form of numerous minute uninucleate "swarm-spores," which carry the infection into the tissues of the organ invaded, or all over the tissues of the host.

Morphology.—Myxosporidia vary in size, according to their species, from 65 μ to 300 μ. Their form is mostly round, and their protoplasm is extremely granular. When examined in the fish's urine their movements are very slow. These movements are not observed when they are placed in water.

In appearance they are small rounded elements containing one or two nuclei. These nuclei are the *primitive spheres*, or *pansporoblasts*, in which the *spores* are formed. Each pansporoblast does not form more than two spores in the case of the Dispora, but in the case of the Polyspora more than two—usually many spores. They also contain, besides the spores, fatty granular matter, which, as well as the spores, are coloured by osmic acid. The spores have a complicated structure, varying according to the species. Their dimensions range from 8 to 36 μ. They are composed of an enveloping membrane and contents. This spore-membrane is formed by two valves attached to one another, like the two equal halves of a walnut-shell. It is homogeneous and transparent. One of the poles of the spore contains one to four vesicles or polar capsules, which are stained by methylene blue, thionin, and safranin, and elongated into a small canal, which becomes fixed to the pole. At this spot a very small opening is found, through which the spore escapes externally.

Inside each of the polar capsules a filament which is rolled up on itself is with great difficulty seen until the preparation is treated with a drop of glycerine or caustic potash, when the filament suddenly unrolls itself and protrudes. These filaments are sometimes well developed, and may obtain eight to ten times the length of the spore. Dilute Lugol's solution shows up the vacuole. Cold concentrated sulphuric acid produces separation of the valves.

The spore contents are made up of homogeneous protoplasm (sporoplasm), which contains a nucleus in its centre, and is coloured by safranin. Spore-formation corresponds to sexual reproduction or sporogony.

Habitat.—In marine fishes Myxosporidia occur chiefly in the gall-bladder, while in fresh-water fishes they occupy various parts of the body.

The bile-ducts and renal tubes, as well as the gall-bladder and urinary bladder, are more frequently the seat of these parasites than any other organ. The parenchyma or stroma of the liver and kidney, exclusive of their ducts and vesicles, and also of the spleen, are not often attacked. The testicle and ovary and the nervous system are rarely invaded. The alimentary canal, including stomach, pyloric cæca, and intestine, occasionally become the seat of their infestation. The gill-rays, intermuscular connective tissue, dermis, scales, and eyes, are the commonest seats of their inroads. The swim-bladder and peritoneal cavity are occasionally attacked.

Pathogenesis.—Myxosporidial infection is characterised by rapid disappearance of nuclei of the infected cells, and invasion of the parasites into all the tissues of the host. During their passive migration in the blood or lymph-streams the parasites may be found in the lymph or blood-plasma. A good deal of the blood must be absorbed by the parasites, as shown by the presence of hæmatoidin crystals in all the Myxosporidia. Cachexia, as seen in other animals suffering from protozoa, is also often produced.

Mode of Infection.—It is generally acknowledged that infection gains the system by ingestion of spores along with the food. Probably the lymph-channels of the connective tissue and the bloodvessels account for the spread of the parasites to the various parts of the system.

Symptoms.—The parasites invade all the tissues of the body, superficial as well as deep, including the skin, spleen, kidney, swim-bladder, testicle, ovary, central nervous system, the eye-cavity, peritoneal cavity, liver, urinary and gall-bladders, and branchiæ. They may cause emaciation, abscess formation, tumours, small cysts, cataract, epilepsy, anæmia, leucocythæmia, and other clinical forms of disease.

In many fishes, especially the stickleback, they often cause tumours, which may grow so enormously as to bring about death by pressure on the heart.

Myxosporidia are, however, mostly present without causing any serious disturbance of health. On the contrary, they often account for great destruction of fish.

The myxosporidial cyst imbedded in the tissues is invariably free from pigment, and always of a cream-white colour. It varies in size from a fraction of a millimetre to clusters of several centimetres in length. It is usually spherical or fusiform, but may be extremely variable in shape. As a rule, it can readily be removed from the tissues. The contents of the cyst are always milky or creamy, usually fluid, sometimes caseous, and consist of spores and more or less granular matter.

The principal diseases occasioned by the presence of Myxosporidia will be dealt with under their respective headings.

Myxoboliasis Tuberosa.

Synonyms.—*French :* La maladie des barbeaux.
German : Die Beulenkrankheit der Barben.

Definition.—A protozoal disease of the barbel, tench, gudgeon, pike, and perch, characterised by nodular purulent swellings on the external surface of the body, especially on the back, and due to a myxosporidium—*Myxobolus Pfeifferi.*

Epizoötiology. — For more than forty years this disease has been known to decimate barbel, and occasionally other fish, in certain rivers in France. It was noticed towards the end of 1870 to affect the fish in the River Moselle, and it has since frequently reappeared, especially in 1884, 1893, 1895, and 1902. In the Meuse, near the Charleville, it raged with all its truly epizoötic characters from 1883 to 1886. It gradually increased in virulence until it gained its maximum of intensity towards the middle of 1885. On certain days quite 100 kilogrammes of dead fish which covered the Meuse were interred. The disease then gradually appeared to become extinct, but actually because there were very few fish remaining. In the district of Ardennes it was only noticed in the Meuse itself; all the affluents have always been spared. The maximum of intensity, according to Railliet, was reached about the middle of 1884. At Mézières, during 1883 to 1886, on certain days as many as 200 pounds of fish were interred. Some years later the disease had disappeared from that region, but raged further down the river at Monthermé and Givet. At the present time it always exists as an enzoötic in the Meuse, with periods of recrudescence, as in 1902. According to Mégnin, barbel die in great numbers in the neighbourhood of Nancy. It gradually gained the Aisne, Marne, Seine, Saône, Rhône, and Garonne, where it produced an enormous mortality, more notably in the Marne, Saône, and Rhône. Its greatest toll was during the summer months, from May to September. It peculiarly confined itself to certain parts of a stream, and contaminated certain regions in a fearful manner, whilst other parts remained almost free from its ravages.

In the Rhine and its tributaries—the Saar and Mosel—according to Ludwig, it appeared several decades past, without, however, ever having obtained the magnitude that it has reached in late years in the Mosel, where it has been observed since the end of 1870, and has so increased that, especially in warm summer months, the dying and dead fish from the Upper Mosel and Saar pass Trier by the hundreds, and at Zell (on the Mosel) it is said that they spread a carrion-like odour. According to Pfeiffer, in the Saar and Mosel during the summer of 1890, no very extensive mortality occurred. An outbreak appeared in Lyons in 1898. Railliet thinks the disease extended to the Aisne and to the Marne from the Moselle via the canals. It is said that it is not rare in whitefish (Coregonus) in the Lakes of Peipus and Ladoga.

Causal Myxosporidium.—The *Myxobolus Pfeifferi* has a small lenticular or oval spore, having a length of 12 μ, by a breadth of 10 μ, and a thickness of 6 μ. It has a bivalve shell cavity, containing a sporoplasm and two capsules, the latter extruding filaments under the influence of caustic potash (Mégnin) or by glycerine (Pfeiffer).

Habitat.—It is found encysted or free in the muscles, mostly of the belly and sides of the body; in the liver, kidney, spleen, connective tissue of various organs; in the ovary, intestinal wall, and also in the heart.

Etiology.—Fish of various ages are attacked. Pfeiffer believes that the presence and development of the Myxosporidia, which are always present in barbel in the Rhine, Mosel, and Saar, only become pathogenic at irregular intervals, probably when other causes so diminish fish vitality that the reactive encapsuling of the parasite is no longer possible. The latter then obtains supremacy, and through the accompanying bacteria rapid death of the fish may result.

Mégnin considers that the parasites escaping from the ulcers are ingested with the water during deglutition or respiration. Under their amœboid form they enter the circulation, and then gain the subcutaneous cellular tissue, which is their seat of election, where they undergo their last stage of development. Ludwig suggests that, in consequence of the branchiæ being affected more than any other organ, the path of infection is through the respiratory tract more than the alimentary canal. The lymph paths of the connective tissue appear to represent the principal channels by which the parasite spreads through the system. Pfeiffer remarks that infection of previously healthy fishes is brought about through the extensive fouling of the water by the numerous dead fishes, and the resisting powers of the spores. Infection may then take place via the stomach, gills, or wounds. The last are of frequent occurrence in the spring at the breaking-up of the ice.

According to Railliet, season appears to have no influence, as fish are found dead in mid-winter, as well as in June, July, and August. Pollution of streams is only a minor factor. Ladague incriminates damming of rivers, which diminishes the rapidity of the current, in the midst of which the barbel ordinarily lives.

Treplin believes that the young fish receive the germ from the refuse deposits of breweries, malt-houses, tanneries, distilleries, etc., in the head-water of the Saar and Mosel, and that these protozoal germs enter by the alimentary canal, passing thence into the rest of the body, and first make their exit therefrom, via the ulcers, in the second or third year. Hanzo, on the contrary, considers the cloth and paper mills are chiefly responsible, as these establishments handle old rags which are, he says, saturated with infective material.

Ludwig considers that while no sufficient evidence exists for holding river pollution by various factories responsible, an indirect connection between such water pollution and barbel myxosporidiosis is by no means to be entirely rejected. It is quite possible for such pollution to favour the Myxosporidia to increase and develop, especially by diminishing the normal resistance of the system, and thereby rendering infection easy.

Symptoms.—The affected fish appear dull and not so sprightly as usual, and have much difficulty in ascending rivers. The surface of the body has lost its gloss, is of a smoky, greyish-yellow appearance, and feels to the touch oily and slippery. They are easily taken in the hand when endeavouring with difficulty to glide through the water. They avoid streams, although these are their natural haunts, but are easily taken in great numbers with nets. When the disease is far advanced, the fish lose in weight, although their bulk may appear normal.

On the surface of the body several or numerous (ten to fifty) tumours appear. These tumours, usually about ten to fifteen in number, vary in size from a pea up to a fowl's egg. They have a very resistant wall of 1 to 1·5 millimetres in thickness, semicircular or oval in outline, and sometimes run into one another, forming patches of 17 to 20 millimetres in length, by 7 or 8 millimetres in breadth, in fishes weighing about 5 pounds in weight. The scales covering the tumour are raised, easily detached, and finally shed. Some fish die before the tumours burst. Not all the fish that die have external tumours, and in that case they are found located in the viscera. They are usually of the size of a walnut. Sometimes, however, they may be in length 50 millimetres, and in width 20 millimetres, single or multiple, usually on the belly or sides, and filled with a yellow or caseous purulent mass. The tumours soften and burst, leaving deep crateriform bloody-bordered ulcers, covered with a yellowish purulent mass, consisting of spores and of cell detritus. Among the latter large bacilli are found.

The active agents in the puriform transformation and opening of the tumour are the bacilli first observed by Pfeiffer in the ulcer contents. These are only found in the myxosporidial infected muscles; never in other organs. The presence of these micro-organisms either prevents connective-tissue proliferation entirely, or obstructs it from becoming complete, the tissue undergoing necrosis, which soon results in destruction of the overlying tissues. These microbes were subsequently studied by Thélohan, who observed two kinds of them:

1. Large motile *bacilli*, having a length of that of the spores, and showing with hæmatoxylin four or five red granules, and a short flagellum. Frequently several of these cohere by their surfaces, and also occur in the form of long separated threads. Their length is 6 μ. Sometimes they appear isolated or in linear colonies, without any motion being witnessed. They rapidly liquefy gelatine, upon which large, slightly yellowish-white colonies appear. Inoculated into rabbits, they cause a small circumscribed abscess to form. They readily stain with methylene blue, gentian violet, fuchsin, etc.

2. Not so frequently, occasionally with or without the bacilli, *cocci* occur, either isolated or united in the form of streptococci or diplococci.

The primary seat of infection appears to be the interior of the muscle-cell. Myxosporidia are found within distinctly transverse striate or markedly atrophied muscular fibrillæ, and also between healthy fibrillæ. Atrophied muscle-cells are seen, containing long rows of well-developed spores, which, on account of the absence of filaments within the capsules, Pfeiffer inclines to believe have reached their present position by a general immigration. In places the fibrillæ are beaded, such muscle bead-strings being generally heaped near together in the vicinity of the hard cysts. Around the cysts the muscular tissue is infiltrated with blood, the infiltration, where superficial, being visible through the skin. Near the ulcers the muscular substance is broken up, loosened, fatty degenerated, and contains blood-coloured tubes, with numerous Myxosporidia not yet encapsuled, and also well-developed spores. According to Thélohan, they are very frequently encountered. Balbiani, who has studied them in the ovary of the barbel, has seen that the sporozoal matter does not confine itself to travelling via the connective tissue, but often invades the young ovules.

Lesions.—The presence of the Myxobolus in the primitive muscle-fibre appears to lead rapidly to degeneration. If fresh fragments are examined, the fibres are seen, which in places have preserved their normal appearance and their striation, and at other points more or less considerable spaces, where the muscular substance is filled with a vitreous refringent mass, around and in the intervals of which lie fatty droplets, yellowish granules, and spores. The degeneration invades gradually the muscular substance of the primitive fibres, and one finds it in parts of these elements where the parasite appears not to have penetrated. On the contrary, the neighbouring non-infested primitive fibres seem exempt from that alteration, and one frequently observes a degenerated fibre surrounded by healthy ones.

The fibres thus degenerated and broken up are soon invaded by phagocytic cells coming, some from the sarcolemma, others from the connective tissue. This latter at the diseased points is the seat of a very marked irritative proliferation. It is necessary to distinguish in the degenerated fibres the parts where spores are found in great number and those where these elements are few or absent, the degenerative process in the latter case having originated from the presence of the parasite at a different point. In this latter case the cells which have penetrated into the degenerated tissue multiply rapidly. In proportion as their number augments, one sees the muscular débris diminish. Very soon they have completely disappeared, the place of the fibre being finally occupied by connective tissue. While these phenomena occur, the irritation is propagated, the connective-tissue proliferation extends itself, and a sclerosis of the neighbouring muscle region, with atrophy of the primitive fibres, is produced. At the points where the degenerated fibres enclose a great number of spores the formation of connective tissue is at first limited to a thickening of the perimysium. There are thus formed connective-tissue bridges separating the spaces occupied by the spores, and which correspond to disappeared primitive fibres. These facts are seen especially clearly on transverse sections. Gradually these bridges increase in thickness; at the same time their tissue becomes more dense. They thus form around each space a fibrous shell, which tends to contract more and more. There seems to be here a true encystment of the parasite, such as is produced around foreign bodies introduced into the tissues (Thélohan).

Treatment.—According to Pierre Mégnin, it is difficult to arrest the epizoötic beyond collecting all the dead and diseased fishes, and destroying them by fire. Ludwig is of a similar opinion, but he thinks rivers should be maintained in a high state of purity, and that pollution should be interdicted. He considers that the most dangerous contamination of the water is by the ulcers containing the *myxosporidia*, which might be mitigated to a certain extent by destroying all dead or diseased fish, instead of throwing them back into the water, as anglers are often wont to do. Railliet has seen good results from this practice, and at the end of some years the disease appears to have left no traces. Pfeiffer is in agreement, but considers that after careful removal of fishes dead of the disease they should be burned, or buried with caustic alkali.

Ladague has succeeded in prolonging the life of the fish, and sometimes curing it, by opening the tumours. Of course, this method is impracticable in an epizoötic.

Public Health.—A case occurred in 1898 in Lyons in which a young man who had eaten diseased barbel from time to time was suspected of tuberculosis, but instead of the bacillus of tuberculosis in his sputum, the *Myxobolus Pfeifferi* was found.

Protozoal Gid in Rainbow Trout.

Synonyms.—*French :* Le tournis de la truite arc-en-ciel.
German : Die Drehkrankheit der Regenbogenforelle.

The young rainbow trout (*Salmo irideus*) reared artificially in fish-raising establishments are subject to a vertigo or epilepsy simulating that in sheep due to the *Cœnurus cerebralis.* In the fish, however, the parasite is a *myxosporidium*—the *Myxobolus cerebralis* Hofer (*M. chondrophagus* Plehn)—which takes up its residence in the brain. It has been chiefly observed in Germany, where so much attention is given to pisciculture and to the diseases of fish.

Symptoms.—The affected trout appears quite well, when suddenly it flexes its body either to the right or to the left, and spins round on the same side on itself from ten to twenty times in rapid succession. After getting exhausted, it then falls to the bottom of the water, and remains there for a very short period without moving. Ultimately it swims about as if it were quite well, until after the lapse of more or less time it has a fresh attack, when it goes through the same series of performances.

These attacks may be repeated for days, and even weeks, when the fish usually succumbs without taking on any peculiar aspect. There is no treatment for it.

Protozoal Jaundice in the Common Trout.

The common trout (*Salmo fario*) is sometimes attacked by a myxosporidium (*Chloromyxum truttæ*), which is found in the gall-bladder, and gives rise to a chronic disease, of which jaundice is the most pronounced symptom. It is usually encountered on trout of about 3 to 10 ounces in weight living in tanks in fish-culture establishments.

The affected fish gradually lose their appetites, become weak, and shortly after develop a yellowish-brown diarrhœa. They become thin, and then lose their activity; and the fins and the normal lighter parts of skin, especially that of the axillæ and belly, gradually take on a yellowish discoloration. This disease may last several months, and the fish end up by becoming very much emaciated and anæmic, and then die.

The gall-bladder and bile-ducts contain a very great number of the myxosporidiæ. These parasites did not exist in healthy trout or in streams in the neighbourhood.

Gurley found the *C. mucronatum* in 20 per cent. of burbot or eel-pouch (*Lota lota,* or *L. vulgaris*) examined.

Epithelioma Papulosum.

Synonyms.—Small-pox, or variola of carp ; Myxosporidiosis.
French : La petite vérole de la carpe ; Variole.
German : Die Pockenkrankheit des Karpfens.

This disease was known to Conrad Gessner, who described it in his book "On Fishes," published in Zurich in 1563. Its nature was studied in 1896, 1898, and 1901, by Hofer and Doflein, who attributed it to a myxosporidium—*Myxobolus cyprini.*

It frequently rages as a destructive enzoötic in carp reared in special fish-ponds, and from the peculiar spotted appearance of the skin fish-rearers term it variola, or small-pox of the carp. The tench is occasionally attacked.

Symptoms.—Small whitish spots appear on the skin. At first these are confined to the head and the free border of the fins, but they end up by gradually extending and covering almost the whole of the body of the fish. The white patches thicken, and then fall of themselves, but it is not long before they re-form. Carp thus affected waste away, and often die in great numbers. At other times they resist the disease, which does not alter the quality of their flesh, but by its presence their market value is considerably depreciated.

The large indurated tumours of the skin, consisting of epithelial proliferations, which are invaded by leucocytes and by an increased growth of bloodvessels from the cutis, do not appear to contain parasites or intruding organisms of any kind. In all the affected fish *Myxobolus cyprini* are found to be plentiful in the spleen, liver, and kidneys. Hofer and Doflein consider this skin eruption of the carp to be an indirect effect of the interference with the metabolism caused by the presence of the parasite in the internal organisms, more particularly the kidney. This view, however, has been strongly opposed. Probably the tumours are set up by invisible organisms.

Treatment.—As this disease, which chiefly occurs in special fish-ponds, remains for a long time in the water, the pond should be emptied, cleaned out, especially in winter, and then well coated with freshly-slaked lime. After a fortnight the pond should be refilled.

All those fish showing spots should be eliminated.

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III. THE MICROSPORIDIA (CRYPTOCYSTES).

The Microsporidia is *Order II.* of Neosporidia, which is *Class II.* of the *subphyllum* Sporozoa. They differ from the myxosporidia by having very minute spores, which are piriform in shape, and with only one polar capsule, which is *invisible in a fresh state.* They are mostly parasites of fishes and the invertebrates, especially of crustaceans and other arthropods. They are found in the silkworm disease and the Isle of Wight bee disease.

They have one *family*—Glugeidæ, or Nosematidæ—which has all the characters of the order. This family is divided into *two groups*—Polysporogenea and Oligosporogenea.

The *group* Polysporogenea is characterised by the trophozoite, producing many pansporoblasts or primitive spheres, each of which gives rise to many spores. It only contains one *genus*—Glugea, or Nosema—to which belongs the causal parasite of silkworm disease—Glugea (Nosema) bombycis—and that of the Isle of Wight bee disease—Glugea (Nosema) apis.

Genus Glugea, or Nosema.

Sporozoön.	Habitat.	Host.
		FISHES :
G. (N.) acuta.	Connective tissue, dorsal fin.	Nerophis æquoreus and Syngnathus acus.
G. (N.) anomala (or G. (N.) microspora).	Subcutaneous connective tissue, ovary, cornea.	Gasterosteus asculeatus, G. pungitius, Gobius minutus, and Lacunculus albus.
G. (N.) cordis.	Heart.	Clupea pilchardus.
G. (N.) depressa.	Liver.	Coris julis.
G. (N.) destruens.	Muscles.	Callionymus lyra.
G. (N.) gigantea.	Body cavity.	Crenilabrus melops.
G. (N.) Girardini.	Skin, muscle, wall of intestine.	Girardinus *sp.*
G. or N. lophii.	Spinal ganglia and cranial nerves.	Lophius piscatorius.
G. or N. marionis.	Gall-bladder.	Coris giofredi, C. julis.
G. or N. ovoidea.	Liver.	Cepola rubescens, Motella tricirrata.
G. or N. punctifera.	Connective tissue of eye muscles.	Gadus pollachius.
G. (N.) Stephani.	Gut.	Flexus passer.
G. (N.) *sp.* (Pfeiffer)	Gut.	Leuciscus phoxinus, Pleuronectes platessa.
		HEXAPODA :
G. (N.) apis.	Organs of alimentary tract.	Apis mellifera (honey-bee), Bombus terrestris, B. lapidarius, B. hortorum.
G. (N.) bombycis.	All organs.	Bombyx mori (silkworm).

The *group* Oligosporogenea have a trophozoite that produces a single pansporoblast. In this group there are three genera—Gurleya, Thélohania, and Pleistophora.

The *genus* Gurleya has a pansporoblast that produces four spores. As far as is known, it does not affect economic animals.

The *genus* Thélohania has a pansporoblast that produces eight spores contained in small spherical or fusiform vesicles. They occur as parasites in crayfish and other crustaceans.

Sporozoön.	Habitat.	Crustacean Host.
T. Contejeani.	Muscles.	Astacus astacus (crayfish).
T. Giardi.	Muscles.	Crangon crangon.
T. macrocystis.	Muscles.	Palæmonetes varians.
T. Mülleri.	Muscles.	Gammarus pulex.
T. octosporo.	Muscles.	Palæmon aspersus and P. serratus (prawns).

The *genus* Pleistophora has pansporoblasts, which produce more than eight spores. They occur chiefly in fishes, but also in invertebrates.

Sporozoön.	Habitat.	Fish Host.
P. acerinæ.	Mesentery.	Acerina cernua.
P. mirandellæ.	Ovary.	Alburnus mirandella.
P. typicalis.	Muscles.	Cottus scarpius, C. bubalis, Blennius pholis, Gasterosteus pungitius.

The Microsporidioses.

The microsporidioses include a few well-known diseases of fishes, crayfishes, bees, and silkworms.

The Microsporidia causing this class of disease commonly infect cells. The pansporoblast or sporont, as exemplified in *Thélohania Mülleri*, has a nucleus which divides without mitosis into eight nuclei, surrounded by the protoplasm, which becomes segmented to form eight sporoblasts, implanted in an intercellular residuum within the envelope of the sporont. Each sporoblast becomes a spore, which has first one nucleus, and afterwards two. When a fresh host is artificially infected, the spores remain some time in the gut before germinating, during which time the two nuclei divide, so that the spore ready to hatch has four nuclei.

The spore is generally minute—about 2·5 μ by 1·5 μ in *Glugea* (*N.*) *ovoidea*, and 4 μ by 3 μ in *G.* (*N.*) *anomala*—and uniformly pear-shaped. The sporozoite or sporoplasm in Glugeidæ contains almost constantly a clear vacuole at the broader or posterior extremity, and often numerous fine fatty globules. In *Glugea* (*Nosema*) *bombycis* of silkworm disease, congenital infection takes place by penetration of the parasite into the ovary, and the formation of spores within the ovum itself, so that the silkworms are, when newly hatched, already infected with the disease, and disseminate it amongst healthy individuals. Infection of older caterpillars is brought about entirely by accidental ingestion of spores along with the food. Careful microscopical examination of the eggs of the insect for cysts, and destruction of all that were found to be infected, together with rigid inspection of the food, prevented a continuation of inheritance of the disease from generation to generation, thanks to the labours of Pasteur, terminated an epizoötic which had cost France almost 1,000,000,000 francs.

Nodular Disease of Fishes.

Synonyms.—*German :* Die Knotchenkrankheit ; Morbus nodulosus.

This is a disease due to the presence of a microsporidial parasite—*Glugea* (*Nosema*) *anomala*—and characterised by the appearance of oval or spherical nodules on the surface of the body.

It attacks various fishes, more especially the three-spined stickleback (*Gasterosteus asculeatus*), the nine (ten or eleven) spined stickleback—*G.* (*Pygosteus*) *pungitius*—and *Gobius albus.*

From one to four or five, up to a dozen or more, of these tumours appear on various parts of the surface of the body, and are more or less in contact or widely separated from one another. The size of the tumour, which varies from that of a pin's head to that of a large pea, bears no relation to the size of the fish. Its colour is generally whitish, but, when covered by the epidermis, silvery.

The microsporidium *Glugea* (*Nosema*) *anomala* has numerous transparent and regularly ovoid spores of 3 to 5 μ in length, by 2 to 3 μ in breadth, the size and form being constant in the spores of larger cysts, but less clear in those from the smaller, The shell is bivalve. There is only one capsule, which has a very long filament of 50 μ that is extruded under the influence of iodine.

The tumours do not seem to cause much disturbance of function, even when they occupy the internal surface of the operculum or the fins, which move as freely without any impediment as those of sticklebacks not so affected. They may, however, bring about compression and atrophy of the muscles and compression of the viscera. The tumours may be carefully dissected out without injuring the fish, which appears as well as ever after the operation. They may, however, increase to such a size as to burst their external covering, and shell themselves out, leaving in place of the tumour an excavation irregularly limited by a ridge formed by the non-empty part of the small sphere. Small secondary vesicles then develop rapidly, and form an irregular strawberry-like mass.

This disease should not be mistaken for other nodular diseases, such as those due to *Myxobolus exiguus*, *M. Mülleri*, *M. dispar*, and *M. oviformis*.

Crayfish and Prawn Plague.

Synonyms.—*French :* La peste des écrévisses ; La maladie des écrévisses.
German : Krebspest.

Crayfish and prawns, which are largely cultivated in France, Germany, Russia, and other countries in Continental Europe, as articles of commerce, are not rarely affected by a malignant epizoötic, which was at one time thought to be due to a microsporidium—*Thélohania Contejeani*—which was present in the striated muscles, in which it caused an opacity and a separation of the fibrillæ. This parasite has no cyst. Its spore is ovoid, 2 to 3 μ in length, and has a clear vacuole in the larger end. In the case of prawns—*Palæmon rectirostris* and *P. serratus*—another species of microsporidium is encountered—namely, *Thélohania octosporo*—whose spore is 3 to 4 μ in length, pyriform, very refringent, has a capsule with a long filament of 40 to 50 μ, which is extruded by ether. It produces a chalky porcellaneous opacity of the muscles. This may be only limited to parts, but in advanced cases the entire body becomes white, except the region of the heart and stomach. As artificial infection by feeding could not be produced in prawns, other lines of investigation had to be sought.

This disease appears during the warmer months of the year—July and August—declining gradually during September and October, and disappears entirely by November, to reappear next springtime.

Two parasites were noticed by Henneguy and Thélohan—a microsporidium and a fungus. The parasite *T. Contejeani* was confined in the intestinal canal in June and July, but in the muscles and intestines in October.

Hofer and Albrecht found that this plague is due to a special bacillus—*Bacterium pestis astaci*—provided with two, three, or more cilia. It is from 1 to 1·5 μ in length, by 0·25 μ in breadth. This bacillus is also the causal organism of Lepidorthosis.

The diseased crayfishes are secondarily affected by a saprolegniaceous fungus—*Aphanomyces schikoras*.

It is thought to be transmitted from one country to another by water-birds.

Microsporidiosis of the Honey-Bee.

Synonyms.—Isle of Wight bee disease ; May disease ; Bee pest ; Contagious dysentery ; Bee paralysis ; Foul brood ; Vertigo dizziness.

French : Mal de mai.
German : Maikrankheit ; Fussgangerei.
Italian : Mal de maggio.

Definition : A contagious disease of the various varieties of the honey-bee—*Apis mellifica*—due to a sporozoal organism—the *Glugea* (*Nosema*) *apis*—and causing a very great loss of bees in various parts of the world.

Geographical Distribution.—This disease is widely spread in England, Scotland, Ireland, Germany, Switzerland, America, Australia, France, Italy, Brazil, Canada, and in other countries where bees are extensively kept in a state of artificial cultivation.

History.—It is evidently one of the diseases of bees mentioned by Aristotle in Book VIII., chap. xxvi., and Book IX., chap. xxvii., of his "History of Animals." It is alluded to by Pliny. No doubt it has been known from time immemorial. Coming back to our own country, we find that Butler recorded that it was very prevalent during 1611, and again in 1621. Purchas informs us that 1648 and 1651 were not favourable to honey-gathering, and that they were inimical to bee life, hundreds of hives being lost. In 1759, and again in 1777, there were great losses, as well as from 1781 to 1783, and again in 1788, when fifteen out of sixteen hives were lost. Disease was particularly rampant in 1806, 1810, 1815, 1820, 1824, 1828, 1829, 1833, and 1837. It seems to have prevailed again in 1844. In France, in 1853 and 1855, after wet and cold springs, this disease raged to such an extent that quite three-fourths, and in some localities two-thirds, of the hives were destroyed. It was particularly noticeable in this country and elsewhere in 1862, 1863, 1881, 1882, 1906, 1907, 1909, and 1911.

Etiology.—Nearly every one of the ancient theories concerning the causation of malignant epizoötic diseases has been utilised to account for the appearance of this disastrous malady. But as far back as 1860 Leuckart advanced the opinion that foul-brood (Nosema) was due to the same organism as that causing the silkworm disease (Pébrine). In conjunction with Dönhoff, this authority in 1857 found small refractile oval bodies in the intestines of diseased bees, and thought that these bodies were the spores of a fungus which, when given to healthy bees, produced the disease. In 1858 Higgins, and in 1863 Leydig, also observed spores. Since that time not much, if any, attention was given to the unrecognised nature of the parasite until it was independently found by Fantham and Porter in 1906, and by Zander in 1907, and named *Nosema apis*. From 1906 to 1911 serious attention was devoted to it by Fantham and Porter, and during 1911 further researches were undertaken by Graham-Smith and Bullamore.

It has been shown that infection may be carried through the agency of infected foods or of living infected bees. Water, especially rain-water, contaminated with excrement in the vicinity of hives, and honey present in hives in which bees have died, seem to be the most important infected foods. Foraging bees infected by ingesting these foods, and "parasite carriers," whether queens, drones, or workers, are the most important agents of infection. The latter may be present in stocks which have never suffered from the disease, or in dwindling stocks showing few symptoms, or in stocks which have suffered from the disease and apparently recovered.

The introduction of infection into a hive is not necessarily followed by the appearance of the symptoms of the disease. Probably some stocks may remain healthy, and the infected bees are gradually cleared out; at other times weeks or months may pass before the disease manifests itself. Occasionally, the hive suffers severely for a time, and then apparently recovers, though generally it succumbs in the end. Not rarely the bees manifest a mild form of the disease, but gradually become weaker and die, and occasionally an acute disease suddenly appears in a few days. As in other epizoötics, some stocks do not develop symptoms, and although partial immunity is acquired, they act as infection carriers.

Other factors play an important part, such as virulence of the sporozoön, natural resistance of the bees, the depressing condition of the weather, the bad quality of the food, and the insanitary state of the hives.

Many bees may have a considerable number of parasites developing in the cells of the chyle stomach, which reach the spore stage, and yet seem to resist. Old spores from dead bees are less virulent than fresh spores from recently dead bees. Probably the spores, having a weak virulence, develop into a great degree of virulence by passage.

Unnatural food, especially for wintering, lessens the natural resistance of the bee, and enables the infection to develop more readily. Cold and damp weather, and other unsuitable conditions, act in a similar manner.

Protozoölogy.—The *Glugea* (*Nosema*) *apis*, like the *Glugea* (*Nosema*) *bombycis*, which causes the very malignant disease of silkworms (pébrine), belongs to the Protozoa. It is included in that section known as Sporozoa, and, in consequence of having very small spores of a somewhat complicated structure, it receives a place in the group known as the Microsporidia, which closely resemble, if they are not identical with, the Myxosporidia—a group of parasites, which has been shown to be very fatal to fishes and other aquatic inferior animals.

The spore, which is taken as the starting-point in the life-cycle, enters the chyle stomach along with the food, where its wall is macerated by the action of the digestive juice of the bee, which penetrates to the spore contents.

The sporoplasm, stimulated by the juice, apparently presses the vacuole, so that the polar filament is forcibly ejected. It serves for a short period as an organ of attachment, fixing the spore to the gut wall. The sporoplasm concentrates and moves forwards, whereby the polar filament is forced still farther outwards, and becomes disconnected from the spore. The sporoplasm, retaining two of the nuclei, creeps out from the sporocyst, leaving the two sporocyst nuclei behind. The free sporoplasm becomes amœboid. The binucleate amœbula creeps about over the intestinal surface. The nuclei may fuse, or more often the amœbula proceeds to form amœbulæ. The final active amœbulæ are small, roundish organisms, each with a single nucleus containing a karyosome. Each amœbula is capable of amœboid movement. It penetrates the cells of the gut, or else between them, and finally either enters one of the cells or goes beyond and floats in the hæmocælic fluid.

Assuming that it enters an epithelium cell of the gut, it becomes rounded and passive therein, and after a period of growth, during which time it is known as a trophozoite, commences to multiply by several methods. There results usually a collection of separate daughter individuals or meronts, or chains of meronts. The chain condition is somewhat less common than the separate forms in Fantham and Porter's experience so far. Each meront is uninucleate. Alternately, a meront may form a large, multinucleate body, in which cleavage into daughter meronts does not occur. Such bodies may be intercellular or intracellular.

After a period of active growth and division producing uninucleate meronts, spore formation begins. The organism is now called a pansporoblast. Active amitotic nuclear division occurs, resulting in the production of five nuclei. Two vacuoles also form in the developing spore—a large one at one end, called the "posterior vacuole," and a small one at the opposite end, called the "polar capsule." The living substance or sporoplasm then forms a somewhat ring-shaped mass between the two vacuoles, and in it are lodged the five nuclei arising by division from the meront nucleus. Two migrate to the sides. These become elongated, and may be termed "sporocyst nuclei." Of the other three nuclei, one controls for a time the polar capsule, and the other two the sporoplasm. The polar capsule gradually forms the spiral polar filament, which extends down into the posterior vacuole. While these nuclear changes are taking place in the sporoblast or young spore, the latter is forming a coat around itself. This spore coat, or sporocyst, gradually thickens, and obscures the nuclei beneath, and the final result is that from one pansporoblast a single spore, resembling a rice grain in shape, with a shining refractile sporocyst, is produced. In this condition the spore passes into the lumen of the gut, is voided with the fæces, and remains a source of infection for some time (Fantham and Porter).

The most dangerous stage to the host is the first part of the life of Nosema, when it is occupied in growth and active division, so that the parasites prodigiously increase in the host. This multiplicative stage is known as "merogony." In many instances the younger stages of the parasite alone are quite enough to kill the bee, and in this case the spore stage of its development is not obtained.

Merogony of the sporozoön serves for the infection and destruction of a single bee; sporogony is a means for the spread of the disease to new hosts.

The multiplicative stages (merozoites) of *Eimeria* (*Coccidium*) *avium* are similarly destructive to young grouse.

Associated Bacteria and Protozoa.—Malden has found in about 40 per cent. of the infected bees, either in the chyle stomach or in the small intestine, a bacillus —*B. pestiformis apis*—which probably lives in symbiosis with the microsporidium *Glugea* (*Nosema*) *apis*. Infection with this bacillus failed to induce the disease in healthy bees. Fantham and Porter occasionally observed several species of protozoa, including various species of gregarines, a flagellate apparently belonging to the genus Crithidia, a new amœba (*Entamœba apis*), and a spirochæte in small numbers in the intestinal tracts of a few diseased and normal bees. These authorities are of opinion that they have no connection with this disease.

Microscopical Examination.—Small portions from different parts of the alimentary canal should be teased out in a drop of water, and mounted under a cover-glass. If the spores of *Glugea* (*Nosema*) *apis* are present, they can be easily seen as oval, highly refractile bodies, 2 to 4 μ in diameter, and 4 to 6 μ in length. Frequently they are seen lying between the cells in preparations made from heavily-infected bees. In the very early stages some of the cells of the gut wall, especially those of the chyle stomach, are distended with them. The parasites do not readily stain.

Fungi rapidly develop in the alimentary canal of the bee after death, and destroy the epithelium, often making the search for the young stages of *Glugea* (*Nosema*) *apis* unsatisfactory or impossible.

The Glugea are fairly well stained with Giemsa, hæmotoxylin, modified Romanowsky, and glycerine hæmatoxin.

Period of Incubation.—This may be as short as two or three days, or last for one to several weeks. It varies according to the means of infection, the susceptibility of the bees, the virulence of the sporozoa, and climatic conditions.

Symptoms.—One of the most characteristic features of this disease is the great mortality among the bees, they are especially slow in developing, and reduced in numbers, notwithstanding they have been carefully tended and have had an abundance of food. The bees usually seem weak, fall off on alighting on the board, or gather in clusters on the ground, or crawl about. In many instances their abdomens are distended, or they have a kind of diarrhœa, which fouls the interior of the hive. In other cases the wings droop, the bees appear paralysed, and either die inside the hive or unaccountably disappear. They usually seem disinclined to work.

The disease generally appears in the foragers first. It attacks the queens, drones, and young bees, as well as the foragers. It is capable of affecting the common humble-bee, the mason bee, and other species of the same order. Wasps are also very susceptible.

Diagnosis.—This can only be well made out by microscopical examination, when, if spores in their various forms or stages are present, they can as a rule be easily detected in a freshly-killed or recently-dead bee.

Treatment.—As the disease is transmitted from bee to bee, from hive to hive, from apiary to apiary, from district to district, and probably from country to country by commercial transactions in bees, and occasions great losses, it should be scheduled under the Diseases of Animals Act. Infected hives should either be destroyed or thoroughly submitted to the action of heat applied by a plumber's or painter's petroleum lamp. The dead bees should be burnt.

The writer acknowledges his indebtedness to the excellent report mentioned below for the subject-matter of this article.

Microsporidiosis of the Silkworm.

Synonyms.—The silkworm disease.

French : La pébrine.
German : Pebrinekrankheit.

Definition.—A malignant contagious and hereditary disease of the silkworm (*Bombyx mori*), due to a microsporidium (*Glugea* (*Nosema*) *bombyces*)—a sporozoal parasite closely related to the *Glugea* (*Nosema*) *apis*, the causal factor of the bee plague or Isle of Wight bee disease.

History.—Chinese tradition has it that the silkworm industry was introduced by the Empress Siling-Chi, wife of the Emperor Hoang-Ti, 2697 B.C. For more than 2,000 years it was a profound Chinese secret, until it gradually extended into Japan and Persia. It was only towards the middle of the sixth century of the Christian era that sericiculture was introduced into Europe. In 1853, in France alone, 26,000,000 kilogrammes of silk were raised; in 1854 this production declined to 21,500,000; in 1854 to 19,800,000; in 1856 to 7,500,000; and then went on gradually declining until 1865, when no more than 4,000,000 were obtained. This great decline was due to the disease under consideration. The disease was so acute and malignant that it destroyed in a few days the best "educations," in spite of the great care and attention devoted to them. The losses in France alone from this scourge were estimated at more than 1,000,000,000 francs.

In the year 1849 this disease first attracted attention, when Guérin Menneville observed in the blood of silkworms small refractive corpuscles or spores, oval in shape, endowed with a special movement, having a well-defined outline, and being in their long axis from 2 to 3 μ. De Fillippi observed these corpuscles in all the organs in the larvæ and moth, as well as in the blood of the worm. Leydig recognised the same or similar spores closely allied to those found in several insects and crustaceans, among the latter the crayfish. This authority, whose opinions were popularised by Balbiani, considered these spores as parasites, and ranked them in the genus Psorosperma, in the division of Microsporidia. Cornelia found in the blood of "charbonneux" moths, having cockled or curl wings and a swollen abdomen, an abundance of corpuscles or spores. Dr. Osimo, of Padua, discovered these spores in the eggs, and Dr. Carlo Vitadini recognised that the size of the corpuscular eggs increased in a measure as the time of hatching appeared, and proposed that a microscopical examination should be made to distinguish good from bad eggs. He also advised that such an examination should be extended to the chrysalides.

But no serious attempt was made to combat the disease until 1865, when Pasteur was commissioned by the French Government to make a scientific inquiry into its nature, with a view of preventing its ravages. The result of Pasteur's labours is much more remarkable when we consider the numerous savants who up to that time had failed to define its cause, and therefore an efficacious remedy. In 1867 Pasteur demonstrated that pébrine was especially hereditary, and that the eggs laid by the moths free from corpuscles or spores hatched out healthy silkworms. He then discovered means of obtaining invariably healthy eggs. By isolating each pair of moths in small cells until they have finished laying their eggs, and then microscopically examining them, one can discover if the moths are free from spores, in which case the eggs are utilised. In the contrary case both the moths and their eggs are destroyed. This means of inspection is so simple and satisfactory that mere children are employed in France to see if the moths are free from spores.

Pasteur's services in this direction to French agriculture have been estimated at an amount in one year that would pay the German War indemnity.

The disease has been found in silkworms' eggs coming from Japan, Turkey, Asia Minor, Roumania, China, etc.

Protozoölogy.—This is similar to that of the *Glugea* (*Nosema*) *apis*, and therefore need not be discussed.

Symptoms.—Mostly the surface of the skin of the diseased silkworms show black spots scattered about irregularly, and from the fact that the worm has an appearance as if it had been peppered it is called by the French "pébrine," derived from a Provençal word, *pèbre* (pepper). These spots should not be confounded with those arising from wounds due to the terminal claws of the anterior feet when they tread on one another. When the disease is contracted in a later stage, the worm is about to make its cocoon, and transforms itself into a chrysalis and even in the moth stage. Sometimes then the chrysalis manifests blackish tints on the wing implantations, and the moth issues forth with curled-up wings, and the down covering its body has a black tint, and from this fact the moth received the name of "charbonneux."

Sometimes a great quantity of eggs do not hatch, or, rather, the young die within a few days of their birth. As a rule, however, they hatch readily enough, and it is only later on that the malady gradually manifests its ravages. At any age the worms appear weakly, do not feed well, growth is retarded, and moult with difficulty. A great number of such worms succumb; their corpses become desiccated and get mixed up with their litter, and from day to day the stock dwindles. Sometimes the symptoms of the disease only develop after the third or fourth moultings, which is very deceptive to the silkworm rearer, who up to then was in great hopes of a good harvest.

As in the bee plague, a great number of silkworms having the disease in their systems do not show any external evidence of it.

Diagnosis.—By a microscopical examination of the eggs, larvæ, pupæ, and moths, under the same conditions as advised in the case of the bee disease, the parasitic spores can be made out. It is not necessary to dissect the worms or moths, but to simply rub them up in a little water in a mortar, place on a glass slide, and examine them under the microscope.

The disease cannot be mistaken for "*La Flacherie*," which is due to a specia vibrion, but which is also very contagious and hereditary; nor for "*La Muscardine*," which is due to a fungus of the genus Botrytis, and named *Botrytis Bassiana*. This disease is contagious, but not hereditary.

Means of Prevention.—As advised by Pasteur in 1865, and since carried out with great success, the eggs coming from only healthy moths should be utilised; all those of diseased moths should be destroyed. The houses, utensils, etc., require to be thoroughly cleansed and disinfected. Japanese and other strains of silkworms, although they may be greatly infected, seem immune to the ravages of the microsporidium. Nevertheless, they are very dangerous to susceptible stocks in a new locality.

In France there is an Act in force against this disease.

Microsporidiosis of Other Species.

It is advisable to just mention that Microsporidia have been met with in other species, the *Glugea* (*Nosema*) *ovoideum* as a parasite of *Motella tricirrata* and of *Cepola rubescens*, and *Glugea* (*Nosema*) *bryozoïdes* in some of the bryozoa. They have also been found in the lepidoptera, various insects, and in a cyprinodont fish.

(For the natural history of the Microsporidia and other Sporozoa, see Lankester's "Zoology," Part I., second fascicule, by Minchin and others in "Introduction and Protozoa.")

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IV. SARCOSPORIDIOSIS.

Synonyms.—Psorospermosis of the muscles and connective tissue; Sarcocysts; Miescher's utricles, sacs, or tubes; Rainey's bodies or corpuscles; Cattle-plague (Rinderpest) bodies; Entozoön-like bodies; Cobbold's spurious entozoa; Amœboid bodies; Corpuscles produced by muscular degeneration.

French: Sarcosporidiose.
German: Sarcosporidiosis.

Definition.—Sarcosporidiosis is a disease of the striped and unstriped muscular tissue, connective tissue, and occasionally the visceral organs, characterised by the presence of spindle-shaped bodies, usually embedded in the sarcous tissue of an elementary fibre, or occasionally lying free amongst the elementary muscular fibres or in the connective tissue. The spindle-shaped bodies, or sarcocysts, are protozoal parasites, belonging to the *order* Sarcosporidia of the *Subphylum* Sporozoa. The disease is thought to operate by a toxin, to which Laveran and Mesnil have given the name *sarcocystin*.

History.—In 1843 Miescher, of Bâle, described peculiar worm-like bodies, which he termed "utricles," in the muscles of the mouse. In 1851 Herbst found similar corpuscles in those of the pig. In 1853 von Hessling, who had already, in 1846, observed them in the muscles of the breast of a roebuck, encountered them in the muscular heart-substance of the ox and sheep. In 1857 Rainey described similar structures taken from the flesh of swine; but it was not until 1865 that this sarcosporidium attracted much serious attention. In that year cattle plague was prevalent in Great Britain, and a panic, originating with the outbreak of trichiniasis in Germany, roused the public to the danger arising from the consumption of meat. In nearly, if not all, of the cattle dying of rinderpest, these utricles of Miescher, or bodies or corpuscles of Rainey, were found, and gave rise to a great deal of speculation as to their nature. Cobbold, in 1866, demonstrated that they were parasitic protozoa, or psorosperms, devoid of harmfulness for man. Since that period a great deal of attention has been, from time to time, given to the pathology and natural history of these sarcosporidia, which are now known to occur in every species of domestic animal, in man, and in various mammals and birds, and even in reptiles.

Species Affected.—The following table gives the species of animal known to be affected by these sporozoal parasites:

GENUS SARCOCYSTIS.

Name of Sporozoön.	Habitat.	Host.
		Mammalian:
S. Blanchardi.	Muscles.	Ox, Javan and European buffaloes.
S. Bertrami.	Muscles.	Horse, ass, and zebra.
S. cameli (Mason, 1910).	Muscles.	Camel.
S. immitis.	Liver.	Man.
S. Lindemanni.	Muscles.	Man.
S. Mischeri.	Muscles and connective tissue.	Pig, ox, sheep, and mouse.
S. mucosa.	Connective tissue.	Kangaroo, man.
S. muris (Blanchard).	Muscles.	Mouse and guinea-pig.
S. Siamensis.	Muscles.	Siamese buffalo.
S. tenella.	Muscles and connective tissue.	Sheep, goat, and man.
S. *sp.*	Muscles and connective tissue.	Llama.
S. *sp.* (von Hessling).	Muscles.	Guinea-pig.
S. *sp.* (de Jongh).	Muscles.	Roebuck, goat.
S. *sp.* (Krause).	Muscles.	Dog, cat.
S. *sp.* (Manz).	Muscles.	Rabbit.
S. *sp.* (Hardenberg).	Muscles.	Hare.
S. *sp.* (von Siebold).	Muscles.	Rat.
S. *sp.* (Besnoit and Robin, 1912).	Skin.	Ox.
		Avian:
S. falcatula.	Intermuscular connective tissue.	Habia Ludoviciana.
S. Rileyi.	Intermuscular connective tissue.	Anas boschas, A. clypeata.
S. *sp.* (Kühn, Stiles).	Muscles and connective tissue.	Gallus domesticus.
S. *sp.* (Barrows).	Muscles.	Parula pitiayumi.
S. *sp.* (Stiles).	Muscles.	Setophaga ruticilla.
S. *sp.*	Connective tissue.	Turdus merula.
		Reptilian:
S. platydactyli.	—	Tarentola mauritanica (Gecko).
S. *sp.* (Lühe).	—	Lacerta muralis.

Protozoölogy.—The various species of protozoal parasites causing sarcosporidiosis belong to the *order* Sarcosporidia of the *class* Neosporidia of the *Subphylum* Sporozoa.

The sarcosporidia are closely related with the microsporidia or cryptocystes, by the fact that the spore-formation continues the whole of the trophic stage, that they are cell parasites, and that the spores have a single polar capsule.

In the sarcosporidia the trophozoite is an elongated, motionless body, circumscribed at first by a delicate membrane, later by a thick envelope, having a complicated structure. Spore-formation commences at an early stage, and continues during the growth of the trophozoite, which may obtain a great size. The spores are produced in great numbers, and are minute, banana-shaped, sickle-shaped, or spindle-shaped bodies, surrounded by a very delicate membrane, containing the sporoplasm, with a single nucleus, and an oval, striated body, placed at the pole of the spore and resembling a polar capsule. In certain instances apparently naked gymnospores are produced, probably serving for multiplication of the parasite within the host.

This order has only one *genus*, Sarcocystis. The genera Miescheria and Balbiania, introduced by Blanchard, and adopted by others, but only provisionally by Neumann. Laveran and Mesnil, Calkins, Minchin, and others, have shown that the characters by which they were differentiated only denote stages in the life-history of the parasites included in the unique *genus* Sarcocystis, founded by E. Ray Lankester in 1882.

In the earliest stage the parasite within the substance of the muscle fibre appears in the form of an elongated body, known as a "Miescher's tube." It gradually grows, until it distends the fibre to five or ten times its normal breadth, absorbing the contractile substance during that process, until it is eventually surrounded only by the sarcolemma and sarcoplasm, the latter increasing in amount, and its nuclei multiplying by direct division. The parasite then escapes into the adjacent connective tissue, still surrounded by a secondary covering derived from the muscle fibre, in which the nuclei disappear. Therefore, the parasite has two phases—the first located in the muscles, and the second in the connective tissue. In consequence of these two phases, Blanchard and others thought that they represented different genera, and hence to the first phase Miescheria, and to the second Balbiania, were given.

In the second phase the parasite circumscribes itself more, and encases itself in a rounded cyst, formed of the tissues of the host.

Morphology and Evolution.—The trophozoite, in its earliest stage, may attain a length of 40 μ by 6 μ in breadth. It appears as a minute, whitish, opaque body, embedded in the substance of the muscle fibre, and is surrounded by a fine, structureless, or homogeneous, membrane. The protoplasm of the trophozoite becomes divided, and gives rise to a number of nucleated corpuscles or cells, to which the name of "sporoblast," primitive sphere," or "pansporoblast," has been given.

During the next intramuscular stage the sarcosporidium increases in size, and becomes encased by two coverings. The outer covering is thick, and has fine radiate striations. Some regard this striation as due to the presence of fine canaliculi, running through a clear membrane; others to a thick fur of fine filaments, fixed vertically to the surface of the trophozoite, and acting as an attachment to the fibrillæ of the muscle fibres. The inner covering is a thin, homogeneous membrane, extending externally into the filaments and internally into the system of chambers. Within these two coverings the protoplasmic body of the trophozoites is contained, and consists almost entirely of numerous primitive spheres, which are constantly being formed at the two poles of the trophozoite. Between the primitive spheres intersections extend from the inner covering, separating them from one another, and forming a honeycomb structure, so that each alveolus contains a primitive sphere. Collections of spores, or other germs, are formed towards the centre of each primitive sphere, and completely fill the alveolus. Primitive spheres, or early stages in the course of sporulation, are found towards the poles, so that these two extremities are occupied in forming new spores. The final stage is occupied by the sarcosporidium encysting itself in the connective tissue. The body of the parasite becomes rounded off, and the two sporulative extremities now circumscribe the trophozoite, forming an outer zone, which contains small alveoli, in which are found elements undergoing formation. Within this outer zone other alveoli are found, containing an abundance of mature spores. In the middle of the body a granular material is observed, which arises from a breaking up of the most centrally-placed spores which have run their cycle, died, and are undergoing disintegration. The sarcosporidium still undergoes development within the cyst, and fresh spores arise towards the outer zone to take the place of those which die off towards the centre. The primitive spheres have at first one nucleus, and later on several.

There are two kinds of reproductive bodies which arise within the alveoli of the primitive spheres—chlamydospores and gymnospores.

The chlamydospores are minute, rod-like bodies, which vary in structure, form, and in the arrangement of their chromatin and polar capsule in different species.

In *Sarcocystis tenella* of the sheep the chlamydospores are about 14 μ in length by 3 μ in breadth. One extremity is obtuse, and the other pointed; they are very fragile and delicate, and their membrane very thin. The pointed extremity contains a clear space, in which, in a fresh preparation, a delicate spiral striation can be seen. The rounded extremity of the spore is almost filled by a large oval nucleus, in which a nuclear corpuscle can be observed. The middle part of the spore contains a protoplasm, with coarse granules. Some authorities, and Wasielewski among them, assert that a filament is extruded from the capsule, as in the polar capsule of the myxosporidial spore; but this has not been observed by such authorities as Laveran and Mesnil.

The gymnospores, also called "sporozoites," and "Rainey's corpuscles," falciform bodies, banana-like, sickle-like, crescent-like, bean-shaped, or kidney-shaped bodies, vary also in shape and size. According to Watson, the crescentic spores of sarcocystis bear a striking resemblance to the "crescentic bodies" described as developmental forms of trypanosomata.

In *S. tenella* of the sheep the sarcocysts may grow to a large size, even to 2 inches in length in the muscle fibres, where they eventually burst, the spores being spread or carried by the alimentary canal, or by the circulation, to various parts of the muscular system, and set up a fresh infection. In some instances the whole body becomes literally infested with sarcosporidia, and in such the animal often succumbs. Probat, of Muansa, and Francis, of Texas, have observed what they took to be free spores of sarcosporidia in the blood of animals.

Histological examination of the lesions in the case of cuticular sarcosporidiosis in the ox observed by Besnoit and Robin showed the presence of a sarcocyst provided with a fine nucleated enveloping membrane lining the internal face of the cuticle, and an absolute absence of flagella on the sporozoites. The corpuscles varied from 5 to 8 μ in length, by 2 μ in breadth. There were also circular or oval, distinctly circumscribed granular masses, from 12 to 20 μ, present in the lesions. These authors consider this sarcocystis to be a new species distinct from *Sarcocystis Blanchardi*, which attacks the muscle fibres, and is provided with a markedly striated enveloping membrane.

The preparations are fixed with ethylic alcohol or Schaudinn. Besnoit and Robin recommend hæmatenia-eosine, and especially by the prolonged action of two hours of Giemsa.

Etiology.—It is not known by what method sarcosporidia gain the system in a state of nature. Theobald Smith, however, was successful in transmitting, experimentally, *S. muris* to the mouse by feeding it on mice dead of the infection. Darling has shown that guinea-pigs can be infected by feeding them with rat's muscle naturally infected by *S. muris*, and by ripe mobile sporozoites from the same source. The period of incubation in guinea-pigs varied from 152 to 164 days. This fact seems to show that the parasite gains the system through the alimentary canal. This view is further supported by the fact that in some animals, notably the sheep, goat, deer, ox, buffalo, and pig, the œsophagus seems to be the primary seat of the parasites, other parts, such as the muscles at the base of the tongue, pharynx, cheek, neck, thorax, abdomen, and thighs, being secondarily invaded. As this sarcosporidial infestation is frequently discovered on post-mortem examination of animals or man, dead of certain acute infectious processes, such as rinderpest or typhoid fever, the question arises, Does this infestation arise *per se*, or is it an affection arising in a system already weakened by some primary malady ?

It is not known if the presence of the parasites operate by the irritation they set up, or by the toxin found in their substance. The experimental evidence afforded by inoculation of the toxin denominated by Laveran and Mesnil "sarcocystin," rather goes to show that the toxic symptoms produced are rarely observed under natural circumstances, and the generally mild clinical aspects under such conditions lead one to suppose that the diminution of the muscular function is due to mechanical rather than toxic disturbance.

Pfeiffer pointed out that an aqueous extract of the sarcosporidia, when injected under the skin of the rabbit, produced symptoms of diarrhœa, paralysis, insensibility, and a subnormal temperature, followed by death within seven or eight hours. Laveran and Mesnil repeated the researches of Pfeiffer, and demonstrated in *S. tenella* of sheep a toxin to which they gave the name of "sarcocystin." These two authorities made an aqueous and a glycerinated extract. The aqueous extract soon lost its activity, which was very much lowered at the end of a week; whereas the glycerinated extract preserved its activity much longer, and was toxic for at least a month. The broken parasites, when inserted under the skin, acted in a similar manner to the aqueous or glycerinated extract; but if the covering of the parasite was intact, toxic phenomena were slow in manifestation. A dry extract was shown by Laveran and Mesnil to be very active.

The sarcocystin, although very toxic for rabbits and white mice, is almost inert on other animals. The sarcocystin prepared from the sarcosporidium of the Hungarian buffalo or of the llama produced on the rabbit phenomena similar to those set up by that prepared from the sarcosporidium of the sheep. Sabrezès and Muratel found the same for the sarcosporidium of the horse as did Besnoit and Robin that of the ox. The aqueous extract becomes inert after being submitted for five minutes to a temperature of 100° C., or for twenty minutes to 85° C. The glycerinated extract resists heat much better. When raised to a temperature 85° C. for half an hour, it was still capable of killing a rabbit if large doese were used.

Ingestion of the aqueous extract did not produce any ill effects.

The parasites produce serious symptoms, and often great mortality, when there are myriads of cysts, which burst and scatter their contents in the surrounding tissues, destroying the muscles and setting up abscesses, which contain numerous parasites, the lesions thereby simulating those occurring in the *Myxoboliasis tuberosa* of barbel, in which there is often a diffuse infiltration of the purulent process (*vide* p. 1126).

It is not known if there is an intermediary host, but it is quite certain that ingestion may be the mode of entry of the parasite into the system, and that, in some animals (mouse) at least, there is no need for an intermediary host.

Clinical Manifestations in the Horse.—This disease is rarely diagnosed during life, and probably it is generally overlooked after death by the practitioner. It is mostly observed in subjects for dissection.

Hendrickx and Liénaux, of Brussels, in 1899 encountered a case of lingual sarcosporidiosis in a four-year-old cart-horse. A few weeks before they saw the animal the owner observed that there was wasting away in condition and a great difficulty in seizing food. When it came under the authorities' observation, the upper lip was palpably swollen at least to three times its normal size, and rigid. In spite of this, the organ had preserved a certain degree of its mobility. The outer surface of the skin of the lip showed nothing in particular, but the internal face, instead of being glistening or smooth and evenly concave, was irregular, and studded over with hard nodules, the size of a hemp-seed to that of a pea, giving a relief to the surface and presenting a yellowish-grey colour. The swelling of the lip was insensibly confounded with the neighbouring parts without any well-marked line of demarcation.

The mouth was incompletely closed, and from each labial commissure long strings of saliva flowed. On inspection of the mouth, the free portion of the tongue was swollen to at least four times its natural size, and markedly distending beyond the lingual canal. On direct palpation, the tongue was hard, resembling condensed fibrous tissue ; yet in spite of this it could still be retracted fairly well ; the lateral movements were very difficult. The sensibility of the organ did not appear to have undergone any change. The two lateral faces and the under surface of the free portion, instead of being smooth and regular, offered a mammillated or irregular aspect due to the existence of small nodules under the mucous membrane raising the surface. In several points there were superficial solutions of continuity covered with pinkish granulation tissue, probably caused by the traumatic action of the teeth or the food. The posterior part of the tongue did not show any alteration. Beyond these symptoms, and the animal being tucked up and the hairs long and dry, no other anomalies were observed. Microscopical examination of a fragment of the morbid tissue showed that the muscular fibres of the tongue contained well-preserved sarcosporidia. The fibres, in spite of their contents, were little changed; the inflammatory nodules or false tubercles in most instances had undergone in their centre a caseo-calcareous alteration, and contained the remains of sarcosporidia.

In the case of a four-year-old horse, observed during life by Moussu and Coquot, there was at first a swelling, without special characters, in the region of the withers; this was soon followed by similar swellings on various parts of the body, ending in completely disfiguring the animal. These tumours appeared as large nodules or salient subcutaneous patches, which were hard, woody, painless, and always more or less adherent to the skin or deeper tissues. The tongue, in its turn, became enormous, hard, painless, and offering all the characters of "wooden tongue." The animal's gait was stiff, the limbs were as rigid as posts, the general state was good, and the appetite was preserved, even though there was a difficulty in seizing the food. Removal of one of the tumours and its microscopical examination enabled the diagnosis of sarcosporidiosis to be made.

Sabrazès and Muratel encountered a case of a four-year-old horse that had been suffering for some months, and became in consequence very much emaciated. The under surface of the chest was disfigured by several tumours, ranging from the size of a pigeon's egg up to that of an ostrich's egg. In this region they seemed to be embedded in the deeper subcutaneous tissues. Several similar tumours formed a ring round the metacarpal region, where the periosteal membrane gave off bony stalactic processes. In the centre of the neoplasmata, and also in their immediate surrounding tissues, encysted sarcosporidia were found scattered about in the muscular fibre.

These authors, along with Marchal, considered the tumours arose from chronic irritation of the tissues, set up by the sarcosporidia or their secretion.

Schultze observed paralysis of the fore-limbs in one case in which, on post-mortem examination, the muscles of the fore-quarters had undergone degeneration, due to the presence of sarcosporidia.

Watson encountered several cases of sarcosporidiosis associated with "loco-disease" and "dourine" in Canada. He suggests certain symptoms, usually attributed to "loco-disease," as being in reality those of sarcosporidiosis—viz., various forms of mental derangement, from mania in the first stages, to a dense stupor or comatose condition in the later; disorder of the muscular system, from increase reflexes to spasms, atrophy, and paralysis; osseous enlargements or degeneration, especially of the jaws and parts of the skull; impaired vision, depravity, emaciation, etc. Cramps are not infrequent: a limb is slowly extended as far as possible, remaining in that position for a short period, and then gradually, and extremely flexed, held high above the ground for a few minutes, the horse moving with a limp.

Clinical Symptoms in the Ox.—Although sarcosporidiosis has frequently been observed on post-mortem examination of oxen dead of acute infectious processes, such as rinderpest, very few symptoms have been noticed during the life of the beast to lead one to suspect its presence.

Watson, however, has observed frequent prolonged extensions of the head and neck, accompanied by a more or less constant trembling and agitation of the muscles of these parts, especially of the jaws, and most marked during attempts at feeding, the power of prehension of food being practically lost, and mastication very difficult and incomplete. There may be a slight serous discharge from nostrils, dimmed eyes, and occasional lachrymation. The animals gradually get worse—emaciated, sluggish, difficulty of progression, and stiff and slow muscular action. Tokarenko and also Brouwier have recorded similar symptoms arising in cattle affected by sarcosporidiosis.

Besnoit and Robin studied a case of cutaneous sarcosporidiosis in a ten-year-old Pyrenean cow in an indifferent condition, although the appetite was excellent. There were partial depilations varying in extent on the upper part of the body, from neck to base of tail. The skin was thickened and covered with whitish flaxen scales, resembling those seen in discrete pityriasis. The udder was completely dry, very small and shrunken ; the skin covering it was thick and hard ; the teats were drawn to one another by induration of the skin, and appeared as if adherent at their base by lateral pairs. On the inferior part of the organ, particularly on the teats, irregular elevations, varying in size from that of a filbert to that of a pigeon's egg, generally separated by cracks, were observed, at the bottom of which the integument was bright-red and slightly oozing. On the larger nodules the skin was pink and normal, but in the vicinity of the smaller ones and on plane surfaces it was thick and covered with whitish, bran-like scales, similar to those mentioned above. The hind-limbs, from the middle of the thigh to the coronets, had the skin reddened, depilated, indurated, and sometimes disposed in the form of transverse cords, the size of the thumb. The general aspect resembled that of grease with "grapes." As in that affection, the lesions were especially marked on the postero-external face of the metatarsal and fetlock regions; they also appeared towards the point and external face of the hock; they were more discrete on the internal face of the limb, as compared with those on the udder.

The fore-limbs were similarly affected from the lower part of forearm down to the coronet, and by preference on the posterior and external surfaces of these regions. A pinkish and hairless nodule of the size of a walnut was present on the skin of the front of the coronet, just above the interdigital space.

The skin of the dewlap was likewise changed, in its inferior part at least. In the vicinity of the tracheal appendix of the sternum a hardened mass of the size of two fists and non-fluctuating was found. It seemed to have developed in the integument, and not adhered to the subjacent tissues, and its surface was ulcerated. Similar warty lesions were found on the head, particularly round the nostrils and on the brow, as well as around the eyelids, especially towards the nasal angle of the eye. Finally, at the ilial and ischial points, the skin was considerably thickened, and showing lesions similar to those on the limbs, but less marked.

It appears that no clinical account of sarcosporidiosis in the camel, buffalo, or llama has been recorded.

Clinical Symptoms in the Sheep and Goat.—No clinical manifestations are present unless the subcutaneous or muscular tissues are widely invaded, when one may observe emaciation, stiffness, and perhaps a difficulty in deglutition. It is said that epizoötics, accompanied by great losses, have arisen when the parasites have occupied the larynx and set up œdema of the glottis, and consequently asphyxia. Some assert that they cause sudden death or epileptiform seizures. This seems very feasible, especially during the wandering stage of the parasite before it has become encysted.

Clinical Symptoms in the Pig.—When the parasites are very numerous, and set up a diffuse purulent infiltration, there may be paralytic symptoms of the limbs, or muscular stiffness, great dulness, loss of appetite, a high temperature, an eruption on the skin, increased thirst, and catarrhal discharge from the eyes ; but there is nothing characteristic in these symptoms, which may also appear in swine erysipelas, swine fever, measles, etc. Trichinosis is frequently associated with sarcosporidiosis.

Clinical Symptoms in the Mouse.—In this creature the *S. muris* sets up a very fatal malady, which is evidenced by a puffy, bloated appearance. After ingestion of the parasite the period of incubation may be as long as six or seven weeks.

Lesions and Prevalence.—All the striped muscles may be infested by sarcosporidia. The muscles of the œsophagus are the commonest seat of the parasite, afterwards those of the larynx, skeleton, diaphragm, and the psoas muscles ; they are also found frequently in the cardiac muscle and sometimes in the eye muscles, and also in the skin.

The parasites may, on close examination, appear as small whitish points in the muscular fibres. When in the muscle fibre, they grow until the primary fibre reaches five to ten times its normal breadth, absorbing the contractile substance in the process. Eventually it becomes encased only by the sarcolemma and sarcoplasm, the latter having greatly increased in amount, its nuclei mutiplying by direct division.

According to Alexeieff, the cyst envelope (the *S. tenella*, Railliet) belongs to the host. The parasite is naked, and always remains intracellular. In the systemic muscles, in which Moulé has found it in 44 per cent. of fat sheep, and in 98 per cent. of those in a cachectic state, it measures on an average 0·5 millimetre in length, by 60 to 100 μ in width. It has also been found in the fibres of Purkinje of the heart and of the muscles of the eye.

In the *goat* Moulé found it in 33 per cent. of fat animals, and in 46 per cent. of those in a lean or cachectic condition. The sarcosporidium is wider and larger than in the sheep, provided with a thicker, more resistant, and more distinctly striated cuticle. It is more difficult to isolate from the primary fascicle, so much more so as the muscular fibres are much more constricted and less distended with fluid, even in the cachectic animal.

In the connective tissue of the œsophagus the sarcocysts are so large that they cannot escape attention. They may vary in size from a grain of millet to a filbert. These large forms of the parasite, formerly considered to belong to a distinct species, and in consequence termed *Balbiania gigantea*, have also been found at the base of the tongue, in the muscles of the pharynx, cheeks, lips, neck, elbow, thorax, shoulder, abdomen, thighs, and in the fleshy portions of the diaphragm, and even in the cuticularis. Sometimes it is encountered under the pleura, peritoneum, and pericardium. Zürn has seen it twice under the cerebral dura mater in the sheep. At first sight they have the appearance of a small abscess, or a mass of fat, but on microscopical examination of the milky or caseous contents they contain a great number of corpuscles of various sizes. Those found under the serosa are generally longer than those in other regions of the body. On section of a whitish nodule one finds a very fine transparent limiting membrane, which contains within it intersections, representing the spores of various dimensions. In the larger specimens the central part has a vacuole of the size of a pin's head. The intersections near the circumference are fitted with very regular falciform corpuscles, with or without two refringent points, and sometimes mixed with circumscribed corpuscles.

Morot found 272 out of 900 sheep in good condition killed at the Troyes abattoir; these showed nodules in the œsophagus, and in this total number affected 6 also showed nodules under the pleura, 10 under the peritoneum, and 27 both under the pleura and peritoneum. The existence and abundance of these appeared to be absolutely independent of good condition, age, and sex. A ewe, aged between eight and ten years, in good condition, had a great number of these parasites in every part of the body, and in particular 227 in the œsophagus and 128 in the tongue. Fürstenberg alleged that they were found in every sheep imported into Germany from France; but although they appear to be common at Troyes, and somewhat frequent at Rheims, they are rarely encountered in Paris. Bergmann, at the Stockholm abattoir, failed to discover sarcosporidia in the œsophagus of sheep, although he carefully examined 111 animals. Wasielewski's statement, that they are regularly encountered in sheep, therefore requires to be regarded with some reserve. These œsophageal parasites have been found in the goat.

In the *ox* the sarcocysts vary in size from 5 millimetres to 1 centimetre in length. Its cuticle is fairly thick, distinctly striated, and gives off ciliæ.

Moulé found them in 6 per cent. of oxen in good condition, and in 37 per cent. of those condemned for extreme emaciation. During the cattle plague outbreak of 1865-66 they were almost without exception encountered in oxen dead of that malady by Axe, Cobbold, Beale, Sir George Brown, Gamgee, Professor Simonds, aad numerous others. Watson has encountered them in cattle suspected of loco-poisoning in Canada.

Bergmann found them in the œsophagus of the ox in 28 out of 166 slaughtered oxen. They were somewhat rare, not more than one being found in any one œsophagus. They had invaded both the outer and inner muscular layers. They measured from 0·7 to 7·7 millimetres in length. He also found them in the œsophagus of a deer. It was, however, present in such numbers that the œsophagus appeared striped with white. They have also been observed in buffalo in various parts of the world.

In the *camel* the œsophagus appears to be the earliest seat of the parasite. According to F. Eugene Mason, it is a widespread disease of aged camels in Egypt. It is fairly frequent in the heart, gluteal region, less numerous in the diaphragm; it was found also in the larynx, head, tongue, neck, thorax, abdomen, sublumbar and dorsal regions, arm, forearm, thigh, leg, and tail.

In the *horse* the sarcocysts resemble those of the ox. Moulé scarcely found 6 per cent. in horses when in good condition, and 20 per cent. when in an emaciated state. Sabrazès and Muratel found 90 per cent. of the horses at the Bordeaux abattoir to be affected; the muscular coats of the œsophagus appeared to be the seat of predilection. According to Friedberger and Fröhner, they are found almost in every aged animal. Siedamgrotzky, of the Dresden Veterinary School, found them in thirteen horses killed for anatomical dissection. In the case diagnosed during life by Moussu and Cuquot, beyond the clinical appearances already mentioned, these authors found, on post-mortem examination, the majority of the muscles were invaded, but especially in the cuticularis, where the lesions were intense. Out of sixty-five horses examined by Moulé, thirteen showed sarcosporidia. In only one case was the sarcocyst as large as that seen in the ox; in the other cases they were invisible to the naked eye. Bergmann, in Stockholm, found, out of nine horses killed for dissection, four were affected with sarcocysts, which were situated in the inner and outer muscular layers of the œsophagus. A few were found in the diaphragm; they measured 1·5 c.c. in length, and 0·75 millimetres in breadth. Watson found five cases in horses suspected of loco-poisoning or affected with *Trypanosoma equiperdum*, in which widespread sarcosporidiosis was observed after death.

In Siedamgrotzky's dissection subjects, certain muscles of the limbs were pallid and atrophied, and also manifested whitish striæ, corresponding to the long tubes of Miescher, averaging from 3 to 4 millimetres. The nuclei of the sarcolemma were multiplied, not only in the invaded primitive fasciculi, but also in the neighbouring ones. Furthermore, the interfascicular connective tissue had proliferated and, by compression, determined simple atrophy of the contractile fibre. The parasites had even, in certain points, undergone calcareous degeneration. Laulanié has observed similar instances. Pütz has observed in a horse the majority of the muscles occupied by sarcocysts; they were very voluminous, rigid, and of a yellowish tint. This pseudo-hypertrophy was due, as recognised by Schulze, Rabe, and Jöhne, to a chronic interstitial myositis, set up by sarcosporidia. Kitt has recorded a similar case.

In the *pig* the *S. Miescheri* is an elongated body, slightly refringent, granular, and tapering at both ends, usually more at one than the other. It rarely reaches from 2 to 3 millimetres in length, by 200 to 300 μ in breadth. The tubes have a firm cuticle, thick, and showing transverse striations, attributed by Leuckart to the presence of numerous canaliculi; by compression, this wall disintegrates, and then has the appearance of a hairy vestment. The internal cavity is occupied by a variable number of spheres from 25 to 50 μ in diameter, becoming polygonal by even pressure. At the extremities there remains a clean space, occupied only by refringent granules. In each of these spheres, which appear to represent the spores, numerous corpuscles are formed; these are at first rounded, and then become banana-shaped, showing two very brilliant vacuoles, and becoming also mixed with granules. All the striated muscles may contain the parasite, which can sometimes be seen with the naked eye in the form of small, whitish streaks. It is situated in the interior of the primitive fasciculus. In many cases the fasciculi are simply distended in their middle, but very often by a multiplication of the nuclei of the sarcolemma. Sometimes two of these parasites are in contact with one another. Laulanié has recorded a case in which the muscular tissue was riddled with yellowish fusiform granulations, often disposed in series of two or three in the direction of the muscular fibres. By separation of these small masses, purulent oil globules, calcareous granules, and rarely epithelioid cells, could be obtained. A few of them were situated in the interior of the secondary fasciculi, but the majority developed in the neighbourhood of the connective spaces surrounding these fasciculi. They were composed of a more or less degenerative central zone, and a proliferative peripheral zone, closely resembling a tubercle. The central zone was more often formed by a mass of pus globules, having undergone caseous degeneration, and often even calcareous infiltration. The zone of proliferation was constituted by embryonic and a few epithelioid cells, extending to the periphery between the primitive fasciculi, which were found incorporated and destroyed in places. In Hendrickx and Liénaux's case in the horse the nodules had similar characters.

The prevalence of sarcosporidiosis in the pig varies according to locality and, perhaps, season. According to Bergmann, of Stockholm, the greatest number were found in August, September, and October—viz., 47·39, 83·88, and 36·15 per cent., in the southern regions, and 30·25, 29·82, and 34·75 per cent. in the northern regions controlled by the Swedish Board of Agriculture. The average percentage during the year was 30·62 and 27·59 for the districts respectively. Koch gives the percentage of pigs affected as 8, Perroncito 25, Leuckart 28, Moulé 40, Herbst and Rupprecht 50, and Kühn 98·5. In Great Britain and Ireland statistics are not available. The disease, however, certainly exists.

According to Bergmann the early summer is the most favourable time for invasion with sarcosporidia in Stockholm. Of twenty-five pigs reared in a well-managed piggery near Stockholm, all were found affected with sarcosporidia, eighteen with tuberculosis, two with trichinæ, and one with tuberculosis and trichinæ. This authority also examined twenty-nine rats found in the slaughterhouse of this establishment and its neighbourhood, and discovered sarcosporidia in two, and trichinæ in one.

Diagnosis.—Diagnosis during life is possible, but as the disease is rarely suspected, means are not adopted for its detection. Hendrickx and Liénaux, Moussu and Coquot, and also Watson, have excised a portion of muscle or lesion from an animal during life and detected sarcosporidia. Harpoonage would be a less heroic method. It should be made in several situations, until a portion of muscular tissue containing the parasite was obtained. Probably many cases of obscure stiffness and unthriftiness in certain animals will be shown to be due to sarcocysts. It should be remembered, where acute cases of obscure disease in animals occur, the carcasses rarely find their way to an abattoir, and that the muscular tissue is not very often submitted to a rigid examination.

Sarcocysts may be mistaken for trichinosis, cysticercosis, distomatosis, the miliary forms of tuberculosis, botriomycosis, actinomycosis, etc.; but such mistakes may be evaded by microscopical examination of the affected tissues.

Public Health.—Theobald Smith and also Koch were successful in transmitting *S. muris* to mice, and Darling and also Negri to the guinea-pig, by feeding them on the flesh of mice dead of sarcosporidiosis. In Smith and Koch's cases the period of incubation was over six weeks. Leuckart was successful in conveying the *S. Miescheri* to the pig in one case; but this isolated instance is not conclusive enough. Virchow and also Manz could not transmit the sarcocysts of the pig to healthy cats, rabbits, guinea-pigs, rats, and mice. Manz affirms that the sarcocysts are destroyed by the gastro-intestinal juices, without leaving a trace in the intestines or in the muscles. Besnoit and Robin failed to infest a cow with a cuticular sarcocyst of a cow, although they tried inoculation by scarification, subcutaneous insertion of a piece of tissue, by Thiersch's grafting method, and intramammary injection of an emulsion. Cobbold, at two meals made out of a sheep's heart intensely infested with *S. tenella*, calculated that he could not have swallowed less than 18,000 sarcocysts. He says: "The fact is, I believe, that lovers of beef, mutton, and pork eat these bodies every day, but take no harm."

Darling described a case of sarcosporidiosis in a Barbadian negro, who had passed through an attack of typhoid fever, complicated with necrosis and myositis of some of the striated muscles. Portions of the biceps removed showed the presence of sporozoa. About three weeks later the sporozoa had disappeared, the parasite apparently being abortive. Morphologically the sarcocystis derived from the negro was identical to that found in the mouse. Brooks considers the eland, caribou, and deer infect man.

Vuillemin believes that systematic researches will show that the sarcocyst is a frequent parasite in man. Lindemann, Hadden, Klebs and Eve, Rosenberg, Baraban and St. Rémy, Hoche, and Kartulis have reported cases of its occurrence.

Gastro-enteric catarrh in man has been attributed to eating the flesh of animals intensely infested with sarcocysts, but no proof is forthcoming to show that sarcocysts or their toxin, sarcocystin, were the cause. Meat being intensely infested with sarcocysts, especially when of inferior quality, should be condemned as unfit food for man.

Treatment.—As this disease is rarely suspected or diagnosed during life, very little treatment has been adopted. Hendrickx and Liénaux had complete recovery following daily doses of 2½ drachms of potassium iodide for thirty-six days. In addition, the tongue was washed four times a day with a 2 per cent. solution of carbolic acid. The animal was three months in the infirmary. In the case recorded by Moussu and Coquot the horse was submitted to a course of iodide of potassium; but although it improved in condition, as soon as one lot of nodules disappeared another lot appeared, and in consequence it was deemed advisable to slaughter the animal.

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V. HAPLOSPORIDIA.

This *order* has for its characteristics a very simple developmental cycle, in which the youngest stage of the parasite is a minute rounded corpuscle, with a single nucleus. It has three *families : Haplosporidiidæ,* in which the amœboid-like parasites are reproduced by encapsulated merozoites, which may or may not possess spines or processes ; *Bertramiidæ,* with two genera—Bertramia and Ichthyosporidium —and *Celosporiidæ.*

Beyond the *genus* Ichthyosporidium there is no other genera in this order that includes species of sporozoa as fish parasites.

Caullery and Mesnil consider this order of protozoal parasites is closely connected with Microsporidia and Sarcosporidia, which all three are characterised by the absence of a polar capsule in the sporozoites.

The Lymphosporidium of Calkins is provisionally included by Minchin in the *order* Serosporidia, and by Calkins in the *family* Celosporidiidæ.

O'Kinealy encountered in a man in Calcutta a vascular tumour in the septum nasi, which was caused by a sporozoal parasite named *Rhinosporidium Kinealyi.*

The tumour contained the parasites in a round cyst. The young parasites possessed a granular protoplasm surrounded by a hyaline membrane, and contained several small nuclei. Later on the enveloping membrane thickened, and the protoplasm separated to surround the nuclei to form sporoblasts. The sporoblasts became in their turn segmented to form sporozoites. By rupture of the cyst these sporozoites were set at liberty to form parasitical elements.

VI. LYMPHOSPORIDIOSIS IN THE TROUT.

Definition.—An enzoötic disease of the brook trout, due to a protozoal parasite, the *Lymphosporidium truttæ* (Calkins), and causing in them a great mortality.

History.—Calkins, of the Columbia University, in 1899 studied this disease, causing a great mortality among the brook trout (*Salvelinus fontinalis*) of a private hatchery on Long Island, New York. From the middle of May to October, 1899, the fish were dying by thousands, and it appeared that every fish in the hatchery was doomed. Twelve years previously a disastrous enzoötic had carried away most of the brook in a neighbouring hatchery. The affected fish had similar symptoms to the 1899 outbreak.

Etiology.—From the life-history of the parasite while within the body of the fish, it appears that the protozoön gets into the fish probably by means of the digestive tract and while in the spore stage. The parasite may be present in small numbers in the trout at all times, becoming epizoötic only under unfavourable circumstances. Possibly there may be an intermediate host, but nothing is definitely known about the life-cycle or the means by which the parasite gains an entrance to the system of the fish.

Symptoms.—When the disease was first noticed in May, a dead young fish was observed to have a small, clean-cut hole in the side. Others were observed from time to time with similar holes. In October the fish appeared sluggish, and to have lost considerable vitality. They were unable to withstand handling of the gentlest kind, and hundreds of them died every day. The dead ones floated to the surface of the water. Some of these had no external marks whatever to indicate the disease, but were plump and apparently sound, the cause of death apparently being some internal organic trouble. Others, however, had numerous round, clear-cut holes or ulcers in the sides or back; in some great patches of the skin and underlying muscular tissue had fallen out, leaving large, irregular, but shallow, pits, sometimes 2 or 3 square inches in area; in others the flesh had fallen away in spots, leaving the backbone visible; and in some others the clear-cut holes went entirely through the body wall, and not infrequently the internal organs—*e.g.*, the testis or liver could be seen, even in the living fish, hanging out of the openings thus made. Again, the eyes would be entirely gone, or the lower jaw would be eaten away, and many instances were seen in which the tail and one or more of the fins had disappeared.

Lesions.—Various parts of the dead fish were fixed in Flemming's stronger fluid, sublimate acetic, sublimate, and Boveri's picro-acetic. Sections were made and stained in iron hæmatoxylin, Flemming's triple stain, and in thionin, with a counter-stain, eosin.

Innumerable quantities of spores, not aggregated in cysts, were found distributed throughout the body. There were no thread-bearing capsules.

In the intestine these spores formed large masses, together with the intestinal bacteria. They were found in the lymph-spaces surrounding the intestine, liver, kidney, and other organs of the viscera; in the muscle bundles of the body wall; in the mesenteries and connective tissue through the body; in the cavities of the gall- and swim-bladder, and in the lumina of bloodvessels. They were, in fact, universally distributed about the cavities and spaces of the diseased fish. They were not generally seen in glandular tissues, although they were occasionally met with in the kidney. The testis, however, seemed to be the main seat of the spores, and was literally crowded with them, especially in fish from 4 to 5 inches in length.

The spores are very small, about 2 to 3 μ, pyriform in shape, and of a homogeneous appearance. Their substance has a decided affinity for chromatin stains, especially saffranin, methyl green, etc. In the intestine and the testis the homogeneous appearance of the spores is lost, and various stages in the formation of exceedingly minute germs, which Calkins interpreted as sporozoites, are found. In consequence of the extremely small size of the spores, Calkins was unable to closely follow the process of their sporozoite formation. The spore material segregates into a relatively thick shell, about the circumference of the spore, but within the spore membrane. It then splits up into eight minute parts, all contained within the spore membrane, which at this period, for the first time, can be distinctly seen. The sporozoites then leave the spore, and in the intestine are lost sight of among the bacteria. In the testis they collect around the various lobes as minute spherical granules, which stain less intensely than the original spores. The empty spore-cases are frequently seen with a rupture or a crack at some part, through which the sporozoites make their exit.

The small rounded sporozoite grows until about the size of a spore, and at this stage appears very much like a spore, although it does not have the same densely homogeneous structure. It soon becomes amœboid, and, when slightly larger than a spore, is variable in shape. At this stage, or slightly later, the amœboid organisms begin to penetrate the muscular bundles surrounding the intestine and other organs. By amœboid movements they work their way between the muscle-cells, where they lie until mature growth is reached, and they are ready for spore-formation. In the adult parasite there is no well-defined nucleus. The body of the cell is well filled with distinctly staining granules, and for a long time the protozoa were overlooked, because of their striking resemblance to the nuclei of muscle-cells, which do not stain so intensely as these cells, which do not possess karyosomes or nucleoli, and are in a different position.

The spore-forming individuals are frequently found in the lymph surrounding the various organs, and are quite numerous in the cavity of the gall-bladder and in the intestine. They are of relatively large size—about 25 to 30 μ—and may be formed from the union of two or more individuals.

Treatment.—Nothing is known as to the prevention or cure of this fatal malady.

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VII. SAPROLEGNIOSIS OF FISH.

Synonyms.—Fungous or fungoid disease of fish; Saprolegnia of fish; Mouldiness of fish; Dermatomycosis saprolegniacea.

French: La maladie de la mousse.

German: Die Verpilzung der Fische durch Saprolegniacean; Dermatomykosis Saprolegniacea.

Definition.—A disease of fresh-water fish, characterised by the appearance on the external surface of the body of woolly- or fluffy-like masses of fungi, belonging to the family *Saprolegniaceæ* of the Phycomycetes or algal-like fungi.

Distribution.—This disease is widely distributed, as was shown in the article on the "Epizoötic Disease of Fresh-Water Fishes," and is commonly known as the "salmon disease." It appears in the clearest icy as well as in the slow-running waters, as well as in lakes, ponds, aquaria, and fish-tanks, not only in Europe, but also in Asia, America, and probably all over the world.

It occurs more particularly in ponds, and less frequently in quick-current rivers, where there are more numerous natural enemies to kill off the diseased or unfit, and thus to maintain the balance of Nature. As a general rule, fungous disease only appears in fish rendered weak by some depressing factor, such as an injury after spawning, living in contaminated water, or suffering from some virulent infection.

Etiology.—Until the last two decades, nearly every fungous appearance in fish was attributed to one species of saprolegnia—*Saprolegnia ferax*, or *ferox*, or *Thureti*. Still, C. L. Jackson and A. B. Stirling, more than thirty years ago, maintained that there were at least two different species capable of producing a fungous disease, but recent investigations by well-qualified botanists have shown that there are several different genera and species of fungi, but chiefly the genera Saprolegnia and Achlya having that function. These fungi mostly occur as saprophytes on dead organic matter, especially dead fish, salamanders, frogs, and other animal remains in fresh water. It is not known if they exist in salt water, but C. L. Jackson, Consulting Naturalist to the Southport Aquarium, in 1880 reported that a sea trout kept in a salt-water tank in that aquarium became affected with saprolegnia; yet previous to and about that period numerous practical observers noted that the affected fish lost their fungous appearance as soon as they gained the sea or where put into tanks containing salt water. This general view has been further supported by J. Hume Patterson, who in 1903 found that *Saprolegnia ferax* (*S. Thureti*) at least would not grow in salt water, and when fish affected with that fungus were put into salt water the fungus disappeared.

The development of this infestation depends on various circumstances, such as temperature and the resisting power of the fish's skin. On dead fish it may cover the skin in twenty-four hours, while in the live fish it may take days or weeks to spread to the same extent. Jackson, however, has seen perch shut up in a coop floating on Lake Windermere become smothered with it in two or three days. This rapid progress of the fungus was also frequently observed during the great epizoötic from 1877 to 1883. Goldfish and other fish not rarely become covered in twenty-four hours when travelling in tanks during changeable weather. Humphrey mentions a fungous disease attacking trout eggs in the Northampton Fish Hatchery, Massachusetts, in 1891. After the eggs were hatched, the young fry did not appear to be injured by the fungus. The hyphæ failed to develop sexual organs, but fresh cultures, obtained by throwing flies into the vessel containing them, produced a new crop, bearing sexual organs. If the dead eggs were not removed early and frequently, all the eggs in the hatching-trays would be killed, as the infection spreads rapidly.

It frequently occurs after spawning, when the fishes are very much run down Moore, of U.S.A., reported that 99 out of every 100, if not all, of the salmon in the upper tributaries of the Sacramento River appear to die from it immediately after their first spawning. Gerard, in 1878, reported a severe mortality among fishes alleged to have been due to *S. ferax*, in the Passaic River in New Jersey.

Fish suffering from furunculosis are sometimes very apt to become attacked by fungi. Why they do not always become attacked by the fungus when suffering from furunculosis seems inexplicable. Patterson found, by using pure cultures of *S. ferax*, he could not transmit the disease, although it grew luxuriantly on healthy dead fish or boiled sheep's liver in the tank containing the live fish. He could, however, induce it to grow readily as soon as he infected the fish with pure cultures of his liquefying bacillus—*Bacillus pestis salmonis*. Fungi will readily grow on the parts of skin denuded of its proper protective covering by rough handling, netting, etc. This, however, generally occurs in waters not highly oxygenated, as Jackson, by using highly-oxygenated water, banished fungous disease from the Stockport Aquarium, where it was hitherto the bane of the establishment.

Some species of fish seem more susceptible than others. Roach, dace, goldfish, gudgeon, and Crucian carp, which are very susceptible to the ordinary aquarium fungus, are somewhat resistant to *Saprolegnia Thureti* (*S. ferax*), to which tench, golden tench, trout, and char may readily succumb.

Where the saprolegniaceæ exist in great quantities, in consequence of the waters and atmospherical conditions being favourable for their rapid development, the waters become depleted of oxygen, and thus the fish become weakened and rendered an easy prey for the growth of the fungi.

In order to understand this disease, it is necessary for one to be acquainted with the different forms and the various means of propagation of the saprolegnei.

These fungi consist either of single or branched stems, terminating at either end in suckers, one end burying itself in the dermis of the fish, and the other end floating free.

It depends on the thickness of these hyphæ or branches as to whether the fungi are woolly, as in some of the Saprolegniæ, or star-shaped and almost prickly, as in some of the Achyla, whose hyphæ or branches have a diameter of nearly $\frac{1}{5}$ millimetre.

Their length varies, and in some cases reaches 3 centimetres, thus circumscribing the area of the disease, making it appear like long floating cotton-wool, the colour varying from white to grey, and in Saprolegnia can best be compared to unbleached cotton-wool.

The growth of the hyphæ and the field of their extent manifests itself by first growing lengthways only, then throwing off side branches, which in turn again branch and fasten themselves in the dermis of the fish.

The progress of the disease depends on various circumstances, such as the surrounding temperature, and the resisting power of the fish's skin.

On the whole, one's knowledge as to the rapidity of the spread of this disease is very incomplete, and it is much to be desired that investigations should be undertaken to ascertain this, as in law cases the question often arises whether the disease could spread in the matter of a few hours during transport.

It is almost impossible to answer such questions as these with one's present knowledge. Hofer points out that rapid progress of the disease is in ratio to the resisting power of the fish. For example, a carp which has travelled and been subjected to a change of temperature, thus chilling the skin, has been completely covered by the fungus in twenty-four hours.

Small fish in brooks suffering from spotted disease or furunculosis become covered very freely and rapidly. During spawning most fish, particularly the perch-pike, are very liable to fungous disease, but are immune to other parasitic and skin diseases, such as trypanosomiasis, costiasis (*Costia necatrix*), icthyophthiriasis (infusoria), myxoboliosis, crustacean and worm infestation, etc.

After the fungus has vegetated to a more or less extent, and formed its field or area, it then commences to propagate. This generally takes place through the mycelia of the hyphæ granulating and forming spore retainers. In the developed sporangia there are numerous zoöspores, sometimes amounting to as many as 800, which are mostly provided with two cilia. In order to classify the various species, it is very important to note their shape. As a rule, they are long, cylindrical, clavate (club-shaped), or elliptical. After the zoöspores have emptied themselves, the newly-formed sporangia develop secondary zoöspores, which burst through the original and now empty zoöspore, so that two to eight skins of sporangia adhere to one another. The zoöspores expelled from the sporangia are either monoplanetic—that is to say, they only undergo one process of forming fresh zoöspores, which are generally elliptical in shape, and provided with two cilia at the anterior extremity, although the latter is sometimes missing—or diplanetic, having a second subdivision of zoöspores, which are at first elliptical, and then become surrounded with a membrane which sprouts from the sides in a kidney or bean shape, with two cilia at the side. If these spores drop on a suitable medium, they develop germ-threads, which force their way into the underlying tissues, and quickly develop into a fungus.

A second form of reproduction is by means of so-called conidia, as seen in Saprolegnia. Conidia can be described as segmented, cylindrical threads of fungi, the end segment of which becomes detached, and, if falling on favourable media, bursts on either side and vegetates.

Besides these two asexual forms of propagation there is a third, by means of oöspores and fertilised by spermatozoids. The egg-cells or oöspores are situated at the base of a short tube, and very quickly develop into zoöspores. These oöspores are found at the end of the mycelia or threads of fungi in pockets called "oögonia," which resemble round balls. In order to propagate, so-called antheridia form, which lie at the sides of these oögonia, and often entirely envelop them (Hofer).

Symptoms.—The symptoms have been fairly well described in the symptomatology of the "Epizoötic Disease of Fresh-Water Fishes" (*vide* p. 946). The fungus may be found in patches, which have a tendency to run into one another or diffusely spread over the whole surface of skin, giving the fish a furred or woolly appearance, the fish thus resembling, when under water, a sheep with long wool. This fluffy fungous material may be of a whitish, dirty white, or pinkish colour, and the filaments streaming from the surface of the skin vary in length from ½ to 1½ inches.

In a great number of outbreaks the subcutaneous tissue of the affected fish is not discoloured, even under the thickest matted patches of fungus, thus showing that up to the time the fish were captured no ulceration, or indication of any, either on the head or scaly parts of the body, had taken place. In such cases the absence of ulceration may be taken as an indication that a fungus disease may appear independently of such primary infections as the *Bacillus pestis salmonis* and the *B. salmonicida,* in the presence of which fungi seem to arise on the necrotic parts of ulcers.

In some instances the fish, when in the water, appear as if covered with a halo. They remain at the surface as if immobile, and frequently put their mouths out of the water. Immediately before death they turn belly uppermost. If taken out of the water and examined, the fungus may be found to be thickly matted or felted on the shoulders just behind the head, choking up the gill openings, on the pectoral fins, and tail portion of the body. In others there may only be a slight ulceration, or rather abrasion on the fins, especially on the pectoral or caudal fins, several of the rays of which may be broken, and detached portions hanging by filaments of the fungus. The fungus may then cover the greater portion or the whole surface of the body, even covering up the eyes and nostrils, and the opercular opening of the gills may be completely closed up. When the fungus succeeds to an ulcerative infective process, such as the "salmon disease" or furunculosis, the tissues underlying the matted fungal mass are destroyed as if by erosion, even down to the bony framework.

It has been thought by some investigators that death may arise from suffocation by the fungus when situated within the mouth and upon the gill-covers at the same time, or by growing over and closing up the opercular openings of the gills. Others have considered it has been brought about by the fungus adhering and spreading over a greater portion of the skin, shutting out the light, preventing endosmosis and exosmosis, and by destroying the secretion of the mucus for lubrication, setting up constitutional disturbance, leading to death; and, finally, others think by the formation of a toxin by the fungus, which causes necrosis, or, when absorbed, a toxæmia.

Lesions.—As a rule, the lesions are those observed in "salmon disease," furunculosis, or some other disease *per se*. When arising in aquaria, tanks, etc., there is a very slight abrasion or ulceration of the epidermis or dermis, usually set up by an injury arising from rough handling, netting, etc.

Prognosis.—When occurring from no appreciable cause, it is not so fatal as when acting in symbiosis with some other infection. If, however, it is neglected, and no active precautions are taken to combat it, the mortality may be fearful. If rational means are adopted early, fish may recover from the fungus.

Prevention.—As the saprolegniaceous fungi are so widely distributed in Nature, and the disease is so prevalent, it is impossible to prevent contagion. What one should endeavour to accomplish is to take care that no practical means are withheld in the removal of causes that are liable to reduce the vitality of the fish. Aquaria, trays, tanks, stock-ponds, should be kept clean, and as free from dead organic matter as is possible. Decaying wood, dead fish, and other animals, should be removed from the water. Affected fish should be isolated, so as to prevent the spores being transmitted to the healthy. In the case of a fish-pond the water should be run off, the pond cleaned out, and then disinfected with chloride of lime. The pond should, after about a fortnight, be refilled with water highly oxygenated before the fish are replaced. Jackson, of the Southport Aquarium, banished fungus disease from that establishment by using a continuous stream of water fed by pumps from a large reservoir. By this method the water is beautifully clear and highly oxygenated, and any fungusy fish put into it recover.

Treatment.—Many authorities have adopted the salt-water method of treatment, and have had great success so far as the disappearance of the fungus is concerned. The late Sir James Gibson Maitland, Bart., found that 56 pounds of basalt to 650,000 gallons of water was sufficient to stop any new cases of fungus. The late Frank Buckland was in the habit of treating his fish affected with fungus with salt water, and found that, if the disease had not gone too far, a cure generally resulted. Carrington, of the Aquarium, Westminster, successfully treated a pike affected with fungus with salt baths. Fungusy fish travelling to the sea lose the fungus. The late Marquis of Ailsa put diseased fish into a solution containing 2 tablespoonfuls of salt in 1½ pints of water, and kept them in it until they became sickly, and then took them out. One dose was generally sufficient to cure them. He gave them a second bath in a fortnight, if necessary.

Hofer, of the Munich Veterinary School, uses at the Biological Experimental Station permanganate of potassium. The treatment should be carried out by sponging the fungus off with a 1 per cent. solution. This only destroys the superficial layer of fungus, leaving the roots capable of reproduction buried. Therefore he considers it necessary to give the fish a bath containing 1 part of the salt to 100,000 parts of water (1 ounce to 625 gallons). Salmon and carp may be left in this solution for thirty minutes. The solution then penetrates through the broken ends of the mycelia of the fungus right into the deeper layers of skin, destroying the spores. The fish should be watched while in this solution, as the thin mycelia of the fungus become detached and absorb the oxygen, making the breathing of the fish difficult. Fish should be put in a few at the time, as the solution soon becomes decomposed and inert.

Fungous Disease of Fish Eggs and of Alevins.

Synonyms.—Byssus.

French : La maladie de la mousse ; Byssus.
German : Die Pilzkrankheit der Eier ; Byssus.

The eggs of fish, especially of salmon and of trout, and the alevins (fry with yolk-sac attached) require the greatest care and attention during the process of artificial hatching in trays or tanks. They should be visited and examined twice a day to see if they are healthy and free from saprolegnial fungi, because, if this fungous disease is not detected at once when present, it is liable to spread so rapidly to the healthy eggs and alevins as to cause the death of the whole hatching.

It is said that eggs kept in obscurity produce stronger fish than when exposed to the light. On the other hand, unless one uses a candle or lamplight (gaslight, in consequence of giving off a great quantity of carbonic acid, is injurious to the eggs and alevins) with which to examine the hatchings, one is liable to overlook dead or dying eggs and alevins or the presence of fungous disease.

Healthy eggs have a yellowish amber transparent appearance, whereas sickly or dead eggs are whitish or opaque.

In a few hours dead eggs become covered with a whitish down, due to one or other of the saprolegnial moulds, formerly attributed to *Saprolegnia Thureti* (*S. ferax*), which was evidently a generic term, and included several species of the saprolegniaceæ. This mould, if not quickly detected, and the dead or affected eggs promptly removed, soon spreads by means of an extension of its filaments to the living eggs, and even to the alevins.

To prevent this disease, the rearing tanks or trays should be kept scrupulously clean, and all remains of artificial food removed. In the hatching and rearing trays or tanks only pure water, free from organic matter in solution, and previously passed through a filter, should be employed. When the disease has declared itself, it is necessary to remove all the dead, dying, and affected, and give the affected an immersion in a bath containing salt water (3 per cent.) three or four times a week for a fortnight.

For alevins it is necessary to stop the current of water, and put a quantity of salt into the water, and leave the young creatures in it as long as is possible.

When the eggs in course of incubation are invaded by the mould, salt should be added to the water in the tanks in the proportion of 10 to 15 per cent., in which they should stay for some minutes. This treatment, however, has some drawbacks when applied during the latter stages of incubation, as it is liable to hasten the hatching. Permanganate of potassium has also been recommended, but cannot be used in a stronger solution than 6 in 100,000. It may, however, be useful in disinfecting the tanks.

The dead or diseased eggs are removed by means of a vacuum glass pipette or suction-tube.

The System of Saprolegniaceæ.

It is advisable to give a brief outline of this system, as much ignorance is displayed in the description of the fungi associated with fish diseases, and the part they play in the etiology of such diseases. They are usually ascribed to one species —*S. ferax*—which appears to have been used in the past as a generic term for all fungal attacks. On glancing over the literature on the subject, one finds it is quite impossible to differentiate one fungal disease from another. It is very probable that when further research is made in this direction it may show that fish are attacked by fungi having special characters peculiar to a particular species.

It is impossible to give here all the species of Saprolegniaceæ known to botanists, and it is useless to refer to those of doubtful or unknown families. Therefore, only a résumé of the special characters of the better-known species will be given.

The *order* Saprolegniaceæ is included in the Phycomycetes, a group of lower algal-like sexual fungi. This latter belongs to the class Oömycetes, in which sexual reproduction takes place by means of oöspores, and either asexually by sporangia or by conidia.

The *Phycomycetes* are either aquatic or aërial fungi, which exist as parasites or saprophytes on plants or animals, and provided with an aseptate mycelium. Their *sexual* mode of reproduction is either by oögonia and antheridia, or by conjugation of morphologically similar branches. Their *asexual* mode of reproduction is by gonidia or zoögonidia.

The order Saprolegniaceæ is divided into two families—(1) Saprolegniaceæ and (2) Monoblepharidaceæ.

FAMILY I.: **Saprolegniaceæ.**—This forms one of that division in which the hyphæ are well developed. The hyphæ bear zoögonidia. Asexual reproduction takes place by zoögonidia, and sexual by antheridia and oögonia, which produce oöspores. The zoögonidia are usually biciliate, and the hyphæ undergo partial or complete transformation into zoösporangia.

This family is divided into sub-families: (I.) Saprolegnieæ and (II.) Apodyeæ. The following is an analysis of the various families, sub-families, and genera:

SUB-FAMILY I.: **Saprolegnieæ.**—The mycelial branches are cylindrical, and of an equal thickness throughout, and are not segmented.

A. Spores given off from the sporangia; oöspores develop externally.

1. All oöspores expelled from one opening in the sporangia.

(1) Oöspores develop in a matured state.

(i.) Monoplanetic: Sporangia elliptical, not developing fresh oöspores through the empty sporangia, but by sprouting (1. *Pythioptis*).

(ii.) Diplanetic: Sporangia, either long and threadlike or nodular; after emptying, growing through each other.

(*a*) The nodular sporangia contain several rows of spores; the oögonium contains large number of zoöspores (2. *Saprolegnia*).

(*β*) Sporangia threadlike, only containing one row of spores; oögonium containing only one zoöspore (3. *Leptolegnia*).

B. Zoöspores still without cilia and without movement; opening of the sporangia is a small cylindrical head; they are monoplanetic.

(i.) Sporangia cylindrical, containing several rows of spores; oögonia contain many rows of oöspores; the tubes are thick (4. *Achlya*).

(ii.) Sporangia threadlike; only one row of spores; oögonia single oöspore; tubes very thin (5. *Aphanomyces*).

2. Every zoöspore proceeding out of one particular orifice, immediately reproducing; monoplanetic; a multiple network of cell-walls remain behind in the sporangia (6. *Dictyuchus*).

(2) Zoöspores which develop within the sporangia (7. *Aplanes*).

SUB-FAMILY II.: **Apodyeæ.**—Mycelial tubes cylindrical, and develop by regular segmentation.

1. Zoöspores develop in the sporangia, and expelled through one and the same opening.

(*a*) Zoöspores swarming separately on escaping (8. *Apodya*).

(*b*) Zoöspores, which remain attached at the mouth of the sporangia on escaping (9. *Apodachlya*).

2. Zoöspores, which are in front of the sporangia, and empty into a cyst before dividing and subdividing into spores (10. *Rhipidium*).

FAMILY II.: **Monoblepharidaceæ.**—Antheridium very much in the form of sporangia; form spermatozoids; oögonia with an opening.

1. Mycelial tubes cylindrical and even, and not segmented (11. *Monoblepharis*).

2. Mycelial tubes regularly segmented and spindle-shaped (12. *Gonapodya* Hofer).

FAMILY I.: **Saprolegniaceæ.**

SUB-FAMILY I.: **Saprolegnieæ.**

GENUS 1: *Pythioptis.*

SPECIES: *P. cymosa.*—The hyphæ are slender and short; zoösporangia are globular to a short clavate form. The oögonia are often terminal and globular; their walls unpitted, and sometimes with very blunted outgrowths. The antheridia are clavate, having one or more, rarely two, on each oögonium, generally arising just below its basal wall, and rarely of diclinous origin. The oöspores are usually single, rarely two, in the oögonium, eccentric, with several oil globules, and have an average diameter of 18 μ.

This species is found on fish and on algæ in Europe and in the United States of America.

GENUS 2: *Saprolegnia.*

SPECIES: *S. hypogynia.*—The antheridium is formed just below the oögonium on the same branch. It is found on aquatic insects and on crustaceans.

S. dioica.—Diclinous plants; small pits. Oöspores commonly ten to twelve, sometimes twenty or more, and often only four to six; centric, and average diameter about 25 μ.

It is found on fish.

S. anisospora.—This has two kinds of sporangia, producing respectively large and small spores. They absolutely agree in structure and development, but the larger are about twice the diameter of the smaller. The oögonial walls are unpitted. Oöspores eccentric.

It is found on fish.

S. monoica.—Oögonia with a terminal or lateral perforation, branches short and straight, oöspores hyaline, globose, 20 μ in diameter, antheridia formed at the tips of lateral branches.

It is found on flies, spiders, larvæ, fish, and crustaceans.

S. mixta.—Antheridial branches often developed, cylindrical. Oöspores up to fifteen, or occasionally more than twice that number, centric, their average diameter about 26 μ

It is found on fish.

S. Thureti (*S. ferax* Thuret).—Oögonia globular, or not rarely cylindrical, not in series, with large pits. Oöspores up to twenty, or sometimes more—forty to fifty or over—in an oögonium, centric, average diameter 26 μ.

Very commonly found on living or dead fish.

S. torulosa.—Oögonia irregular, often in torulose series, with few and small pits. Antheridia rarely present; oöspores as many as twelve, or rarely more, in an oögonium, centric, average diameter about 25 μ.

It is found on the majority of water insects.

S. monilifera.—Oögonia in moniliform series, early falling apart; not all pitted. No antheridia developed.

It is found on fish.

S. asterophora.—Oöspores one or two, rarely three, in an oögonium; centric, thick-walled, diameter 30 μ; hyphæ slender.

S. Treleaseana.—Oöspores several in an oögonium; hyphæ very stout.

GENUS 3: *Leptolegnia.*—This differs from the saprolegnia, inasmuch as the oögonium contains a single oöspore, which completely fills it.

SPECIES: *L. caudata.*—This single species has narrow sporangia, ovate oögonia on short racemose branches, and antheridia, usually one to each oögonium, on branches of diclinous origin.

It is found on fish.

GENUS 4: *Achlya.*—Diœcious or monœcious; oögonia terminal or intercalary, polysporous, rarely unisporous; antheridia terminal on simple or divided branchlets, usually springing from the hypha just below the oögonia in monœcious species; zoösporangia formed from swollen apices, usually thickest near the middle; fusiform. The zoögonidia are enclosed in a thin pellicle after liberation from zoösporangia, and usually form a compact hollow sphere at the moment of escape from zoösporangia, from which they eventually do so in the naked form.

SPECIES: *A. racemosa.*—Oögonia not apiculate; antheridial branches always from the oögonial branches.

Fish and eggs raised in hatcheries.

The *A. racemosa*, var. *stelligera* (*A. racemosa*, var. *spinosa* Cornu), is possibly a transitional form between the smooth and spiny species of Achlya, not only as regards their oögonial structure, but also in the reduction of the antheridial branches, which is carried even to their entire disappearance in some spiny species (Humphrey).

A. gracilipes (*A. polyandra* Hildebrand).—Oögonial branches long; oöspores centric.

It is found on putrid fish and insects in water.

A. apiculata.—Oögonia usually apiculate; antheridial branches oftenest from the main hypha.

Found on decomposing fish.

A. prolifera.—Abundantly pitted walls of oögonia, and numerous and long antheridial branches of diclinous origin.

Found on fish.

A. oblongata.—Strictly diclinous plants; smooth unpitted walls. Oögonia occasionally intercalary, usually terminal and elliptical, ovate or obovate, rarely globular.

Found on dead fish.

A. oligacantha.—Oöspores averaging more than five; antheridial branches long.

Found on dead fish.

A. spinosa.—Usually barrel-shaped oözonia, with numerous blunt, and often broad or even forked, outgrowths, each containing one or two, rarely three, oöspores, which often take an elliptical form corresponding to that of the cavity of the oögonium.

Found on dead fish.

A. stellata.—Has no antheridia. Its globular or elliptical oögonia are covered by rather less numerous spines that are short or sharp and conical. Oöspores always single.

A. polyandra (*A. de Baryana*).—Oögonial branches short; oöspores eccentric, unpitted.

Found on fish.

GENUS 5: *Aphanomyces.*—Zoösporangia very long, slender, cylindrical; zoögonidia uniserate; oögonia usually containing one, rarely two, oöspores; antheridia apical on vermiform branchlets, encircling the oögonia.

SPECIES: *A. lævis.*—Oögonia quite smooth; antheridia always present.

Found on water insects.

A. scaber.—Spines small or reduced to mere roughness; antheridia often absent.

Found on water insects.

A. stellatus.—Spines large and prominent; antheridia always present.

Found on water insects.

A. phycophilus.—Oögonia spiny.

Parasitic on algæ (Zygnemaceæ).

A. Schikoras.—Grows on crayfish and prawns.

GENUS 6: *Dictyuchus.*—Zoöspores of two forms, escaping from their proper membranes before leaving the zoösporangia, the empty membranes remaining in the zoösporangia and presenting a reticulated appearance. Oögonia, one or more spored, not perforated.

SPECIES: *D. monosporus.*—Oögonia under 30 μ in diameter, encircled by antheridia.

On insects and rotting bulbs of tulips and hyacinths in water.

D. Magnusii.—Oögonia over 30 μ in diameter, not encircled by antheridia.

On *Trapa natans.*

D. polysporus.—Androgynous plants; oögonia several spored.

Found in water.

D. clavatus (*Thraustotheca clavata*).—Has short, clavate sporangia.

Found on flies.

GENUS 7: *Aplanes.*—The swarming stage is entirely suppressed. The zoöspores encyst within the sporangium, and germinate there, producing germ-tubes, which penetrate the sporangial wall, and thus make their way into the surrounding water.

SPECIES: *A. Braunii* (*A.* (*Saprolegnia*) *androgynus* Archer).—Several spored, barrel-shaped, or spindle-shaped oögonia, with pitted walls, often formed in series from a hypha, its numerous antheridial branches arising just below the basal wall of each oögonium.

Found on flies and putrefying bulbs.

SUB-FAMILY (*b*): *Apodyeæ* (*Leptomiteæ*).—Hyphæ constricted at regular intervals, sparingly branched; zoösporangia formed from the terminal portions of the branches, one or several superposed; zöogonidia maturing within the zoösporangium, and not escaping until after germination.

GENUS 8: *Apodya* (*Leptomitus*).

SPECIES: *A. lactea.*—It forms tassel-like way tufts, attached at a base; is dirty white and slimy; filaments up to 5 centimetres long and 6 to 12 μ thick, constricted at intervals, dichotomously branched, flaccid; zoösporangia terminal on the filaments or axillary; zoögonidia ovate, 2 μ long, furnished with two cilia about 12 μ long.

It is found in water greatly polluted with organic matter from beet-sugar factories, tripe-houses, etc. It also arises from decaying algæ and other vegetable matter. It does not appear to develop on decaying animal matter.

GENUS 9: *Apodachlya.*—In this the hyphæ are slender throughout, marked off at intervals by constrictions into distinct segments; branching monopodial; branches arising from any part of a segment. Zoösporangia from considerably swollen segments, renewed by sympodial branching. Zoöspores usually become grouped at the mouth of a sporangium on escaping, rarely swarming separately; soon leave their cysts and swarm; finally, encyst again and germinate. No sexual organs are known.

SPECIES: *A.* (*Leptomitus*) *brachynema.*—This has hyphæ with short segments, and globular sporangia opening by short necks, and often occurring in series.

Found in water.

A. pirifera.—Has hyphæ with long segments, pyriform sporangia developed singly, and each opening by a distinct papilla, and large globular chlamydospores.

GENUS 10: *Rhipidium.*—Resembles Apodya in having constricted filaments, but is distinguished by having flabellately arranged branches.

SPECIES: *R. interruptum.*

R. elongatum.

R. spinosum.

FAMILY II.: **Monoblepharidaceæ.**—Zoögonidia uniciliate.

GENUS 11: *Monoblepharis.*—Mycelium aseptate, branched, oögonia terminal, sometimes appearing to be lateral, owing to a continuation of growth of the hypha beyond the oögonium, the latter being deflected, solitary, or superposed in chains of from three to twelve; antheridia formed from the cells immediately below, and supporting the oögonia, or springing from the walls of the latter organ, solitary, or superposed; antheroids motile, furnished with a single cilium; zoösporangia thin-walled, sometimes proliferous; zoögonidia motile, uniciliate (Magee).

SPECIES: *M. sphærica.*—On decaying animals and leaves in water.

M. polymorpha.—On decaying animals and leaves in water.

GENUS 12: *Gonapodya.*

SPECIES: *G. prolifera.*—On decaying plants.

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LEISHMANIASIS IN THE DOG

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Leishmaniosis; Kala-azar; Dum-dum fever; Tropical splenomegaly.

Leishmaniasis in the dog is an important disease because it is considered to be the reservoir for infantile kala-azar in young children.

It belongs to the group Leishmaniases, due to protozoal parasites termed "Leishman-Donovan" bodies, which Laveran and Mesnil included formerly in the *genus* Piroplasma, Marchand and Ledingham in the *genus* Trypanosoma, and Rogers in the *genus* Herpetomonas. Ronald Ross, however, created for them a new *genus*, Leishmania.

Geographical Distribution. — The Leishmaniases are widely distributed. They have been found in Calcutta, Madras, Bengal, Annam, China, Sudan, and countries in the Mediterranean littoral, as Algiers, Tunis, Tripoli, Portugal, Spain, Sicily, Italy, Malta, Greek Archipelago. Monsieur and Madame Yakimoff found it to occur in five dogs in 300 examined in Tunis.

The principal forms of Leishmaniasis are—

1. Kala-azar, or adult Leishmaniasis, due to *Leishmania Donovani.*

2. Infantile Leishmaniasis, caused by *Leishmania infantum,* which is transmitted from the dog.

3. Aleppo, Oriental, or tropical boil or sore, or cutaneous or superficial Leishmaniasis.

Other forms of Leishmaniasis have been said to occur in man, but their causal parasites have not been satisfactorily determined. One of these said diseases is typhus, another dengue, and the third the spotted fever of the Rocky Mountains, which is said to be transmitted by the tick *Dermacentor andersoni.*

With the exception of infantile Leishmaniasis, the other forms are not transmissible from the human subject to the dog, or, as far as is known, to any of the other domestic animals, and will not be described.

Etiology.—Nicolle believed infantile Leishmaniasis in children in Tunis to be of canine origin, and this view has since been confirmed by Gabbi and Basile in Italy, Critien in Malta, Alvarez in Lisbon, the Sergents in

Algiers, and Monsieur and Madame Yakimoff in Tunis. Spontaneous Leishmaniasis in the dog is milder in manifestation than by experimental infection. In several instances the children attacked had been playing with manifestly affected dogs. It is believed the dog-flea—*Pulex serraticeps*—and the human flea—*Pulex irritans*—act as vectors. To what extent bed-bugs—*Cimex lectularius, C. rotundatus*—and a reduviid bug—*Connorrhinus rubrofasciatus*—play in the genesis of the disease, is not known. Leishmania were found by Basile in the intestinal contents of several fleas removed from dogs dying of Leishmaniasis. These fleas, when placed on healthy dogs, transmitted the disesase. The infection is not transferable from the bitch to the fœtus.

Causal Organism.—The *Leishmania infantum* is shown under the form of small pyriform rounded or oval organisms measuring 2 to 4 μ in diameter. It is stained by the methods Romanowsky or Leishman, which give a large, spherical, slightly-coloured nucleus and a blepharoplast having the form of a short rod, placed perpendicularly or tangentially to the nucleus, and staining well. Sometimes there is seen a prolongation in the form of a tail placed perpendicularly on the blepharoplast, and is probably the remains of a flagellum. The parasite reproduces itself generally by simple longitudinal division, and sometimes by multiplication, when the forms are larger, circular, and show several couples of chromatin (nuclei and blepharoplast), around which the protoplasm is segmented.

It grows readily in the media of Novy-McNeal and of Nicolle, but does not develop in Roger's citrated blood-medium. In Nicolle's medium at 22° C. the culture commences to grow by the seventh day, and attains its maximum by the twelfth day, and maintains its vitality at the end of three months.

The parasite is abundant in the enlarged spleen and bone-marrow, and stains well with methylene-blue or Giemsa. The parasites may be free, but are mostly found in the white blood-corpuscles.

Infantile Leishmaniasis in the Child.—Although it simulates kala-azar in adults, it generally attacks children under two years of age, sometimes later, rarely over six. There are, however, some forms of kala-azar which occur in children ten or twelve years of age, but it is not known if they are identical with that seen in young children.

Symptoms in the Child.—The most characteristic symptoms are extreme pallor, marasmus, considerable enlargement of the spleen, a somewhat less enlargement of the liver, fleeting and painful œdemas, a very irregular temperature capable of exhibiting several "maxima" on the same day, a notable quickening of the pulse-rate, digestive disturbance, and an increase of the mononuclear elements of the blood.

Hæmophilia, noma, gingivitis, and purpura are frequent complications. The course of the disease is long. Death generally occurs, although spontaneous recoveries may be rarely seen.

In the Dog.—The acute form attacks young dogs, and may last for three or four months. The chronic form is seen in adult animals, and continues for several months before death ends the scene. A dog experimentally inoculated lived eighteen months before succumbing. The disease remained latent sixteen months before an abnormal temperature manifested itself. There is usually progressive anæmia and emaciation, and the hairs are shed on the least traction. Microscopical examination of the blister serum may show the parasites, but usually that of the bone-marrow, liver or spleen tissue, must be had recourse to before the parasites can be found.

To obtain bone-marrow from the living dog a long bone should be trephined, and some of the marrow removed. It is a trifling operation in the dog, and can be performed under the influence of cocaine. The hepatic or splenic tissues or pulp can be obtained by puncturing these organs. The peripheral blood does not show the parasites.

Lesions in the Dog.—The lesions are an enlargement of the spleen and of the liver, and the bone-marrow is of a red colour. Dogs and monkeys can be infected.

These are similar to those observed in the child. The spleen may be very much enlarged. In mild cases the bone-marrow appears of a brick-red colour, and in acute cases of a bright red colour. It is very rich in parasites. It is, however, well to mention that the colour of the bone-marrow is of no pathognomic value, as it is also encountered in piroplasmosis.

Treatment.—Quinine does not influence the fever. There does not appear to be any medicinal treatment of any value. Removal of the spleen in an infected dog does not influence the course of the disease. Recovery in one child followed the use of Tr. senegæ. In tropical and semi-tropical countries where the disease has been proved to exist, dogs should not be allowed in the beds or bedrooms of children. Fleas should be destroyed by paraffin diluted with a bland oil. This disease requires to be studied from a veterinary point of view.

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THE COCCIDIA

By HENRY GRAY, M.R.C.V.S.

The coccidia are protozoa with motile organs, and belong to the *Class* Telosporidia, in which sporulation ends the life of the individual. They are included in the *Order* Coccidiidea, in which the cell-infesting sporozoa are usually reproduced by schizogony or by sporogony, and thus giving them a life-cycle with an alternation of asexual or sexual generation. After becoming fertilised, the oöspore forms sporoblasts, which may or may not be covered by a sporocyst membrane, and which may become transformed into one or several sporozoites. They belong to the *Suborder* Asporocystinea, because their sporoblasts have no sporocysts.

The principal *Genera* engaged in the production of disease in the higher animals are—

1. *Cyclospora,* in which each sporoblast forms two sporozoites. The chief species is *Cyclospora caryolytica* Schaudinn, which produces a fatal enteritis in moles.

2. *Diplospora,* which has tetrazoic spores. It has many species, affecting birds, snakes, lizards, and frogs. The chief species infesting birds is *Diplospora Lacazei.*

3. *Eimeria* Schneider 1875 (*Coccidium* Leuckart 1879), in which the dizoic spores are spherical or oval, and the fertilised cell produces four sporoblasts with sporocysts or chlamydospores. They are found as parasites in nearly all the vertebrates, and among the domesticated animals they sometimes occasion great loss. Man is not exempt from their ravages. From a utilitarian point of view this genus is the most important.

4. *Klossiella,* in which the sporoblasts are polyzoic, and the subspherical sporocysts contain as many as thirty to thirty-four sporozoites. There is only one species—*Klossiella muris*—found in the kidney of the mouse.

The Coccidiidea are found in nearly all species of vertebrates and invertebrates. They are cell-parasites, usually attacking the epithelial cells, especially of the intestine, bile-ducts, or urinary tubules, rarely the submucous tissue or lymphatic gland, and never the blood-cells.

The genus *Eimeria* (*Coccidium*), mostly confined to vertebrates, contains parasites which chiefly attack the epithelial cells, especially of the intestine, bile-duct, or urinary tubules. They are also found in the lymphatic glands, submucous tissue of the gut, and other parts. They always gain an entrance to the system by the alimentary canal, to which they mostly confine themselves. Occasionally, however, they pass through the wall of the intestine, and are probably carried by the blood or lymph stream to the seat of their elective affinity, such as the kidney or urinary bladder. Some coccidial parasites of other genera attack the spleen or testis, never the ovary. A certain coccidial parasite may confine its ravages entirely to a particular organ, or it may infest several organs at the same time, as in the case of *Coccidium minutum*, which invades the liver, spleen, and kidney of the tench. In some animal species two or more coccidial parasites of different genera may be found present at the same time. In other cases a coccidium and another protozoal parasite may be associated in a particular animal. For instance, the gudgeon may be invaded by a coccidium—*C. Metchnikovi*—and a myxosporidium—*Myxobolus oviformis*—which seem to act in association. The myxosporidium is found in the liver, spleen, and kidney, containing the coccidium in various phases of development, especially in the stage of cysts with spores. In this case the myxobolus generally contains no spores of its own. Free coccidia not included in Myxosporidia are present only in the intestine.

THE COCCIDIOSES.

This is an important group of diseases occurring in the domestic animals, especially in the bird and rabbit, and probably not rarely in the sheep and goat, and perhaps calf. In the pig and horse, disease is not usually associated with coccidia—at least, they are rarely recognised as causing disease. In the dog and cat, possibly, they are more frequently the cause of morbid phenomena than most have allowed to them.

The presence of the parasites in the epithelial cells of the intestine, bile-ducts, kidney tubules, or urinary bladder, completely destroys the cells which harbour them. As a general rule, the trophozoite occupies the protoplasm, and does not attack the nucleus directly. It produces an enlargement of the cell, and especially of its nucleus. The cell undergoes rapid growth, and forms a fatty substance, which act as a nutrient for the parasite which consumes them. The surrounding tissues are often stimulated, and a proliferation of the connective tissue is induced, forming a healing process on the part of the bearer. After the infected cell is exhausted, it can no longer assimilate, but soon dies, and is ultimately consumed by the parasite. The parasite then passes into the reproductive stage, either still covered by the remains of the cell it has killed, in schizogony, or clear from the cell, in sporogony. Some parasites are intranuclear, as in the case of *Cyclospora caryolytica* in the mole. But those included in the genus *Eimeria* are always intracellular. Each individual trophozoite or vegetative organism destroys only one cell in the host. The parasites are frequently so numerous that large patches, or even the whole of the epithelium of the organ invaded, are removed, so that no barrier remains to prevent invasion of saprophytic organisms, or the toxic elements in the intestinal or other organic contents, from being absorbed into the system, and setting up intoxication or other clinical phenomena. In this case death usually results. Still, a great deal of destruction of the epithelial layer may be present without clinical manifestations. The extent of destruction of the epithelium varies according to the conditions of the multiplication of the parasite by schizogony. Schizogony cannot continue indefinitely, but has an inherent limit, which, when reached, the parasite requires for the continuance of the species—conjugation with resulting sporogony. If the parasite fails to reproduce itself, the animal may overcome the earlier stage of an attack by the regeneration of the epithelium. One attack may not give immunity to a fresh infestation from without.

Method of Study.—The course of coccidia can be studied by placing them on small fragments of charcoal or sand in a watch-glass containing a few drops of carbolic lotion, in order to prevent the growth of bacteria or moulds, and then to place them in a moist chamber. They can also be studied by putting them in a little water or normal saline solution, and then placing them under the microscope.

Method of Staining.—Coccidia do not take up colouring matter very well. Tissues should, according to Pianese, be immersed for thirty-six hours in the following solution :

Ten per cent. aqueous solution of chloride of cobalt	20 c.c.
Two per cent. aqueous solution of chromic acid ..	5 c.c.
Formic acid	1 drop

Bertarelli's method is fixing the tissues in a saturated aqueous solution of sublimate, then staining them for twenty-four to forty-eight hours in dilute hæmatoxylin Grenacher (1 c.c. hæmatoxylin in 200 c.c. water), and differentiating them with acetised alcohol.

Borrel's method of fixing and staining sporozoa in sections is very good to enable one to well study them. Small portions of tissue are fixed for twenty-four hours in a refrigerator in the following mixture :

Osmic acid	2 grammes
Platinum chloride	2 ,,
Chromic acid	3 ,,
Acetic acid	20 ,,
Distilled water	350 ,,

They are afterwards well washed in a good stream of water, and then embedded in paraffin wax. Thin sections are coloured, in cold, for an hour in a saturated solution of Magenta red, then to differentiate, for five to ten minutes, in the following :

Saturated aqueous solution of picric acid ..	1 volume
Saturated aqueous solution of indigo carmine ..	2 volumes

They are then washed rapidly, decolorised with absolute alcohol, and placed in oil of cloves, in which they should be kept in contact for some time before finally mounting. By this method the nuclei are coloured red, the protoplasm bluish-green, and the hæmoglobin yellow or yellowish-green.

Diagnosis during Life.—For diagnostic purposes during the life of the animal the fæces should be examined, when, if coccidiosis is present, numerous oöcysts or other coccidial stages are present in more or less numbers during the active stage of the disease, in the convalescent stage, and long after an apparent recovery. Minute particles of fæces or quantities of the serous or watery fluid placed on a glass slide on which a drop of eosin solution is allowed to fall show under the microscope colourless ellipsoidal or rounded parasites, resembling in outline a bird's egg in miniature.

In renal or vesical coccidiosis the urine should be centrifugalised, and the deposits examined under the microscope. In the case of birds, the parasites should be sought for in the white urates on the fæces. In ascitic or pleuritic fluid they may be found after centrifugalisation.

The life-cycle of coccidia is outlined in the description of that of the rabbit ; it stands good for that of the bird. The life-cycle of the coccidia of the ox, sheep, goat, dog, cat, and other domestic animals, has not yet been elucidated, but probably it is similar to that of the rabbit and bird coccidia.

I. COCCIDIOSIS OF THE RABBIT.

Synonyms.—Psorospermosis; Gregarinosis; Gotch; Pot-belly; Protozoal enteritis; Hæmorrhagic enteritis.

French: Coccidiose du lapin; La phtisie hépatique coccidienne du lapin; La psorospermose coccidienne hépatique du lapin; Gros ventre.

German: Coccidiose von Kaninchen; Psorospermosis cuniculi.

History.—This is the best known of the coccidioses, and was in the earlier part of the nineteenth century regarded by Sir Robert Carswell as of a tubercular nature. In 1839 Hake considered it was of a cancerous nature, and mistook the coccidia for pus globules. In 1843 Nasse opposed these conclusions, and asserted that these so-called "pus globules" were nothing more than abnormal or changed epithelial products. In 1846 Handfield Jones believed them to be transformed liver-cells. In the same year Rayer and many authorities looked upon them as the eggs of *Distoma lanceolatum*, or of various other entozoa. The true nature of these bodies was first pointed out by Remak in 1845, who saw in them a resemblance to Johannes Müller's fish psorosperms, who described them in 1841. In 1847 Kauffmann supported this conculsion after studying their incubation in water. In 1854 they were studied by Lieberkühn, who concluded they were related to the Gregarines. They were afterwards studied by Davaine, and then by L. Stieda. In 1865 Lindemann named the parasite *Monocystis Stiedæ.* Waldenburg, in 1860, 1862, 1865, 1866, and 1868, gave a good deal of attention to the subject. In 1869, 1873, 1878, and 1879, S. Rivolta made a very extensive inquiry into the nature of psorosperms infesting animals, and gave to that species found in the rabbit the name of *Psorospermium cuniculi.* In 1879 Leuckart published his researches on this parasite of the rabbit, and created for it a new generic name, *Coccidium.* Prior to this Eimer described in 1870 the endogenous life-cycle of the coccidium infesting the mouse (*Eimeria* (*Coccidium*) *falciformis* Schneider). Eimer, however, wrongly denominated it a Gregarine, but Schneider created for it a new genus, *Eimeria*, in honour of Eimer. And since zoologists are agreed that nomenclature should be based on priority of claim, Schneider's nomenclature of 1875 replaces that of Leuckart of 1879, and hence the denomination *Eimeria* takes precedence over that of *Coccidium.* In more recent times Balbiani, Léger, Labbé, Delepine, McFadyean, and numerous others, have given a great deal of attention to the disease. But the suggestion of an alternation of generation in the complete life-cycle of coccidia arose from L. and R. Pfeiffer, who

(1890-1892) met with a great deal of criticism for advancing such ideas, which are now universally accepted. Until then the coccidium found in the liver lesions was generally considered to be distinct from that in the intestine; but the careful researches of Metzner showed that coccidiosis of the liver and that of the intestine belonged to one and the same species.

Causal Organism : its Morphology and Life-Cycle.—The parasite of rabbit coccidiosis, *Eimeria* (*Coccidium*) *cuniculi* Rivolta (*Eimeria Stiedæ* Lindemann 1865 ; *Psorospermium cuniculi* Rivolta 1878 ; *Coccidium oviforme* Leuckart 1879) has an ovoid, smooth, fairly thick shell (oöcyst of some authors), showing at one of its poles, generally at its narrower extremity, a small indentation in the form of a micropyle. Its maximum length is from 40 to 49 μ by 22 to 28 μ in breadth, or, on an average, 33 to 37 μ by 15 to 20 μ. In the epithelial cells of the bile-ducts the oöcysts measure about 40 μ in length by 20 μ in width. They are at first filled with a granular protoplasm, which soon begins to contract and form a spherical body, that is somewhat separated from the poles, and possesses a central nucleus. When in this state the parasite is termed an "oöcyst," which is the last stage of its development in the liver. The oöcysts pass into the intestine, from whence they are discharged with the fæces, to undergo a further cycle of changes in the external surroundings.

The oöcysts, under favourable conditions of temperature and moisture of the atmosphere and soil, split up into two spheres, and then into four smaller spheres or *sporoblasts* (archispores). This takes from two to four days to accomplish. After a short period each sporoblast becomes elongated to form a *sporocyst,* which then undergoes a subdivision to form three segments. Two of these segments form *sporozoites* (gymnospores) or falciform corpuscles, each of which is supplied with a nucleus, and a *spore relict,* or remains of a mass of unused granular material.

The cyst containing the sporocysts is very resistant, and preserves its integrity for some time in the soil. Ingested by the rabbit, its covering is acted upon by the action of the intestinal juices, so that it ruptures and sets the sporocysts free. These, in their turn, open and let the sporozoites escape. The sporozoites are endowed with active movements, by which they gain the biliary ducts. They then enter the epithelial cells of the intestine or bile ducts by their elongated, narrow, or pointed extremity, and take up their residence in them between the free surface and the nucleus of the cell. Soon after they have gained the cell protoplasm, between the nucleus and the cell wall, they lose their power of movement, and are not long before they take on the *schizont* form, which increases at the expense of the invaded cell, and undergoes multiplication by non-sexual division (*schizogony*).

The *schizont*, which does not possess an enveloping membrane, develops in the form of a sphere, which has a large alveolus, and contains a clear fluid. Its nuclei soon subdivide themselves into a great number of daughter nuclei, which are in contact with one another on the surface; the protoplasm surrounding these nuclei separates, so that the claviform corpuscles are at first arranged like the quarters of an orange, and afterwards become free and endowed with movements similar to those of the sporozoites, and constitute *merozoites*. Arrived at this point, the epithelial cell bursts, and the merozoites are set at liberty, either to die in the intestinal contents or in the external surroundings, or to wander into fresh epithelial cells of the host or fresh hosts and undergo a different cycle of evolution. At times the merozoite which has entered a healthy cell loses its power of movement, becomes globular, grows, and forms a schizont, which immediately commences to multiply by schizogony. This *repeated schizogony* enables the parasite to undergo rapid multiplication in the rabbit's system, and to this process should be attributed the rapid course and invasion of certain outbreaks of coccidiosis. At other times, instead of the process of repeated schizogony, the merozoites penetrating the epithelial cells undergo their changes by sexual reproduction or *sporogony*.

In this case the merozoites, after having entered the epithelial cell, are transformed into *female* elements or *macrogametes*, and into *male* elements or *microgametes*.

1. *Macrogametes*.—In this the merozoite slowly develops, and is supplied with granules of chromatin formed from a reserve of matter. Its nucleus contains a karyosome. The macrogamete thus constituted is elliptical; it soon expels its karyosome; at the same time it shows a few contractions, which are generally made with the object of escaping from the epithelial cell; it then rests on the surface of the epithelium, where it readily comes in contact with the microgametes. At this time the macrogamete, then in a state of maturity, is spherical and immobile; it possesses a well-defined nucleus. The granules, which are carried to the periphery, become fused to form an enveloping membrane, which is pierced at one of its extremities by an orifice termed the *micropyle*.

2. *Microgametes*.—Contrary to the case of the macrogamete, the merozoite rapidly develops. It is deprived of an enveloping membrane and of reserved granular matter. Its nucleus possesses a large karyosome. Thus is constituted the microgametocytes, in which are formed the microgametes. The nucleus undergoes subdivision to form daughter

nuclei on the surface of the coccidium, and around each of which a hyaline protoplasm accumulates ; soon the daughter nuclei become flattened, elongated, and curved upon themselves in the form of a comma. The microgametes thus constituted continue to lengthen, and two flagella appear at their anterior extremity. The microgametes showing movements become free, and quit the microgametoblast, which forms a residual body, that soon undergoes degeneration.

The microgamete has a great resemblance to the spermatozoids of the higher animals ; it is very small (6 to 8 μ in length by 1 μ in width), active, generally falciform ; its body is refractile, homogeneous, and almost entirely formed of chromatin, surrounded by a very thin layer of protoplasm.

3. *Fecundation.*—After the macrogamete has become matured, it attracts the microgametes by a chemiotaxic influence ; one of these microgametes enters the micropyle, which is closed immediately, thus excluding other individuals, which then degenerate and die. The male element comes in contact with the nucleus of the macrogamete, and ends up by becoming fused with it ; fecundation is accomplished ; it terminates in the formation of a zygote or oöcyst, which, gaining the external surroundings, ripens and subdivides by sporogony to give birth to sporocysts, which infest fresh hosts.

The completion of this process varies from four to five days or ten to fifteen days, according to the conditions of environment.

Contagion.—One can understand that, after the coccidium passing through the multiplicative process several times, the original organism may be represented by tens or even hundreds of thousands of similar parasites. It is introduced into a rabbit warren or rabbitry by a diseased rabbit or by infested media, such as hutches, baskets, food, and such-like. Rabbit shows, dealers' shops, and markets help to spread the infestation. Rabbit fanciers have from time immemorial incriminated the giving young rabbits freshly collected green food before it was allowed to dry or wilt. This idea has some foundation in fact, but how fresh green food acts is not clear, unless it acts as a predisposing cause. The droppings of animals are very much infested. Dirty hutches favour the disease.

Prevalence.—The disease is very common in England, France, and Italy, and to a less extent in America. Delepine estimates that 92 per cent. of the rabbits in Great Britain are infested. Periodically it causes serious losses, not only in domestic rabbits, but in the wild ones at liberty. It may also attack the hare and the guinea-pig.

Age.—The disease is usually observed in young rabbits from four to six or eight weeks old, the adults being more immune. It may occur in the suckling young. Adult animals may be infested without manifesting any palpable signs of the presence of the parasites.

Climatic Conditions.—Warm weather with a certain degree of moisture favours the spread of the disease. It is rare in very hot, dry weather and in dry, frosty or cold weather.

Symptoms.—For the convenience of description it is advisable to subdivide them into *hepatic coccidiosis, intestinal coccidiosis,* and *nasal coccidiosis.*

Hepatic Coccidiosis.—Very often the liver of rabbits may be fairly infested with coccidia without showing any appreciable sign of their presence. When the infestation is of a high degree, the animal becomes dull, less inclined to move itself; its pelage is dull; the appetite is poor or nil; emaciation is gradual; the mucous membranes after a time are pallid or even yellowish; the fur is easily pulled out; weakness progresses; the breathing becomes increased in frequency; the gait is staggering, and towards the end the abdomen becomes "pot-bellied," ascitic, or tympanitic; diarrhœa is profuse; emaciation is advanced, and the creature dies after a few weeks' suffering. Sometimes death takes place during convulsions.

Intestinal Coccidiosis.—This form is usually acute in manifestation, especially in young rabbits and young hares, in which it may simulate an acute septicæmia, to be followed by death in a few hours after a short phase of dulness, loss of appetite, indifference to surroundings, and finally a comatose state.

In less acute cases there is dulness, loss of appetite, diarrhœa, emaciation, and gradual weakness. In some cases death takes place after a few days' illness, before marasmus has had time to develop. In more chronic instances the disease runs a slower course, when cachexia sets in and carries off the animal. The subacute form is usually termed by fanciers "gotch," "pot-belly."

Nasal Coccidiosis, Psorospermal Catarrh, or Snuffles.—This is usually associated with intestinal coccidiosis, and liable to be confounded with those septicæmic maladies of the rabbit manifested with catarrhal discharge from the eyes and nostril. Coccidiosis may, however, complicate these diseases (infectious rhinitis, malignant catarrhal fever).

The affected animals are not so active or bright as usual; they are dull, and have very little appetite, and their coat is dull and open. They sneeze or snuffle. The nostrils discharge at first a thin serous or clear sero-mucous material, which afterwards changes to a profuse thick, sticky muco-pus that adheres to the nostrils. There is also a flow of saliva from the mouth that soils the lips, shoulders, and feet, which parts the animal licks almost without cessation. There is frequent sneezing and coughing, and the animal often rubs its head with its front feet or against some obstacle. The rise of temperature is at first slight, but as the disease advances it goes several degrees above the normal. The breathing becomes difficult, and ultimately the sides lift or have a pumping action. The appetite gradually disappears; emaciation and great weakness set in; the animal grinds its teeth, and death supervenes during convulsions.

In many cases there may be pharyngitis, then mastication and deglutition become difficult or impossible. It is accompanied by a rattling or gurgling sound emitted from the throat. At times it may be associated with otitis media, which is only seen when the ears are infested with *Psoroptes communis* var. *cuniculi*. Nervous symptoms, such as strabismus, vertigo, torticollis, etc., may supervene.

At other times there may be conjunctivitis, evidenced by a swollen condition of the eyelids and a discharge of thick, purulent material.

Lesions.—In chronic cases there may be extreme emaciation and a profound anæmic appearance of the tissues; the blood may be watery and pale.

The *liver* may, in chronic cases, be enlarged and deformed, and show on its surface a number of yellowish-white raised nodules, which vary from the size of a poppy-seed, millet-seed, or hemp-seed, to a pea, or even to a hazel-nut. When the nodules are discrete, they contrast well with the red hepatic tissue. They are circumscribed, elongated, or curved in appearance. Sometimes the organ is only enlarged in one of its lobes, but generally the whole is involved. When the liver is cut into, the nodules are usually found irregularly scattered through the hepatic substance; or they may be so numerous that very little of the normal tissue remains. When the nodules are incised or punctured, a quantity of whitish purulent fluid or yellow caseous material exudes, containing coccidia, degenerated epithelial cells, fat, and crystals. On microscopical examination, the nodules are found to be local dilatations of the bile-ducts. In certain instances the dilatation is almost uniform, but in the majority it is varicose or very irregular. The lumen of the dilated duct is almost occupied by coccidia in various stages of development, mingled with some granular material or residue of epithelial cells and leucocytes. Now and again the interior of the dilatations is lined with an even mucous membrane, but generally cavities are partly occupied by verrucose vegetations, springing from the mucosa. The submucosa is then infiltrated with small cells. Occasionally there is total cirrhosis, the new tissue containing a number of minute bile-ducts. The epithelium lining the dilatations is always invaded by the parasites, which are found in various stages of development in the cells, between the cells, or covering their free surface. The invaded cells do not proliferate, but soon succumb, degenerate, and disappear; in this cases the epithelium is found absent in the part attacked. The parasites found in the lumen of the dilated duct vary in size from 5 μ upwards, according to their stage of development, all stages being present. McFadyean has observed them occupying the vein. They are also found in the gall-bladder, which may, in rare instances, become dilated, have thickened walls, and its epithelium invaded with the parasites.

In many instances the ducts are obliterated, and cysts appear, the interiors of which become intersected, so that they contain honeycomb-like divisions. When the nodules are numerous and confluent, the interposing hepatic tissue undergoes atrophic changes.

In the *intestine*, especially when the disease has concentrated itself on this organ, there may be found small whitish punctiform or more or less larger spots, mostly on the mucous membrane on the small intestine. They are also observed in the cæcum, and especially in the inferior cul-de-sac. In the contiguous parts the mucous membrane is the seat of a catarrhal inflammation, which is sometimes very extensive, and may show some small but distinct ulcerations. In the exudation covering this membrane degenerated epithelial cells and fat globules, and also numerous coccidia in all stages of development, are found mingled with particles of ingesta. In the more serious cases the intestine is invaded by whitish spottings, which occupy almost more space than the normal tissue. The spots persist after the mucus, and even a slight scraping has been removed. On section these spots correspond to a group of coccidia inhabiting the epithelial cells of the villi and glands of Lieberkühn, which are sometimes enlarged to double their size, in consequence of their containing such a great number of the parasites. The surrounding submucosa is involved in inflammatory changes, and may contain coccidia. The mesenteric glands may also contain immature coccidia.

The liver may alone be involved, or the intestine only; or both organs are attacked at the same time. Sometimes the liver is only slightly affected and the intestine extensively, and *vice versa*. Similar lesions are found in the hare.

In the nasal form of coccidiosis the nasal and pharyngeal mucous membranes may be found inflamed, or the vessels deeply injected. The nasal channels contain a more or less reddish or orange liquid, made up of red and white blood-corpuscles, pus globules, and coccidia.

There may also be small extravasations or larger blood-clots. The mucous membrane lining the middle ear, the tympanum, and the external auditory canal, contains a discharge more or less glairy, muco-purulent, or hæmorrhagic. This material, like that of the nostrils, also contains coccidia. Coccidia may be found in the Eustachian tube, as well as in the interior of the epithelial cells of the mucous membrane. They may also be found in the subepithelial tissue. When the tympanum is perforated and the inflammatory process has spread to the internal ear, there may be injection of the cerebral meninges.

Diagnosis.—At first sight hepatic coccidiosis might be mistaken for tuberculosis, pseudo-tuberculosis, or cysticercosis; but a microscopical examination of the lesion enables one to detect the coccidium. The palpable lesions of coccidiosis are confined to the liver or intestine, whereas in tuberculosis and many of the pseudo-tuberculoses the nodules, or tubercles, usually attack several parenchymatous organs at the same time.

During life, examination of the fæcal material enables one to detect the parasites if present. In the nasal form the presence of coccidia in the discharges would be confirmed by microscopical examination, but in the present state of our knowledge on this subject it would be difficult to affirm if the disease were one of coccidiosis or some other infection, with which coccidia may be associated accidentally.

Treatment.—Freshly-purchased rabbits, especially if in a rather bad condition, should not be introduced into a rabbitry unless they have been isolated for at least a fortnight, or their fæces microscopically examined for coccidia. Old hutches should not be purchased. Scrupulous cleanliness and regular feeding, without any sudden change of dietary, should be observed. The sudden giving of freshly-gathered green food with the moisture on it is a fruitful source of the malady in very young rabbits. This is especially noticed when it has been denied them for a long time. The general experience of rabbit-fanciers is that it is much safer to give green food when it has lain for some time and become somewhat withered or "wilted."

When the disease is very virulent, the giving of iron sulphate or copper sulphate, dissolved in water and sprinkled on the food, is spoken highly of. An infusion of catechu, logwood, or cloves, might be tried. Even the bark of oak, juniper, spruce, and such-like terebinthinous plants, may be offered to the rabbits to nibble. Freshly-gathered green broom (*Cytisus scoparius*) used to be spoken highly of, but the writer never saw any benefit derived from its use.

Rivolta advises hyposulphite of soda or sulphur. Calomel or iodide of potassium has also been advised.

In the case of rabbits in a wild state nothing much can be done beyond the planting of suitable shrubs having a depurative effect on the rabbits nibbling the bark. Catching the rabbits and killing them off is an impracticable method, because the coccidia cannot be killed in the soil, or in consequence of 92 per cent. of rabbits being the bearer of the parasites.

Coccidia outside the animal body are very resistant to ordinary disinfectant agents, but from this one should not conclude that medicinal agents do not have any deleterious effect on their growth when infesting the system. A very low temperature and absence of moisture retards their growth, but, nevertheless, may prolong danger to infestation.

Burning hutches with a painter's lamp is a very effectual method of destroying the parasites. Freshly-slaked lime is also effectual.

Public Health.—Numerous cases of coccidiosis in man are on record. Some authorities have suggested that man has obtained the diseases by ingesting some food or water soiled with the fæces of infested rabbits, but there is no reliable evidence to confirm what is a mere assumption. The disease being so widely spread in rabbits belonging to children, one would expect it to be more prevalent in the human species than it is—that is, if the view of its transmission from the rabbit were well founded.

II. COCCIDIOSIS OF BIRDS.

Synonyms.—Protozoal enteritis; Hæmorrhagic enteritis; Psorospermosis or gregarinosis of birds; Typhilitic coccidiosis; White or chalky diarrhœa of chicks; Enlargement of the liver, with non-absorption of the yolk-sac in the chick; Spotted liver; Blackhead; Infectious entero-hepatitis; Perityphlo-hepatitis of turkeys; Going-light; Limber-neck; Leg-weakness; Dysentery.

French: Coccidiose des oiseaux; Typhlite coccidienne.
German: Coccidiose; Coccidiosis intestinalis.

History and Geographical Description.—Coccidia in birds was first described by Rivolta in Italy in 1878. This authority found them in various birds, such as the fowl, crow, blackbird, and the smaller passerine birds. According to the description he gave at the time, there were several species of these organisms, such as *Gregarina avium intestinalis* (*Eimeria dubia* Railliet), *Psorospermium avium* (*Isospora avium* Railliet), and, later, *Coccidium Rivoltæ* (*Eimeria* (*Coccidium*) *avium*). Piana, Silvestrini, Perroncito, Railliet and Lucet, and numerous others, have since given great attention to the rôle these organisms play in the production of disease, and their life-cycle. Podvissotzky junior and also Eckardt, have studied the part the extra- and intra-infected egg plays in the spread or maintenance of the malady. McFadyean and Sjöbring have described coccidiosis in the pheasant. Theobald Smith, Veranus A. Moore, Cooper Curtice, Railliet and Lucet, Jowett, and others, have studied the disease in the turkey. Fantham, in investigating the cause of mortality among grouse, observed that the greatest fatality in grouse chicks was due to coccidia.

The disease is widely spread in Europe, Asia, Africa, America, Australia, and in other parts of the world. It is commonly encountered in all species of birds in the United Kingdom, where the investigation of bird diseases is generally left to those absolutely ignorant of pathology and bacteriology, thanks partly to the commercial spirit of the avian press, and partly to the apathy of the veterinary profession.

Causal Organism.—The species of coccidial parasite commonly causing coccidiosis in birds is *Eimeria* (*Coccidium*) *avium* Silvestrini and Rivolta (*Psorospermium avium* Rivolta 1878; *C. Rivoltæ* Harz 1886; *C. tenellum* Railliet and Lucet 1891; *Amœba meleagridis* Theobald Smith; *Hæmamœba Smithi* Laveran and Lucet).

This delicate coccidium is more compact than *E. oviforme* or *E. perforans*, often rounded but mostly rather ellipsoidal than ovoidal, the two poles being equal in breadth; the cyst wall is thinner or more delicate; the dimensions are less, averaging from 21 to 25 μ in length by 17 to 19 μ in width—at least, in the last stage of development. Bigeminal forms are sometimes encountered. According to Pfeiffer, the coccidium of the pigeon measures 18 μ in length by 16 μ in width. To the parasite of this species Labbé gives the name of *Eimeria Pfeifferi*, which is probably only a variety.

Theobald Smith believed the micro-organism discovered by him in cases of blackhead disease in the turkey to be an amœba (*Amœba meleagridis*), resembling the amœba of dysentery in man. Cole and Hadley, however, consider this Smith's amœba is, in all probability, the schizont stage in the development of a coccidium. These two authorities succeeded in inducing the disease experimentally in turkeys, chickens, and sparrows, by feeding them with portions of cæcal contents or parts of cæca of diseased turkeys. In the fresh state in the intestinal contents small bodies are observed measuring from 10 to 12 μ in diameter. In the liver and cæcal wall they may measure from 4 to 10 μ in diameter, but in rare instances only 3 μ in diameter. Larger bodies of an oval shape, measuring from 17 to 20 μ in length by 12·8 to 16 μ broad, are also detected in the liver lesions.

This parasite invades the epithelial cells lining the internal face of the gut wall, especially that of the duodenum, and also of the cæca, in those birds possessing these appendages. In very young chickens or those only a few days old they also invade the liver. The albumin in the interior of the egg is not exempt from their presence.

Life-Cycle.—The life-history of this coccidium is similar to that of the other coccidia. It usually runs a complete course, from eight to ten days, from the time of entrance of the cysts into the bird's system until a new generation of cysts is evacuated from the cloaca. Birds may get reinfested.

Railliet and Lucet, feeding two chickens, from two to three weeks old, on the falciform corpuscles obtained from the cæca of chickens whose death were brought about by multiplication of the parasites, produced death at the end of twenty and thirty-one days, with numerous well-developed coccidia in the cæca. The two controls showed no lesions.

Eckardt infected chickens of five weeks of age, young pullets, aged hens, and two cockerels one and a half years old. These animals were seized after an incubation of three to fourteen days, and died in four days, three to four weeks, and two months. Only one cockerel recovered. In some of those birds showing profound emaciation before death hepatic coccidiosis was found on post-mortem examination.

Spread of Contagion.—The fruitful source of the disease is purchase of birds, apparently healthy, giving off the parasites from their intestine and contaminating the farmyard, poultry pen, or aviary. Eggs soiled on their outer covering with fæcal material containing the parasites, and also eggs containing the parasites in their interior, are not rare means of introducing the disease. Old cages, baskets, crates, fowl-houses, incubators or rearers, and such-like, purchased at sales, are not free from blame. Rearing birds on too confined a space or on the same ground two years running account for many outbreaks. Pigeons and other birds coming from an infested place may introduce it into a clean farmyard. Incubator-hatching establishments trafficking in one or more day old chickens widely disseminate the disease.

Markets, bird-shops, and shows also assist in spreading it. The parasites are taken in with the food or drinking-water.

Jowett believes *Eimeria oviforme* is capable of producing coccidiosis in young chickens under certain conditions, but there is no evidence to show that the rabbit under natural conditions is capable of spreading their infestation to birds. Mice, rats, insects, wind, etc., may be the media of contagion. Dirty moist floors and perches and dirty feeding utensils favour the spread of the disease.

Species generally attacked.—The fowl, turkey, pheasant, swan, guinea-fowl, peafowl, quail, partridge, grouse, duck, wild-duck, goose, pigeon, and wood-pigeon, are very commonly infested. Of the smaller passerine birds, the canary, and the usual cage birds, are not rarely victims to it. A great number of wild birds confined in aviaries frequently succumb to its inroads. The grouse at liberty is not exempt; in this species it destroys a very great number of the young chicks. Carnivorous as well as graminivorous, seminivorous, and frugivorous birds are equally susceptible.

Age and Susceptibility.—Chickens a few days old are very susceptible, and the mortality is then very great, especially in those hatched in incubators. Young pigeons just on leaving the nest mostly succumb. The older the bird, the more removed the species is from selection, and the greater the liberty allowed, the more resistant to the disease it seems to be.

Young canaries rarely survive its inroads; also birds captured from a wild state and confined in cages for the first time. The susceptibility in wild species, however, varies, and the mortality may even be very small. It is supposed to destroy the internal protective layer of the intestinal tube, so that in very acute cases the saprophytic organisms of the intestine gain the circulation and bring about a rapid septicæmia.

Seasons and Climate.—No country in the world where domestic birds are reared is exempt from this malady. It occurs just as often in tropical and subtropical climates as it does in the more temperate ones. Hot, dry weather, especially in spring months or the early summer, is very favourable for its progress. The mortality is very great if the young chickens are kept in a very warm rearer. Chilly weather, however, also brings it into evidence. Change of weather frequently puts a stop to the spread of infestation. Birds kept in cages in the house may develop the disease, whatever the state of the weather may be outside. Wet weather or moisture and a low temperature delays the development of the oöcysts and sporocysts, but in consequence the power of infection is maintained for a long period.

Symptoms.—In chickens only a few days old the appetite is suppressed, the little creatures are constantly crying, seem restless—incessantly on the move; the comb or beak turns to a leaden hue, the bird becomes dull, and quickly succumbs. At this age there seems to be no diarrhœa. This form is commonly noticed in chickens reared in the incubator, when hundreds—nay, thousands, or all those hatched in this manner in a large establishment—may succumb. Poultry-farmers usually ascribe the mortality from this cause to the *unabsorbed yolks* or an *enlarged liver*.

In older chickens, or those of five days to a fortnight or more, the disease is markedly manifested by a whitish or chalky material adhering to or soiling the cloaca or down in that vicinity (*White Diarrhœa*). This somewhat resembles the milky white fæces of centipedes suffering from the acute stage of coccidiosis. The young creatures cry, appear as if constipated, and frequently strain in order to get rid of the source of irritation; they waste away, or rather do not thrive, appear dull, the wings droop, and the chickens seem to be continually looking for food, which they often do not touch, and eventually, but somewhat suddenly, succumb. In other cases there may be mopiness, a ravenous appetite, great thirst, rapid wasting, retardation of growth, prominence of the breast-bone, palish fluid diarrhœa, followed by death. When the disease is slight, the fæces are soft and brownish-yellow; in very acute cases yellowish and very fœtid.

In chronic cases the featherless parts are pale; this progresses until they have a bloodless aspect, and changes just before death to a peculiar bluish tint. The feathers do not seem to develop, but often fall out. Paralysis of both legs often occurs. The mouth often gives off an offensive odour, and frequently contains a glairy mucus. It often disappears to reappear. Just before death there may be a discharge of this material from the eyes, nostrils, mouth, and ears. The fæces of the birds contain oöcysts.

In birds that are fairly well feathered there may be at first constipation, then a copious diarrhœa, which is sometimes bloody or brick-red in appearance. At other times it is at first whitish and afterwards greenish. They have a dull, open plumage; the wings droop; they get into corners or hide themselves under obstacles, are indifferent to their surroundings, have a bluish comb, give off an abundance of saliva from the mouth, and die in two or three days. In adult birds death may be delayed two or three weeks, or the disease may pass into a chronic state. Emaciation is usually profound, gait difficult, and appetite capricious. Sometimes death takes place suddenly before any clinical symptoms can be observed.

Pheasant.—The clinical appearance is similar to that of the fowl. Bloody diarrhœa is rarely observed in birds under four or five weeks of age.

Duck and Goose.—There are often all the clinical appearances of enteritis, followed by profuse diarrhœa, great weakness, rapid emaciation, and a speedy death, which is sometimes apparently sudden. The fæcal material is sometimes bloody. The duck may be the bearer of these parasites without showing any manifest evidence of their presence.

Turkey.—It is a very common and often fatal disease in young turkeys, especially in those from three weeks to three months old, although they may succumb at any age. There may be dulness, drooping of the head, wings, and tail, mopiness, loss of vivacity, capricious appetite, loss of condition, and tardiness of growth. The fæces are generally liquid and greenish. In some instances the appetite is good, and the birds thrive fairly well. The featherless parts of the head frequently assume a dark bluish or bluish-black colour, and from this fact the disease is usually known as "blackhead."

Grouse, Quail, and Partridge.—The clinical characters are similar to those obtained in the fowl.

Canary and Other Cage-Birds.—In young nestlings there is a sickly diarrhœa; the birds do not thrive, and often become deformed in their legs. Older birds have a diarrhœa of a greenish tint and of a sickly odour. Very often the cloaca is painful and hot, and the bird exhibits straining movements of the vent, as if to free it of some irritant. Death is sometimes sudden. In the chronic form there is profound emaciation, which is usually termed by bird fanciers "going light." The infested birds usually have a difficulty in moulting, are sickly, liable to catarrh, and usually end up by succumbing.

Pigeons.—These birds show similar symptoms to those in other birds. Young pigeons in the nest have a dirty, pasty diarrhœa, which soils them and the nest. They do not thrive, often become cripples, and die before they have commenced to feed themselves. Even when they have reached adult life they do not seem to have any idea of feeding themselves. In the peracute form it somewhat resembles avian cholera in clinical appearances.

Birds of all species, when they have reached adult life, may be bearers of coccidia without manifesting any clinical appearances of disease. When present during the moulting period the moulting process is often retarded or difficult, the birds not rarely becoming sickly or chronic invalids, and finally succumbing. As in the sickly birds, the fæces, on microscopical examination, may be found to contain oöcysts. They may be present in birds suffering from other infections, so their mere presence cannot always be depended upon as an indication of the cause of disease.

Coccidia in the Egg.—Eggs collected in the spring, summer, or autumn months of the year in those localities where coccidiosis abounds may contain coccidia in the albumin of their interior. So far as is known, they are only found in the albumin. On section of a hard-boiled egg, the parasites appear as small greyish-brown, blackish, or yellowish specks, reaching to the diameter of a pin's head. On naked-eye examination they are liable to be confounded with pigmentary spots or a minute particle of the yolk. In eggs that have been kept for some time the parasites give them a somewhat rotten appearance. Microscopical examination after staining with hæmatoxylin shows these specks to be coccidia undergoing every stage of evolution. It is supposed that the parasites gain the interior of the egg before it reaches the terminal or shell-forming portion of the oviduct. The external covering or shell of the egg may also be contaminated with coccidia. It is therefore quite possible for the chick to become infested during the fœtal stage, or soon after hatching, and this would explain in a great number of instances why young chicks a few days old succumb to the malady, especially in incubator-hatching or brooder-rearing establishments, where air and light are insufficient.

Probably the insufficient boiling of eggs for the purpose of feeding young canaries and other cage-birds accounts for so many deaths from this cause.

Course.—In certain outbreaks the affection runs such a rapid course that many of the birds are found dead before the disease has been observed. In other cases the birds will linger for a few days and then succumb. In many instances, especially in the case of more resistant or older birds, death follows after a few weeks, when the birds will be found to be very anæmic and much emaciated. In young pigeons on the nest the course of the disease is usually slow.

Mortality.—This is usually very great in very young birds, especially in those artificially hatched and reared. It is also great when introduced for the first time to a yard of highly-bred birds. The same may be said of the pheasant. It often reaches 100 per cent. The writer has seen every one of a hatching of 200 succumb in a few days. Biernacki observed an outbreak of intestinal coccidiosis in a flock of 320 geese destroy 100 in the space of four days. The mortality, however, is often erratic, killing only a very few out of a large number, when at other times the majority, if not all, succumb.

Lesions.—The carcass of the bird is usually emaciated, soft, and lustreless. The feathers or down near the cloacal opening or vent are soiled with liquid or adherent lumpy fæcal material.

The lesions caused by the presence of the coccidia are usually confined to the intestine, but in many instances the liver becomes secondarily involved.

The mucous membrane of the *small intestine*, and in particular the *duodenum*, is of a dullish red colour ; its surface is dotted with irregular whitish streaks or spots, either isolated or confluent, varying in number, and which contain the coccidia. The intestinal contents, which are usually not great in amount, may be stained by a reddish fluid, which contains a great quantity of encysted or free coccidia. The invaded epithelium is denuded in parts, or in severe cases entirely. The epithelium lining the glands of Lieberkühn is invaded, and the lumen of the glands filled with coccidia, immature as well as mature. In many acute cases almost all the epithelial lining of the whole length of the duodenum may be found to be destroyed. In cage birds the duodenum often has a distended blackish appearance, due to the hæmorrhagic contents, which contain oöcysts and merozoites in immense numbers.

In the *large intestine* the cæca are often affected; in fact, in a great many cases the disease is confined to these appendages—at least, in those birds possessing them. The morbid appearances vary according to the age of the bird or its resisting power. In young chickens or young pheasants from a few days old to two or three weeks the cæca offer no congested appearance, but whitish spots containing coccidia may be seen through the clear walls of the gut, in the lumen of which is found a compact or cheesy yellowish exudate distending the viscus. In birds two or three months old the mucous membrane, which is more or less widely deprived of its epithelium, is intensely reddened, and shows here and there small ulcerations; the cæcal contents have a putrescent odour, are of a brick-red colour, and fluid in consistence; the fæcal material contains a few coccidia, altered epithelial cells, red and white blood-corpuscles, and a quantity of fat globules. In young birds, at least those under a month, the intestinal contents are not usually sanguinolent. In the young turkey the commonest seat of the lesions is in the blind end of the cæca. Sometimes only one cæcum is involved, the other remaining healthy. These organs are very much enlarged and thickened. The adenoid tissue between the tubules and in the submucosa is greatly increased, and contains in its meshes one or more groups of the parasites. In advanced cases the serous membrane is inflamed or congested, and the mucous membrane is shed and the cæcal lumen filled with an isolated or continuous adherent yellowish-white mass, built up of concentric layers, composed of fibrin, blood-corpuscles, and other elements. In advanced cases the gut wall is from seven to ten times its normal thickness. Sometimes yellowish exudates are found on the serous coat, which bring about adhesion of one cæcum to the other, to the intestine, or to the abdominal wall. When these advanced lesions are found, very few coccidia are discoverable, in consequence of them having left their habitat after doing all the damage to the tissues they could. In the pigeon dead of the acute intestinal form, subcutaneous hæmorrhages are often pronounced in the cervical region, all the organs are congested, and the intestines distended either with gas or catarrhal exudate.

In the *goose* the intestinal contents may be found to have in part a dirty greyish-white appearance, and in part bloody, liquid, fœtid, and holding in suspension greyish nodules, which may reach the size of a hemp-seed. The mucous membrane has similar nodules scattered over its course, and also small ulcerations. Numerous coccidia may be found in the mucous membrane, and also in the intestinal contents. In those cases running a chronic course the destruction of the epithelium is found so widely spread and the submucosa infiltrated so as not to be able to regenerate itself, and in consequence the bird becomes exhausted and dies from inanition.

The *liver* is secondarily involved. In very young birds, attacked soon after hatching, this organ is very much enlarged, hyperæmic, and very friable. It may then contain the parasites, which in older birds are not often found in the liver. In the turkey, and to a lesser degree in the fowl, the liver is generally, but not always, involved. The organ is enlarged, perhaps, to two or three times its normal size; its outer surface is studded with several or innumerable circumscribed discoloured spots or patches, which show a distinct line of demarcation from the normal hepatic tissue. They may be widely and uniformly distributed over the whole surface of the liver, or they may be confined to a few parts. They may vary in diameter from 3 to 15 millimetres, to that of a millet-seed, a pea, or a shilling. In the earlier stages they are of a lemon, greyish, or ochreous yellow colour; in other instances they may be of a mottled brownish colour, containing a central yellowish nucleus; in other cases they are uniformly whitish, and gradually lose themselves in the surrounding hepatic tissue. Some of the spots are deeply imbedded in the liver, and therefore are not visible on the surface. At times large portions of the liver are destroyed. These areas are not always homogeneous in structure, but made up of a fine network of greyish-yellow necrotic tissue. The liver cells are, in the earlier stage, partially or totally absent from these necrotic areas, and the coccidia are found in the meshes of the tissue. Later on the parasites are rarely found. Similar lesions of coagulation necrosis are sometimes also found in other birds.

The *lungs* sometimes show lesions which contain coccidia. Coccidia may sometimes be found in the crop, probably ingested with the food.

Diagnosis.—The peracute forms of this disease are liable to be mistaken for avian septicæmia, avian cholera, and avian plague. As a rule the two former attack fowls of all ages, and the blood of the living birds contains numerous bacteria. In *avian plague* the blood appears sterile, and the fæcal discharge, as in the case of avian septicæmia and avian cholera, is free from coccidial parasites.

It may also be mistaken for strongylosis, tæniasis, or ascariasis. But the absence of the parasites in the intestinal contents, and the presence of coccidia, are fairly certain points to distinguish the disease. One must not, however, conclude absolutely from the presence of coccidia in the fæcal material that they are the cause of death, as often in birds, especially adult ones, these parasites are present without causing any signs of ill-health, and they may also be in association with other parasitical infestations. As a rule coccidia produce death in very young birds.

Microscopical examination of the cæcal walls and the fæcal material will enable one to find coccidia if they are present. In chronic cases, however, the parasites, after damaging the tissues, leave their host, and the saprophytic bacteria invade the resulting lesions, and in consequence continue the morbid process, and perhaps terminate the existence of the bird.

Treatment.—Prevention should be aimed at, but this can only be fairly obtained by microscopical examination of the fæcal material of newly-purchased birds, which should always be isolated at least for a fortnight before they are allowed to commingle with other birds of the farmyard or aviary. Newly-purchased eggs for sitting purposes should be washed with methylated spirit, allowed to dry, and then dipped in 1 per 1,000 of sublimate.

Incubator trays and drying boxes should be washed with a 5 per cent. solution of lysol, and then dried before being used for hatching purposes. Rearers or brooders should be dressed inside with the green creosote oil of commerce or formalin, allowed to dry, and then washed over with an emulsion of freshly-slaked lime. Coops and hatching boxes should be similarly treated. New ground should be utilized every year for hatching and rearing of pheasants, turkeys, and even chickens. Fowl-houses and perches should be scraped, creosoted, and then lime-washed. The floor should be scraped and sprinkled with freshly-slaked lime. Market pens, baskets, cages, and crates, should be disinfected after thorough cleansing. Where practicable, sparrow-proof wire netting should be utilized in the wiring of coops, pens, and aviaries, in order to prevent small birds getting in and spreading infection. Pigeons should be trapped or confined. The disease should be scheduled under the Diseases of Animals Act, in order to put a stop to the trafficking of infested birds, especially one or a few days' old chickens, which are a fruitful source of dissemination of the disease. When an outbreak occurs, the birds should be submitted to medicinal treatment, and after they have discontinued passing oöcysts in their fæces they should be put in clean pens or put on to fresh ground. At least, the pens or ground should be well sprinkled with freshly-slaked lime. The parasites are capable of resisting destruction for months, and even as long as two years. Sulphuric acid is advised as a good disinfectant for the destruction of coccidia.

Curative Treatment.—It is not necessary to destroy the affected and contaminated birds, as suggested by many authorities, as, if suitable remedies are adopted, recovery of the majority of those affected will be obtained. It is impracticable to force pills or potions on individual birds; simply dissolving the intended medicament in the drinking-water is sufficient. One of the oldest remedies is ferrous sulphate, given in the proportion of 10 grains to the gallon. Some speak very highly of potassium permanganate, gradually added until the water is permanently stained. Others recommend sodium salicylate, or even salicylic acid. Zürn speaks highly of glycerine, but this is only applicable to a few birds or to those confined in cages. Sulphuric acid, hæmatoxylin, and cloves may be given. Paraffin or oil of turpentine mixed with the food seems very efficacious. Lime-water might also be tried.

The Grouse Commissioners, in experimentally or naturally infected birds, including fowls, pheasants, grouse, pigeons, geese, ducks, turkeys, and canaries, found that crude catechu given in the drinking-water brought about recovery in nearly, if not all, the affected. It was given in the proportion of 10 to 15 grains to the gallon to the larger species, and continued for ten to fourteen days, when, if the voiding of oöcysts had entirely ceased for several days, the dosing was discontinued. If, on the other hand, passage of oöcysts continued during the ten days' treatment, though steady decrease in their numbers occurred, the birds were then given water containing 5 to 8 grains per gallon, until practically no oöcysts were found in their fæces. In very severe cases, treated late, as much as 20 grains per gallon was administered; but as a rule this strength was too great, causing the birds to be constipated. Pigeons received 10 grains, and canaries 5 grains, per gallon. The birds showed no dislike of the catechu-water, but drank it readily. This simple method should be tried on other animal species.

RENAL COCCIDIOSIS IN THE GOOSE.

This disease has been described by Railliet and Lucet. It is due to a special coccidium, *Eimeria* (*Coccidium*) *truncata* Railliet and Lucet. This parasite somewhat resembles the *Eimeria* (*Coccidium*) *avium*, but it is slightly rounder and less in dimensions, being 20 to 22 μ by 13 to 16 μ. At the narrower pole there is a distinct micropyle. Some of the coccidia contain a granular protoplasm uniformly distributed in the oöcyst; others have the protoplasm formed into a ball in the centre.

Symptoms.—The first noticeable thing observed was the progressive emaciation without any apparent cause. After a certain lapse of time the birds could scarcely walk, standing with difficulty, but resting with their breasts mostly on the ground. At times they would assume most peculiar attitudes by turning themselves on their backs with their feet wide apart in the air. When placed on their feet, they took a few steps, and then fell again and assumed their former dorsal position. In every case they finished up by no longer taking food, and then succumbing.

Lesions.—The kidneys were generally crowded with small whitish nodules, the size of a pin's head. More rarely the lesions were diffused.

The nodules were constituted by a considerable mass of coccidia, free or encysted. On section the coccidia were found inhabiting the uriniferous tubules. In the earliest evolutionary stage of the parasite it took on the form of a circumscribed, nucleated, granular body, situated in the interior of the epithelial cells, and pushing the nucleus of the cells towards the base. In some cases only a single coccidium occupied a cell, but generally two or three were found. In the latter case the parasite seemed to have multiplied by repeated division; and these multiple bodies were often polyhedral in consequence of the compression they underwent on every side, thus forming a series of radiations. When they were removed accidentally, they left a kind of a granular matrix, which might be taken for the cell proper; but a nucleus was never observed. At other points the epithelium of the uriniferous tube had disappeared, and in its place coccidia were found, which were already surrounded by an envelope having a double contour. In other parts well-formed coccidia were observed in the lumen of the tubules, where the epithelium remained intact; these parasites were evidently developed higher up the tubule, and probably being discharged. Railliet and Lucet satisfied themselves that these coccidia were following the course of the urine, and were expelled with it, to undergo further evolution outside the animal body.

By keeping them in water for a certain time, these authors found that the protoplasmic mass divided itself into four circumscribed sporoblasts, like those of the group of tetrasporous coccidia of Aimé Schneider, or genus *Eimeria*. Mesnil believes that the coccidia reach the kidneys by the circulation, which they gain by passing through the intestinal walls after ingestion; they therefore should rise to a generalized blood-infestation. *Eimeria* (*Coccidium*) *truncata* may also be observed as a parasite in the intestine of the goose ; but intestinal coccidiosis does not necessarily accompany renal coccidiosis.

Treatment.—No treatment is known for this type of coccidiosis, but Biernacki found that in intestinal coccidiosis in the goose sulphate of iron and glycerine added to the drinking-water gave good results.

III. COCCIDIOSIS OF THE OX.

Synonyms.—Intestinal psorospermosis ; Intestinal coccidiosis ; Protozoal gastro-enteritis ; Bloody flux ; Red diarrhœa ; Red dysentery ; Hæmorrhagic enteritis.

French : Coccidiose intestinale du bœuf ; Dysentérie rouge ; Entérite hémorragique ; Flux de sang.

German : Coccidienruhr ; Darmcoccidiose ; Coccidiosis intestinalis ; Dysenterica hæmorrhagica coccidiosa.

History.—Although dysentery or bloody flux in cattle has been recognised from antiquity, there seems to have been no mention of it being due to coccidia until 1877, when Zürn discovered the parasites in the lesions in the intestine and mesenteric glands from a calf forwarded to him by Pröger, Departmental Veterinary Officer for Borna, who had encountered the disease in four calves, aged from five to six weeks. In 1890 and 1891 E. Zschokke, of Zürich, studied a dysenteric disease which was very prevalent in young cattle and oxen in the cantons of Aargau and Lucerne. Hess and Guillebeau, of Berne, about the same period, also paid great attention to the malady. These three eminent authorities found in every case intracellular parasites, mostly circular in outline, but the larger examples being decidedly oval in shape, and concluded the micro-organisms were coccidia, which did not exist in the fæces or bowel of healthy cattle, although in contact with the affected. In 1898 G. Gair, of Conon Bridge, N.B., observed cases in two-year-old and adult cattle. Besides the classical symptoms outlined by the Swiss authors, Gair's cases also manifested swelling of the limbs. McFadyean, to whom a portion of the large intestine was sent, attributed the disease to a coccidium, which was present in great numbers in the inflamed areas of the mucous membrane. In 1902 Pieroni encountered the disease in Italy. In 1903 Degoix studied a prevalent epizoötic affecting young cattle, aged from six months to two years, in Avalon and the surrounding districts. On microscopical examination of the dejecta and of the inflamed mucous patches on intestine, numerous coccidia were revealed.

In 1905 E. Ducloux observed similar facts in young cattle in Tunis. It was encountered by Storch in Germany during 1905. Marotel in 1907 found it in the calf. In 1910 R. E. Montgomery drew attention to the presence of coccidiosis in British East Africa, and warned observers in Uganda and the Soudan to be on the lookout for this disease, which was spreading north from German East Africa. The mortality was usually 10 to 20 per cent. of the affected, especially in young calves and badly-nourished young stock, deaths being rarely observed in strong, healthy, or full-grown animals. In young calves it generally ran its course in three to five days; in older animals, seven to ten days on an average. This peracute form of coccidiosis closely resembled rinderpest clinically, as it did in the Swiss epizoötic. Jowett encountered a case of red diarrhœa in a calf at Rosebank, Cape Town, during the year of 1910; in the fæces there were coccidia—*Coccidium bovis.* It may be added here that this author considers he has been able to infest young chicks with the coccidium of the rabbit—*Eimeria cunicula.* Züblin was unable to convey the coccidium of the ox to the rabbit. This disease has also been observed in the United States of America and other countries besides those mentioned above. No doubt, on further investigation into cattle diseases, this affection will be found to be more widely spread and more prevalent than seems to be the case at present.

An enzoötic form of hæmaturia or hæmorrhagic cystitis has been attributed to coccidia.

Causal Organism.—The incriminated parasite is denominated *Eimeria* (*Coccidium*) *Zürni* Rivolta (*Cytospermium Zürnii* Rivolta 1878; *Coccidium perforans* var. *bovis* Railliet; *Coccidium bigeminum* var. *bovis* Stiles). The prevailing type in the intestine of the ox is round, but in larger examples it becomes distinctly oval in outline. The oöcysts measure 18 to 30 μ in length by 13 to 20 μ in breadth, and sometimes more. They are found in variable numbers in the fæces, singly or in groups in shreds of mucus, much more abundantly in the mucous membrane, and not at all or very rarely in pure coagulated blood. Zschokke, in a 1 millimetre section through the rectum, found as many as 1,500 parasites, and calculated that in a square millimetre of the thickness of the bowel there were 150,000, and in a square centimetre of the surface of the mucous membrane 15,000,000 of coccidia. In the majority of examinations carried out by Montgomery only merozoites were discovered. They occurred either free or arranged within the epithelial cells like the quarters of an orange. In fresh cover-glass preparations the merozoites appeared as comma-shaped bodies of a pale straw colour, capable of flexion and extension, but of limited progression. The oöcysts measured on an average 17 by 15 μ, were clean and transparent, with a definite contour, and contained fine granules, frequently arranged towards the pole opposite the micropyle. Marotel found in the superficial layers of the intestinal mucous membrane of a calf whitish granulations, scarcely larger than a pin's head, globular in outline, 200 to 300 μ in diameter, which were constituted by a fairly thick envelope and innumerable, slight curved, spindle-shaped or crescentric corpuscles, of 8 to 14 μ in length by 1·4 to 2 μ in thickness, formed of a mass of cytoplasm surrounding a nucleus. In consequence of the resemblance of these nodules with the asexual forms of reproduction of *Eimeria Faurei* of the sheep, Marotel regards them as the merozoite forms of a coccidium of the ox, and probably of *Eimeria Zürni*. The endogenous cycle of *Eimeria Zürni* is unknown. Ducloux has seen a form which he regards as a microgametocyte, on the surface of which were numerous microgametes. Put into water, the oöcysts give four sporocysts, each containing two sporozoites. In the cyst stage they are resistant to the elements of destruction, and may conserve their integrity for a lengthy period in fæcal material or moist surroundings.

This coccidium is non-pathogenic to the rabbit or bird, as that of the bird is to the rabbit, or *vice versa*.

Age and Occurrence.—It is mostly seen in animals between six months and two years, but more often one to two years, and somewhat more rarely in older cattle or very young calves. It seems to prevail mostly during the warm season: in Europe from July to September, and to disappear about October, although occasionally it may extend into November. It has, however, been seen in the months of January and February in those animals kept at pasture until the previous November before being brought up into sheds and fed on dry, nutritious food. Gair has seen the malady reappear at the commencement of April on the same farm where the disease prevailed the previous summer and autumn. It is as frequent during warm, moist, and rainy years, and amongst animals pastured on swampy grounds containing numerous springs and streams, as when cattle are grazed on elevated and dry tracts. It seems, however, to be more prevalent on particular pastures, where the disease recurs from year to year, but may occur during the housing period of the year.

Period of Incubation.—This is approximately fixed at three weeks, although Degoix asserts the disease appears from one to two months from the time the animals are turned into infested pastures.

Mortality.—This varies in different localities. In Switzerland, where, according to Hess, it attacks 5 per cent. of cattle in certain districts, from 2 to 4 per cent. of these succumb. In France Degoix approximates the mortality from 12 to 15 per cent., according to the year. In some places it is as much as 20 to 25 per cent. In Gair's small outbreak it was 66·4 per cent. In Montgomery's observations 10 to 20 per cent. succumbed. The mortality is greatest in young calves, older cattle being more resistant, and adults much more so. The better the condition of health, the more resistant to the disease. Hüber found the disease more fatal when the cattle were grazed on the hills than in the valleys.

Transmission.—There is no doubt the disease is taken in by water or food ; in fact, it is a water or food borne disease. It cannot be conveyed by inoculation of the fæcal material, discharges, or blood. The disease may be widely scattered over certain districts, and often only one animal in a large herd is affected. It may, however, appear as a veritable shed plague, attacking animals indiscriminately. Pastures contaminated the previous year, or fodder or water soiled with fæcal material containing coccidia, are without a doubt the means of the propagation of the malady. There is no evidence that sporogony plays an active part in its cause, but it is generally assumed that the infestation is maintained by schizogenesis. Rivolta observed when rabbits affected with coccidiosis were kept in premises where the hay became contaminated with their fæcal discharges, that this hay, when given to cattle, did not cause the malady in the latter.

Symptoms.—The disease is rarely observed during its initial stage, but the first symptom usually noticed by the owner is the frequent passage of a thin serous or watery, dark greenish, stinking fæces, without much effort on the part of the animal. On the second or third day the fæces become mucous, reddish-black, and contain several blood-clots of various sizes. At first there does not appear to be much general disturbance of health, loss of appetite, or an elevation of temperature. As the disease advances from the fourth to eight day, there is frequent straining, with passage of small quantities of fæces, composed almost entirely of grey-coloured shreds of mucus, to which are added clots of pure blood, varying in size from a pea to an egg, or even as large as one's fist. The clots are either dark and mixed on the surface with ingesta, or bright red. After each act of defæcation there is an effort of straining, and even eversion of the anal mucous membrane or prolapse of the rectum, the mucous membrane of which appears much reddened, very painful, and even excoriated. These characters seem to be much more marked as the disease advances in its course. The animals arch their backs for a minute or two, or even longer, and the fæcal material, which is generally passed in great quantities, soils the quarters, lower part of the hind-limbs, and tail; they become dull, show a certain amount of colic by stamping their feet, and often grind their teeth. The appetite is lost, rumination is suspended, and the secretion of milk is diminished or checked. Emaciation becomes rapidly profound; the coat is open and dull; the facial expression is pinched; there is a difficulty in maintaining the standing position; the temperature ascends up to 105° to 106° F.; and the pulse rises to 60, 70, and even 140, when, in fatal cases, it becomes very small, thready, and scarcely perceptible. The muzzle becomes dry, roughened or reddened; the coat, ears, and extremities feel cold; the flanks fall in; the tail projects from the body; the eyeballs sink into their orbits; and sometimes there are attacks of sweating. These symptoms may prevail for nine, ten, twelve, fourteen, or more days, when the animal either succumbs or recovers.

In many cases the appetite, even though diminished, persists; some animals appear stiff, and often move their hind-quarters. Rectal straining, even when little or no fæces are passed, and eversion of the rectum, may be encountered even in mild cases. Peristalsis is often arrested, and in consequence little or no fæces are passed. The mucous membranes become pallid and occasionally icteric. Sometimes an offensive odour persists or arises during convalescence.

Montgomery, in an acute outbreak of coccidiosis in cattle in East Africa, observed lachrymation, muco-purulent nasal discharge, and often stomatitis, ulceration of the labial and lingual mucosa, gastritis, frequently ulceration of the abomasum, enteritis, as well as the other characteristic symptoms—dysentery, diarrhœa, tenesmus, and proctitis. A scaly eruption of the neck and shoulders frequently occurred towards the end of the disease. In Pröger's outbreak in calves in 1877 similar symptoms to those in Montgomery's enzoötic were observed. In some of Gair's cases there was swelling of the limbs from knee and hock downwards, the swelling terminating abruptly above the knee and hock. Mammitis was also observed in three cases. Hess has often seen slight bloody diarrhœa, followed in about three to six days by the expulsion of casts of croupous membrane, which are always fœtid, and may reach a length of several metres.

Course and Termination.—In mild cases in which the appetite is not suppressed and the animals do not seem much distressed recovery is often rapid—within five to seven days at the most. If, on the contrary, the appetite has become diminished, and the acute symptoms continue for more than ten days, the course is usually slow. The diarrhœa, although losing its bloody character by the sixth or eighth day, may continue for some time. The rectal tenesmus becomes gradually rarer, and ceases between the tenth and fifteenth days. The appetite may remain fastidious for a lengthy period. There may be alternate improvement and relapse. Lice, and even ticks, appear on those debilitated by disease.

In those animals that have become very anæmic and profoundly emaciated, have sunken orbits, occasionally grind the teeth, continue to assume the recumbent position with the head extended, and appear indifferent to their surroundings, death usually sets in towards the tenth to fifteenth day, preceded by a subnormal temperature and a thready or imperceptible pulse.

Sometimes in well-bred animals in a good condition the disease suddenly assumes a peracute aspect even when up to then it did not show a rapid course. There may be on the first day a serous diarrhœa, next day the diarrhœa is bloody and contains clots, and then occur violent fits of convulsions, which are sometimes preceded by paresis of the hind-quarters, ataxic symptoms, or want of co-ordination, followed by death in nine cases out of ten. In some of these cases the animal assumes the lateral recumbent position, with rigid extension of the head on the neck, and outstanding rigid limbs, and in thirty-six hours death takes place in an attack of convulsions.

In Gair's cases, two cows in calf during the disease calved, but both calves died shortly after birth. In two cases the disease ran its course, with recovery in a month; in another, in a fortnight. In two cases death followed, one after twelve days and in the other after a fortnight's illness. Montgomery found that in young calves it frequently ran its course in three to five days, and chronic cases lasted months, the most common symptom being anæmia.

The disease may run its course and end in death in twenty-four hours. It may apparently disappear for a time and then recur. Convalescence may continue for several months, or death may result after a few months' suffering from profound anæmia.

Lesions.—There is generally emaciation of the carcass, pallidity of the muscles, and soiling of the skin with fæcal matter. If the disease has run a short, rapid course there is not much perceptible wasting. In advanced cases the eyes are sunken. In acute cases in young calves there may be corneal opacity, excoriation of the skin of the face, due to lachrymal discharges, and abrasion of the muzzle. The oral mucous membrane, especially in the vicinity of the teeth, may be congested; the buccal membrane in contact with the molars may show gangrenous ulceration; the lateral surface of the tongue shows evidence of catarrhal stomatitis; the hard palate sometimes offers a few ulcerations; the laryngeal, nasal, and tracheal mucous membranes may have an intense red discoloration. As a rule, however, the intensity of lesions is confined to the large intestine, which usually contains very little fæcal material; its mucous membrane is reddish-brown in colour, œdematous, uneven, lumpy, granular, or lies in well-marked longitudinal folds, and is covered with mucus, which may vary in density according to the part. In some places the mucus forms more or less thickened patches of a greyish or yellowish colour, and of a resistant character, as though mixed with coagulated fibrin; they are fairly well defined, irregular in outline, and vary in width from some millimetres up to several centimetres. The patches are more or less adherent to the mucous membrane, from which they may be detached quite easily, even by a stream of water. The mucous membrane, from which the mucous patches have been removed, is slightly depressed, palish in colour, and, contrasting with the neighbouring inflamed parts, showing a red coloration. The inflamed mucous membrane appears swollen, loosened, and more vascular than normal. Numerous points or streaks of hæmorrhage and vascularisations are found along the folds of mucous membrane throughout the whole extent of the larger bowel, from the cæcum to anus. Branchli, Schlatter, and a few others, have found erosions of the mucous membrane of the abomasum. Pröger, Ducloux, and Montgomery, have also observed congestion of the pyloric portion, and petechiæ and small ecchymoses in the other portion.

Microscopical examination of the mucous patches shows epithelial cells belonging to the mucous membrane and vesicular cells to the glands of Lieberkühn, numerous blood and lymphoid corpuscles, and, scattered all through this mass of cells, coccidial oöcysts and merozoites. In places where the mucous membrane is infiltrated with round cells the superficial epithelium is wanting. The lesions gradually lessen in intensity from the rectum towards the anterior terminal portion of the colon. The glands of Lieberkühn are the chief seat of the coccidia, and become unequal in width, often very narrow, and are frequently provided with claviform dilatations at their lower extremity, and thus the regular structure of the glandular region is totally lost. The lumina of the glands are entirely filled with coccidia and a few leucocytes; some of the slender columnar cells appear lost, so that the rows of cells are interrupted by the invasion of the cells by coccidia. In some cases all the epithelial cells seem to be lost. As in the other animal coccidioses, normal glands may be found next to diseased ones. In those parts where the lesions are pronounced, the interglandular areolar tissue is infiltrated and enlarged. Degoix failed to find coccidia in the epithelial cells of the intestinal mucous membrane. The coccidia are found in every stage of development in the fæcal matter. According to Hess, they are also found in blood-clots, but Zschokke and others have failed to find them in clots composed purely of fresh blood.

In Pröger's cases there were yellowishness of the muscles, fairly thick and dark blood, and an accumulation of a certain amount of yellowish serosity in the pericardial sac. The nasal, laryngeal, and tracheal mucous membranes were intensely reddened. The pyloric extremity of the abomasum was highly injected. The mucous membrane of the small intestine was red or yellowish, and showed here and there a few spots of erosion and loss of substance. The whole length of the bowel was lined with a thick layer of mucus. The mucous membrane of the large intestine was softened in places, swollen and spotted with numerous nodules the size of a pin's head. The mesenteric and other lymphatic glands were swollen and infiltrated with serosity. Coccidia were not only found in abundance in the glands of the small intestine and solitary follicles forming the yellowish nodules, but also in the mesenteric glands. Montgomery's observations concord almost entirely with these findings of Pröger. Marotel observed similar yellowish nodules in the intestine.

Diagnosis.—This disease, in its acute form, may be mistaken for rinderpest, subacute anthrax, certain forms of piroplasmosis or trypanosomiasis, black-quarter, or even pasteurellosis. All these diseases, however, are transmitted by inoculation, which is not the case with coccidiosis. It may also be confounded with Jöhne's (more correctly Bang's) disease, and intestinal tuberculosis, but these two diseases are also inoculable and react to their specific vaccines. It may furthermore be mistaken for intestinal strongylosis. All the above-mentioned diseases may be eliminated by the detection of coccidia in the fæcal discharges. It should not be forgotten that it is quite possible for other infestations or infections to be associated with the presence of coccidia in the intestine. As to the question of hæmorrhagic cystitis (hæmaturia), it will be dealt with in Vol. II.

Treatment.—In semi-tropical and tropical countries it may be advisable to slaughter off all the infested animals in order to prevent the spread of this malady. At any rate, where the disease prevails, careful avoidance of pastures, at least for one year or alternate years, should be adopted. Infested cow sheds should be evacuated, scraped, scrubbed, cleansed, well disinfected, lime-washed, and allowed to dry before cattle are replaced in them. Cattle passing oöcysts with their fæces should be isolated. In presence of the disease, dry, easily digestible, and highly nutritious food and pure water should be given to fortify the system against the inroads of the parasites.

During the active stages of the malady, gruel, milk, eggs, linseed tea, or such-like demulcent agents, should be offered, or, if there should be no appetite, forced down the animal. The animals should be provided with rugs in inclement weather, kept scrupulously clean, and their fæces and urine carted on to arable land, from which cattle should be prohibited for some months at least.

Weak solutions of catechu, hæmatoxylin, or bael fruit, may be given in the animal's drinking-water. Warm enemas of permanganate of potassium (1 in 1,000) or solution of peroxide of hydrogen should be administered from time to time. If there should be much tenesmus a hypodermic injection of morphine, a suppository of cocaine or morphine, or a drench containing opium, may lessen the straining efforts. A small enema of chloral hydrate in linseed tea has a soothing effect on the mucous membrane, as well as a hypnotic influence on the animal. On no account should heroic or irritating remedies be used, but a large dose of a sedative is much more efficacious than smaller doses frequently repeated.

Some authorities give the old chalk mixture, others tannigen or tannalbin. Storch had good results with tannoform. If there should be great prostration, as in the peracute forms, normal saline solution should be given under the skin by means of a funnel or douche arrangement, connected to a large hypodermic needle by a rubber tubing. This is quite harmless, can be rapidly administered, and is generally beneficial by replacing the lost fluid from the circulation.

The medicinal treatment should be *gently* and *patiently* maintained until the active symptoms have disappeared and the fæces no longer contain any oöcysts, which may be found in the fæces some time after the activity of the disease has abated.

The resistance of coccidia in their oöcyst stage to disinfectants and to the elements of weather is very great. They may retain their virulence in fæces for months.

Public Health.—Although man suffers from a form of coccidiosis, there is no reliable evidence to connect the human disease with that of animals; therefore there are no grounds on which to be alarmed over man contracting the disease from animals. Cattle killed in the earliest stage of coccidiosis, before their system has become infected with bacterial products, and before their flesh has undergone any perceptible change, might be used for human consumption without danger. In any case the flesh of such animals has never produced any injurious effect on those who have partaken of it.

IV. COCCIDIOSIS IN SHEEP.

Synonym.—Intestinal psorospermosis.

History.—As far back as 1874 S. Rivolta, of Pisa, encountered coccidia in the intestines of sheep. In 1890 Theobald Smith and Cooper Curtice recorded that the disease had been observed by them in the United States of America. In 1891 Nocard described a case of intestinal coccidiosis in a lamb. In 1893 Maske, in Germany, reported his observations on the same subject. In 1896 McFadyean described an outbreak that he encountered in England, and gave a good résumé of the histological characters of the lesions. In 1901 Moussu and Marotel recorded their researches into the disease. In 1905 Mazzanti in Italy observed coccidial enteritis, and described the parasite.

Causal Organism.—*Eimeria* (*Coccidium*) *Faurei* is the name given to the coccidium of the sheep by Moussu and Marotel, who consider it is a distinct species. The parasites are found in various forms and sizes in the intestinal contents and mucous membrane. They are either ovoid or spherical, and measure 30 to 40 μ in length by 18 to 26 μ in width. In the nodular lesions of the bowel the coccidia are from 250 to 300 μ in their ripe schizont stage or microgametocyte stage. The ripe schizonts contain numerous spindle-shaped merozoites, and the microgametocytes nuclei, grouped into the form of a crown or areola, which are the microgametes.

The parasites prefer the small intestine, but they are also seen in the abomasum of 70 per cent. of the sheep examined (Maske).

Age and Occurrence.—It chiefly occurs as an enzoötic in young lambs two or three months old, just before or soon after leaving the ewes. The ewes seem resistant. It is seen mostly in April and May.

Mortality.—This is usually very high, varying from 10 to 30 or more per cent.

Transmission.—There is no doubt it is due to soiling of the food with the fæces of some animal giving off coccidia.

Symptoms.—These resemble those of a progressive pernicious anæmia, in which hitherto vigorous lambs without any apparent cause fall ill, become dull, careless, or indifferent to their surroundings, limp, and very tame. The appetite generally becomes lost, there are slight colicky pains, tympanitic distension of the flanks, slight diarrhœa, and shortly before death a spumous discharge from the nostrils. In those animals in which the appetite may remain very good there is great emaciation and profound anæmia. There does not appear to be much, if any, elevation of temperature, but often the diarrhœa is serous and abundant, which prevails for some weeks before death takes place from exhaustion. In acute cases lambs often die within forty-eight hours or so after showing dulness, great depression, and loss of appetite.

Lesions.—Unlike those of the ox, the lesions in the sheep are generally confined to the small intestine. There is mostly emaciation of the carcass and a profound anæmia of all the tissues. The blood is pale and watery. The small intestine, especially the duodenal part, shows an increased vascularity, and the mucous membrane, when held up to the light, is seen to be riddled with innumerable minute opaque points scarcely visible to the naked eye. Microscopical examination of the intestinal yellowish and serous contents and scrapings of the mucous membrane show numerous coccidia in an oöcyst or macrogamete stage. Larger spots, varying from the point of a needle to the head of a pin, are found distributed at various depths in the mucous membrane, and correspond to the ripe schizont stage or that of the microgametocyte. Besides these minute lesions, there may be on the diffused inflammatory parts, which often show pronounced injection of the small vessels of the villi, small, pedunculated or sessile, pear-shaped or cauliflower-like tumours or excrescences, of the size of two or three oats, or a pea to a filbert-nut, scattered here and there, the small tumours containing innumerable coccidia, varying from 10 to 12 μ in length by 7 to 9 μ in width (Nocard), or 20 by 14 μ (McFadyean). Maske found small nodules of the size of a pin's head implanted in the depth of the mucous membrane of the abomasum. Cooper Curtice and Stiles found whitish spots scattered along the intestine, the villi of which were considerably enlarged, and had their epithelial cells enlarged, each containing one or several parasites, from 18 to 21 μ in length by 15 to 16 μ in width. Mazzanti observed enteritis and, in the intestinal canal, coccidia of 30 to 50 μ in length by 14 to 28 μ in width. The tumours observed by Nocard, McFadyean, and others, result from hypertrophy of the villi and the glands of Lieberkühn, which increase to more than double their normal size. The villi are denuded of epithelium.

Diagnosis.—This disease, without a careful post-mortem examination, is liable to be confounded with the various strongyloses, which may, however, coexist, and the many blood diseases known to affect sheep. Careful examination with the microscope of the fæces, and also of the duodenal contents, and scrapings from the mucous membrane of the small intestine or the lesions situated therein, will enable one to pronounce as to its real nature.

Prognosis.—It is generally very serious, although the disease up to the present has been rarely recognised.

Treatment.—Change the ground from day to day, a practice that should be adopted in all obscure sheep enzoötics. Take care not to feed young sheep in the pastures on which the manure from sheepfolds has just been scattered. Avoid feeding lambs on notoriously bad pastures.

To an infested flock give dry nutritious food, over which should be sprinkled a weak infusion of catechu, logwood, bael fruit, or some such mild astringent. Sulphate of iron, common salt, gentian, and fenugreek made into a powder and sprinkled on the food, act as a tonic.

V. COCCIDIOSIS OF THE GOAT.

Synonyms.—*French :* Coccidiose de la chèvre.
German : Darmcoccidiose der Hausziege.

History.—Coccidiosis as a disease of the goat was first described by G. Marotel in 1905. In 1907 F. P. Martin, of Dresden, gave a description of the histological character of intestinal coccidiosis in this species. In 1908 and 1909 Stevenson, when working in the Wellcome Tropical Research Laboratories at Khartoum, discovered coccidiosis in several goats, and in 1911 published the results of his investigations in the Fourth Report of that institution (Vol. A., Medical). In 1912 A. Martin, of Toulouse, described the histological characters of intestinal coccidiosis in a Tunisian kid.

Causal Organism.—Marotel, who has made a complete study of the coccidial parasite of the goat, has concluded that it is a new species, and has given it the name of *Eimeria* (*Coccidium*) *Arloingi.* His description of it is as follows : Dizoic, tetrasporocystic coccidium, having ellipsoidal oöcysts, measuring 22 to 27 μ by 16 to 18 μ, provided with a polar capsule. Microgametocytes of 30 to 40 μ, giving an infinite number of vermiform microgametes, 5 μ long, with flagellum of 3 μ. Schizonts of 18 to 23 μ, forming thirty to fifty globular merozoites (2 to 2·5 μ) having a large crescentic nucleus. Sporogony comparable to that of *Eimeria* (*Coccidium*) *cuniculi.*

Martin of Toulouse gives somewhat different figures to these. He finds that the oöcysts measure 32 to 36 μ by 20 to 24 μ, the microgametocytes 22 to 30 μ, and the schizonts (about forty nuclei) 16 to 20 μ. But dimensions, although frequently at variance, do not constitute an absolute differential characteristic. The refractile micropylar capsule which Martin has found on numerous oöcysts confirms the relationship.

Minchin suggests that schizogony takes place before any symptoms are seen.

Habitat.—Epithelial cells of the glands of Lieberkühn.

Symptoms.—These resemble those seen in the lamb. The animal appears suddenly ill, refuses food, wastes away rapidly, assumes the recumbent position, from which he is roused with difficulty, gives plaintive cries, and dies during an attack of convulsions after two or three days' illness. There may also be a difficulty in defæcation in older animals, which seem more resistant than kids two or three months old.

Lesions.—These are similar to those found in the lamb. The small intestine may show on its mucous membrane many slightly salient spots. Some of these spots are irregularly circular, yellowish-white, opaque, varying in size from a pin's point up to 2 to 3 millimetres in diameter, and formed by the contact of three to twelve whitish points placed side by side. Other spots are similar in form and dimension, but have a greyish appearance, and, being less visible than the first. These spots are caused by the coccidia lodged in the epithelial cells of the glands of Lieberkühn. In some cases the glands of Lieberkühn are considerably enlarged and dilated, giving rise to papillary or nodular projections on the mucous membrane. Granulations appear in these glands in the depth of the submucous tissue, which give the glands the appearance of a glandular adenoma, resembling very much that seen in the liver of the rabbit. There is also a more or less loss of epithelium, and even of the entire mucous membrane, which may be replaced by an increase of fibrous tissue and cell degeneration.

VI. COCCIDIOSIS OF THE HORSE.

Very little is known of coccidiosis of the horse beyond the fact that Alois Pachinger, in 1886, found in three cases coccidia in the kidneys. In every case the kidneys had undergone such changes that death was evidently due to the lesions. Pachinger did not hesitate in considering the parasites as identical to the *Eimeria falciformis*, which usually live in the intestinal epithelial cells of the mouse. Further researches into obscure equine maladies may yet throw more light on the frequency of coccidia in the horse.

VII. COCCIDIOSIS OF THE PIG.

It is doubtful if the coccidia found by Jöhne in the pig were, as he asserts, the *Eimeria* (*Coccidium*) *cuniculi*. He found at the superior border of the liver, circuitous cysts containing a turbid fluid, holding in suspension a great number of ovoid bodies, measuring 120 μ in length by 70 μ in breadth. In consequence of its dimensions, Railliet believes it is a new species, and therefore has given it the name of *Eimeria* (*Coccidium*) *gigantea.* Zürn found coccidia in the intestine of a sucking-pig, which had succumbed to enteritis. This animal belonged to a lot on the same farm which gradually succumbed. The contents of the intestine contained oöcysts, but as the organ was undergoing putrefactive changes it was impossible to determine if the enteritis was set up by these parasites. Zschokke, Olt, and others have recorded a disease in which there are very numerous vesicles appearing on the hairy parts of the skin, found chiefly after slaughter on removal of the hairs, and giving to the finger the feeling of shot in the skin. These vesicles do not surpass the dimensions of a grain of pepper, and the various authors believe they encountered in them a new species of coccidium, *Eimeria* (*Coccidium*) *fuscum.* But Lühe considers this alleged parasite is not a coccidium.

VIII. **COCCIDIOSIS OF THE DOG.**

The coccidium of the dog, *Diplospora begemina* Stiles (*Coccidium begeminum* Stiles 1891 ; *Cytospermium villorum intestinalis canis* Rivolta 1878 ; *Coccidium begeminum* var. *canis* Railliet), is not found in the epithelial cells, but in the interior of the intestinal villi below the epithelial cells. It receives its qualification "bigeminum" from the fact that usually two of the oöcysts are attached to one another, suggesting that they undergo a longitudinal division. The oöcysts are ellipsoidal, slightly asymmetrical, with a double contoured membrane, and measuring 12 to 15 μ in length by 7 to 10 μ in breadth. According to Stiles, the young encysted coccidium has its contents divided into two masses, which are mostly surrounded by a shell, and remain attached to another by the lateral surface. It is believed that these two coccidia have their protoplasm divided into four sporoblasts, which would justify them being placed in the genus *Eimeria.* Von Wasielewski, however, has shown that in each oöcyst there are only two ovoid sporocysts, each giving rise to four sporozoites. In consequence a disporous tetrazoic coccidium belongs to the genus *Diplospora.* As far as is known, this is the first disporous tetrazoic coccidium found in mammals. This parasite is not only found in the depth of the intestinal villi, but also in the intestine.

It does not seem to influence the health of dogs, as one sees numerous healthy dogs or those having succumbed to various diseases bearing these parasites. J. Basset, of Alfort, however, believes coccidia give rise to a disease resembling distemper in young dogs. This question is, however, well worth inquiring into.

Coccidia have been found in the intestinal epithelium of young dogs. Pachinger has encountered a coccidium in the kidneys of a dog. Liénaux in 1891 found a coccidium in the lung of a dog.

IX. COCCIDIOSIS OF THE CAT.

A similar coccidium to that in the dog is found in the cat, and as it is only a variety, it is denominated *Diplospora bigemina* var. *cati* Stiles (*Coccidium bigeminum* var. *cati* Railliet). It is found in the intestinal villi, but not in the epithelial cells. It measures 8 to 10 μ in length by 7 to 9 μ in breadth. Finck, who was the first to observe it, attributed to it the function of assisting in the phenomena of absorption of fatty material. But von Wasielewski stated that it is pathogenic, because he has seen the majority of young cats experimented upon with this parasite succumb with diarrhœa and emaciation. Poénaru and Georgesco, Bucharest, in 1912, observed a case of chylous ascites in a cat in the interior of the gall-bladder and the lymphatic glands of which, on post-mortem examination, they found coccidia, and in consequence these authors suggest that the coccidia played a mechanical part in producing the chylous ascites. Pachinger found coccidia in the œsophagus, stomach, and intestine of a cat. Grassi relates the history of another coccidium, under the name of *Coccidium Rivoltæ*, found in the epithelium cells of small and large intestines, as well as in the intestinal contents. It develops in the epithelial cells, and measures, in a state of maturity, 27 to 30 μ in length by 22 to 24 μ in width. Freed from the cell, it falls in the intestine, and is then expelled. After being in water, the protoplasmic mass divides into two, and then into four, sporoblasts, giving each four falciform corpuscles with the protoplasmic relicts or remains. Chierici records a case of coccidiosis of the liver in the cat. He found numerous coccidia in the bile-ducts, which were oval, had a double contour, and measured 26 to 30 μ in length by 17 to 20 μ in breadth. The liver was more consistent and slightly enlarged than in the normal state. The bile-ducts were dilated, their walls thickened, and contained a dense yellow-greenish exudate, formed of degenerated epithelial cells, débris, and numerous coccidia. The small bile-ducts were obstructed by desquamated epithelial cells and coccidia, whilst the connective tissue of their wall was thickened and infiltrated with leucocytes. Oöcysts were found in the intestinal mucus, and develped four sporocysts and two sporozoites. The polecat also harbours a variety of *Diplospora bigemina*—*Diplospora bigemina* var. *putorii*—which measures 8 to 12 μ in length by 6 to 8 μ in breadth.

X. COCCIDIOSIS OF THE MOUSE.

The coccidium of the mouse—*Eimeria falciformis* A. Schneider—was first observed by Thomas Eimer in 1870. The intestinal cells contain the parasite. It is also found in the intestinal canal. Eimer for the first time observed schizogony in this species, which has often been termed the "Eimerian phase" in the life-cycle of coccidia. In the mouse sporogony takes as long as four days. The average oöcyst measures 26 μ in length by 16 μ in breadth.

No doubt mice get infested through the alimentary canal when they take in food soiled by their infested fæces. They rapidly succumb to the disease.

The mole suffers from a pernicious anæmia and enteritis accompanied by violent diarrhœa due to a coccidium—*Cyclospora caryolytica*—which is generally fatal. Schaudinn believes the mole becomes infested by eating wood-lice and other dung-feeding arthropods which have fed on fæces of other infested moles.

XI. COCCIDIOSIS OF MAN.

Coccidia have been found in man by Kunstler and Pitres, Virchow, Gubler, Dressler, Perls and Slattler, von Sömmering, Silcocks, Podvissotski, Leuckart, Kjellberg, Grassi, Rivolta, Railliet and Lucet, Kartulis, Labbé, Metzner, Eimer, Schneider, etc.

Railliet and Lucet obtained coccidia resembling *Eimeria bigemina* from a woman and her infant, both of whom had been suffering for some time from a chronic diarrhœa.

Similar clinical manifestations are observed in man as in animals. The species of coccidia found in animals have been attributed with the causation of disease in man. There is, however, no evidence that the animal coccidioses are transmissible to man, or from one class of animal to another class.

Coccidia have been found in the liver, intestine, kidney, sigmoid valves of the aorta, and mitral valves, in pleuritic effusions, etc. For a good account of the pathology of coccidiosis in man, etc., *vide* R.

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ASPERGILLOSIS

By HENRY GRAY, M.R.C.V.S.

Synonyms.—Mycosis; Aspergillary pseudo-tuberculosis; Abdominal mycosis; Bronchomycosis; Pneumomycosis; Cytomycosis; Rhinomycosis; Gutturomycosis.

French: Aspergillose; Mycose aspergillaire; Mycose bronchique, pulmonaire et abdominale; Mycose viscérale; Aspergillose nasale.

German: Pilzkrankheit; Aspergillose; Pneumomykosis Aspergillana.

Definition.—A fungal disease of various domestic and other mammals, including man, and also of birds, due to the presence of *Aspergillus fumigatus* Fresenius, and characterised by membranous exudates or nodules in the respiratory channels or parenchymæ, and sometimes in other organs, and occasionally around the articular surfaces. It may also attack eggs during the process of incubation.

Causal Organism.—The fungus *A. fumigatus* Fresenius is included in the *Class* Mucedinæ, *Order* Ascomyces, *Family* Perisporiaceæ, and *Genus* Aspergillus Micheli. Constantin yet places it in the first group of his Simple Mucedinæ, which includes all those filamentous fungi developing on the surface of living or dead material, and producing external spores.

The order *Ascomyces* includes those fungi consisting of a thallus or mycelium divided by intersections, and reproducing themselves by means of spores formed by partial division within the interior of asci or mother-cells, which are contained in almost every case in special organs or perithecæ.

The family *Perisporiaceæ* is characterised by having absolutely closed perithecæ with dense walls, the bursting or absorption of which is requisite in order that the mother-cells or asci can be set free, and in association with them the ascospores. This family is almost entirely reproduced by free globular cells or conidia, clustered on the surface of sterigmata—special modifications of the thallus.

The *Genus* Aspergillus includes those fungi consisting of filaments or hyphæ of colourless mycelium, with thin transparent walls irregularly intersected by partitions, and from the filaments lateral branches of similar nature are given off. The filaments form on the surface of the substance, on which develops a branched felted mass, the superficial portions of which resemble a tuft of down. From this mycelium the fertile filaments arise. They contain few partitions, and exhibit a swollen club-like head. This swollen extremity becomes covered with ciliæ termed "sterigmata," each of which ends in a chaplet of conidia, the whole forming a cluster resembling a bottle-brush, or the brush used for sprinkling of holy water in the Roman Catholic services; hence the name of *Aspergillus* for the fungus.

The *A. fumigatus* in its adult state forms on the substance on which it lives greyish, bluish, or greenish clusters, according to the nature of the substance. It is made up of a more or less furnished mycelium, composed of creeping branched filaments, having thin transparent walls, unequally intersected and colourless. These filaments form on the surface of the substratum a compact felted layer, which is uniform or plaited, white, yellowish or greyish, which constitutes the thallus, above which it extends constituting another but looser one in the form of down, forming the aërial mycelium. From this arise fertile branches, which are erect, non-intersected, colourless, or slightly greenish or brownish, swollen on the summits, in the form of a ball, covered entirely with elongated cells, and pointed towards the exterior, forming the sterigmata or basidia, each of which gives off a cluster of spores or conidia, which in reality resemble the inflorescence of an onion. These last are round or, rarely, oval, smooth, uniform, pale, colourless, or slightly greenish or brownish, and measuring from 2·5 to 3·5 μ in diameter.

The filaments or hyphæ are 2 to 3 μ in diameter, and at those places where they show bulgings from 8 to 30 μ in diameter. The upright conidia are from 100 to 300 μ in length, by 5 to 6 μ in width at the base, and increase in size towards the summit, which consists of a slightly elongated bulbous head, 10 to 40 μ in size, covered in the upper two-thirds with powdery sterigmata, 6 to 15 μ in length, sometimes shorter towards the extreme end. The round or oval conidia are 2 to 3 μ in size, generally brownish, and easily removed from the conidiophore, on which, however, the sterigmata remains fixed. Perithecæ have not often been observed. The *A. fumigatus* grows by preference in Raulin's fluid or beer yeast. It is exclusively aërobic, and develops between 22° to 40° C. It is well stained with aniline colours, and takes Gram. It forms a toxin, which is attenuated by prolonged heating at 90° C., and only destroyed at 120° C. The rabbit and dog are very sensitive to its effects; then come the guinea-pig, cat, and white rat. It causes convulsions, tetanic spasms, and paralysis. The dog and cat are refractory to inoculation of the spores. The pigeon is very sensitive to the spores, but, on the contrary, very resistant to the toxin.

Besides the *A. fumigatus* there are several other species, of which it is well to mention a few :

A. glaucus does not seem to be pathogenic. It develops in low temperatures, and has very large spores—8 to 15 μ in diameter.

A. repens resembles the former, but has small spores—4 to 8 μ in diameter. It does not seem to be pathogenic, and is found in wax in the ear.

A. malignus has been found in the ear of man, is pathogenic for the rabbit, and may grow at 35° to 37° C.

The *Genus* Sterygmatocystis is characterised by spore-bearing hyphæ, terminated by a globular swelling covered with primary sterigmata, which give off by division several secondary sterigmata or basidia, forming chaplets of conidia.

Sterygmatocystis (*Aspergillus*) *nidulans* has been found in otitis, and is pathogenic to animals. It grows between 15° and 38° C., and forms on culture media a grass-green coloration. The bulb-like swellings of the spore-bearing hyphæ are triangular, with rounded borders, and the conidia measure about 3 μ in diameter. It is the cause of certain cases of mycetoma.

Sterygmatocystis (*Aspergillus*) *nigra* is frequently found as blackish spots on decomposing organic material. It has been observed in several cases of otitis, and other affections of man and animals, but it is not pathogenic. It does not grow at a temperature above 25° C. The conidial bulgings carry primary and secondary sterigmata and very dark brown conidia measuring about 4 μ in diameter.

Etiology.—The ingestion or inhalation of the spores of the *A. fumigatus* determines the disease under suitable conditions. The fungus is found on grain and straw, such as wheat, maize, rye, oats, millet, and vetches ; it is also found on lucerne, trefoil, sainfoin, and other forage. For the spores to develop, certain conditions of heat, aëration, and humidity are necessary. Although the fungus abounds everywhere where grain, straw, and forage are found, it does not appear to cause any trouble until some favourable conditions enable it to thrive in the animal's system. Once it has thriven in the animal body, it becomes more virulent, and then it is easy for it to be transmitted from animal to animal, especially of the same species, as from bird to bird.

Any condition by which the system becomes depressed renders the animal an easy prey to its ravages. Animals that are weakened by parasites, previous infections, insufficient food, bad quality of food, dark, ill-ventilated places, or by overcrowding in damp, confined spaces, excessive cramming with meals, etc., are predisposed to the infestation. Horses living on musty oats on which the Aspergillus is luxuriantly growing often develop the malady, and generally succumb to it.

Man develops the disease by cramming pigeons or poultry affected with the disease with the bird's mouth in his own. It is also seen in hair-combers who use rye-meal to remove the grease from the combings, rye-meal being a good nidus for the fungus. It may be introduced into the system by particles of straw, chaff, grain, or other material passing into the nostrils, sinuses, air-sacs, guttural pouches, or bronchial tubes. *Eggs* get infested during the process of incubation by being placed on damp straw on which the mould is growing. The more highly-bred poultry and pigeons are subject to it in consequence of being more closely confined as a result of their great value. The disease is rare, if not absent, in animals, such as the sheep, continually living in the open.

Species Affected.—Birds as well as mammals confined to a limited space are subject to this malady. It is common in the fowl, pigeon, goose, duck, turkey, pheasant, partridge, and quail; rare in the peacock, guinea-fowl, and swan. It is very common in the green cardinal (*Gubernatrix cristata*) bred in confinement in this country. It is encountered in every species of wild bird kept in confinement in a cage or aviary, or restrained from flight by pinioning. Water-birds are just as susceptible to it as the Gallinaceæ; insectivorous and frugivorous birds being less so in consequence of the nature of their food.

In the mammal it has been observed in the ox, sheep, horse, dog, and man.

Experimentally, it has been conveyed to the rabbit, guinea-pig, monkey, and the susceptible animals. Intravenous injection of cultures or spores produces a septicæmia, or, if the animal live long enough, a generalised pseudo-tuberculosis.

I. ASPERGILLOSIS IN BIRDS.

This disease was first observed by A. C. Mayer in 1815 in the bronchi and air-sacs of a jay. A year later Jäger found a green mould in the air-sacs of a swan. In 1826 Heusinger found this fungus extending to the cavities of the long bones of a stork. Then came the observations of Theile in 1827, Owen in 1833, and Rousseau and Serrurier in 1841, the two first in the crow and flamingo, and the other two in the parrakeet and hind. In 1841 E. Deslonchamps gave an excellent description of the disease in the eider duck, which died of languor after passing six months in the farmyard. In 1842 J. Müller and Retzius found the green mould in the air-passages of a *Strix nictea* and a hawk. The same year Rayer and Montagne found the mould in the air-sacs of a tubercular bullfinch, and Reinhardt in the swan. In 1848 Spring saw it in the air-sacs of a golden plover. Robin, in 1853, minutely described the disease in a tubercular pheasant. In 1873 Hayem made known two cases of pneumomycosis in the duck, and Carville related that Bouchard saw, in 1866, the mould in the lungs of a parrot. In 1875 Heusinger found the fungus in the lungs of a flamingo, and Fresenius in a bustard, the latter giving the fungus the name of *A. fumigatus*. In 1876 Bonizzi encountered the mould in a pigeon. Bollinger, in 1878, saw a cardinal dead of an obstruction of the trachea due to the fungus. In 1879 Generali described a serious enzoötic of the disease in a pigeon-loft. In 1879 Siedamgrotzky observed similar occurrences. In 1880 Bollinger encountered the disease in various birds, notably in pigeons. Rivolta and Delprato described the buccal form (canker) in the fowl in their " Ornitojatria " (" Poultry Doctor ") which appeared in 1881. Kitt, in 1881, recorded two cases of pneumomycosis in the pigeon. In 1882 Schütz and Zürn saw the disease, the former in the lungs and air-sacs of several species of birds. Perroncito in 1884 described a case of miliary aspergillosis in a fowl. In 1887 Bizard and Pommay described two cases in the ostrich, one of them showing generalised pseudo-tuberculosis, and the other arthritis and periarthritis, caused by a fungus which extended into the cavity of the bones. During the same year Rivolta described aspergillary pneumomycosis in the pheasant, and Zschokke a mycosis in the swan. In 1889 Zürn reported fresh observations. In 1890 Dieulafoy, Chantemesse, and Widal, studied the disease in pigeons, and its transmission to man. They found that it usually attacked the buccal membrane in the form of a whitish nodule having a cheesy appearance, and of the size of a pea to that of a small filbert, and might extend and spread to the lungs, liver, œsophagus, intestine, and kidneys. In 1893 Rénon made an extended study of this buccal form in the pigeon, commonly termed " canker " by pigeon fanciers. In 1894 Lucet studied the disease in a hen pheasant and a goose, the latter also showing the fungus in and around the leg-joints, as well as in other parts. This author also studied the fungus attacking incubating eggs. Liénaux the same year described a case in a swan. In 1898 Lignières and Petit published their observations on a deadly enzoötic of aspergillary peritonitis occurring in a flock of forty-three turkeys. In 1912 Roquet described an outbreak of broncho-pulmonary aspergillosis in young pheasants. Leeney and the writer have encountered in geese an arthritis and periarthritis in the tibio-tarsal and the tarso-metatarsal joints, showing abscesses containing the *A. fumigatus*. The writer has also seen it in green cardinals bred in confinement, in the parrot, crane, and numerous other wild species, as well as in domestic poultry.

It is a common disease in Great Britain, but there is very little literature to guide one as to the frequency with which it has been observed in this country. So far as the writer's experience goes, it does not attack several species at the same time, and may only affect one here and there, often only one bird in a collection. Its contagion is only of a low degree, and does not resemble that of the true contagious maladies.

Symptoms.—These vary according to the situation or the extent of the disease in the system, and the age of the bird or the delicateness of the breed. Very often the disease runs a silent course until the bird becomes anæmic, weak, and emaciated, when it dies in a state of exhaustion. In very young birds it may run an exceedingly rapid course, and appear as an acute enzoötic, carrying off the majority in two or three days. Older and aged birds are much more resistant, and may linger for some weeks before succumbing. When the bronchi, cervical and thoracic air-sacs and the lungs become extensively involved with membranous deposits or cheesy lumps of fibrinous material and fungi, or with pseudo-tubercular nodules, the breathing is quickened, often gasping, and not rarely accompanied by a gaping of the mouth during each respiratory effort, similating the clinical appearance of gapes. There may be a frequent cough or sneezing, and a coarse, harsh, or noisy breathing resembling that of avian diphtheria. There may also be a discharge from the eyes and nostrils, accompanied by symptoms of catarrhal tracheitis or bronchitis, when the temperature may rise a few degrees, the appetite be diminished or in abeyance, and the thirst great. The sick birds isolate themselves from their fellows, become weak, have their feathers opened out, wings and tail drooping, eyelids partially closed, head lowered, seem drowsy, are very light, and die in a comatose condition.

When the mouth and œsophagus are involved, as in crammed birds, there are cheesy yellowish-white or grey nodules inside the angles of the mouth, which give off an offensive odour; the birds have a difficulty in swallowing; the pellets often remain in the gullet; there is neither natural appetite nor thirst; the birds become listless, indifferent to their fate or surroundings, and give off a sourish or musty odour; the comb and wattles are pallid or marbled, with bluish and whitish streaks; emaciation is great, and death seems to take place from intoxication.

Diarrhœa often sets in during an advanced stage of the disease, the feathers around the vent becoming soiled and often matted. When the disease is confined to the thoracic or abdominal air-sacs, the only clinical appearance is emaciation, the bird feeling light. In some cases of cervical cytomycosis the breathing suddenly becomes of the pumping kind, the tail moving during each respiratory effort, and the bird, especially if a cage or aviary one, dies in a few days. Sometimes the appetite seems fairly preserved, but yet the bird wastes rapidly or becomes extremely light.

In geese, ducks, and some other birds, there may be lameness, disinclination to move, no inclination to follow the flock ; the joints are hot, swollen, and painful ; loss of appetite, extreme emaciation, and a difficulty in breathing, which is often of the gasping kind are also observed.

Appearance of Affected Eggs.—Recently incubated eggs, still fairly transparent when examined by a good artificial light in a dark place, show in their central part and near the periphery a fairly large opaque spot, immotile, vascularised, fixed to the vitellus, and possessing a vague resemblance to a large spider. This is the germ which has succumbed after commencing to develop. At the large extremity of the air-chamber one sees a black spot, varying in extent, immovable, and completely intercepting the luminous rays and rendering that part of the shell opaque on the internal face on which it is situated. When the incubation is more advanced, the transparency is lost by the greater development of the embryo, but the air-chamber becomes larger. In eggs affected with aspergilli it is easy to see the black spot just mentioned. When such eggs are carefully cracked, the black spot is found to consist of a green or smoky green mould. This spot is situated either exclusively on the external layer of the shell membrane or may extend into the internal layer. The eggs may not give off any rotten odour, but they have a musty or mouldy smell.

Course.—The disease generally runs a course varying from one to four weeks. It may, however, end in death in a few days or be prolonged for two or more months. It is usually fatal.

Diagnosis.—Microscopical examination of the lesions and detection of the mycelia and spores of the *A. fumigatus* should offer no difficulty in distinguishing this disease from other pseudo-tuberculoses, tuberculosis, and parasitic nodular diseases. If there should be any doubt as to the species of fungus, recourse can be had to its culture in Raulin's fluid.

Lesions.—The lesions are more often found in the air-sacs (cytomycosis), lungs (pneumomycosis), or bronchial tubes (bronchomycosis), than in other organs. They are occasionally found in the nasal channels (rhinomycosis), air sinuses of long bones, in the areolar tissue around the joints, trachea, liver, peritoneal cavity, mouth, œsophagus, kidney, intestinal wall, and other parts.

The lesions appear in the form of membranous exudates or tuberculiform nodules. The *nodules* are generally found in the depth of the tissues, and vary from the size of a pin-head to that of a hemp-seed, split-pea, or monkey-nut. They are fairly well circumscribed, generally firm in consistence, and sometimes caseous or calcareous. They are whitish, yellowish, or greenish in colour, and vary in number from a few discrete nodules to many, of which some are confluent. They may give to the lung or other organ the appearance of caseous pneumonia or tuberculosis.

Histologically, the nodules resemble those of tuberculosis, but they show mycelia or spores instead of tubercle bacilli. Intravenous injection of spores cause similar lesions in the rabbit, pigeon, and other birds, notably in the lung, liver, and kidney. Recent nodules are composed of a mass of leucocytes or epithelioid cells surrounding one or more mycelial filaments. Older nodules show in the centre a felting of mycelia, of which the branches are more vigorous at the circumference than in the centre. In some instances the nodule is represented by a single giant cell, whose protoplasm contains a branching of mycelia, which may be living or undergoing degeneration. The *membranous exudates* are flat, flaky, or lumpy, with an irregular outline, often resembling that of a monkey-nut, of a dirty yellow, greenish, or bluish colour, and of at first a soft consistence, but later on of a tougher character, often simulating that of tough cheese. The lumps seem to be made up of irregularly concentric layers by the gradual addition of fibrin, leucocytes, micrococci, and the elements of the aspergilli. These masses show fatty changes. Microscopical examination of the outer layer of the exudate, whether from the air-passages or peritoneum, show filaments of intersected mycelia of a bright greenish-grey colour, sterigmata and conidia. The deeper layers or older lesions show an immense number of spores.

The air-passages, such as the trachea, bronchi, and air-sacs, are sometimes blocked with thick and tough lumpy masses of exudate. In the peritoneum there may be small pockets, having thickened walls containing a turbid liquid, rich in spores, but no mycelia. In other cases this membrane shows abundant small greenish tufts, containing conidia and other fungal elements. In still other cases the peritoneum as well as the membrane of the air-sacs show small nodules no larger than a pin-head, and containing mycelia.

In poultry, pigeons, and even in some of the aviary-confined larger birds, especially in those that are forcibly crammed, cheesy masses, varying in size from a pea to a fowl's egg, are not rarely found in the mouth, especially on its lateral surfaces or on the floor. The œsophagus is also involved in the diseased process. It may show lines of yellowish beads, varying in size from a pin-head to a pea, containing a yellowish material and aspergilli, running longitudinally the whole length of the organ. The tissue involved appears to be the mucous follicles of the œsophagus. In some instances in which during life there seemed to be paralysis of the œsophagus and inability to swallow during the cramming process, barley-meal, bread and milk, and other food may be found filling the whole length of the lumen of the œsophagus. Particles of ingesta may also be found in the trachea.

In the limbs more recent lesions show soft swellings in the areolar tissue, especially round the joints, containing a pearly-white, stringy pus, intermingled with an abundance of mycelia, sterigmata, and conidia, almost pure in culture. Older lesions are hard, yellowish, cheesy, or calcareous.

Very acute infestation causes hæmorrhagic lesions, and a more chronic one nodular lesions, and not rarely caseous or calcareous changes.

In the *egg* the more or less developed embryo is dead, but there is no trace of rotten odour. The fungus invades the air-chamber or the thickness of the vitelline wall of this chamber, and attacks the vitellus in the form of mycelial masses, or, when far advanced in the incubation process, the embryo itself, on the surface of which it develops. Infestation of the egg seems to take place by entrance of the spores through the porosities of the shell. Microscopical examination of sections of the decalcified shell show an abundance of branched fertile mycelia on the free face of the shell membrane, and a compact felted mass of luxurious mycelia infiltrating the whole thickness of this membrane, and penetrating the interstices of the albuminoid meshwork of the shell.

II. ASPERGILLOSIS IN THE OX.

This disease is of importance because it somewhat resembles, on the one hand, contagious pleuro-pneumonia, and on the other tuberculosis. Zürn, in 1876, found the *A. fumigatus* in the trachea of a cow which had had tracheotomy performed on it. G. Röckl, in 1885, and Piani, in 1886, observed the disease. In 1894 Lucet described a case, as did Bournay in 1895. Pearson and Ravenel recorded in 1900 another case occurring in a six-year-old Jersey cow. In 1907 Fölger reported a case of pneumomycosis manifesting itself soon after calving. He considered the infestation started in the uterus, and was carried to the lung by the blood-stream. Moussu considers it is only an accidental disease in ruminants, and that the infestation gains the lungs by inspiration of the fungal spores, and develops only in those animals weakened by disease and bad sanitation. Although only a few cases are on record, this is no indication that the malady is rare. But considering the widespread practice of giving mouldy forage to cattle, it is surprising that so little of the malady is known in them. If the spores of the Aspergillus are virulent, as imagined, the disease should be more prevalent. As it is, according to evidence, the ox seems very resistant to its development. Perhaps it will be shown on further investigation that the disease is not so rare as supposed.

Symptoms.—The symptoms are not very characteristic, and the true nature of the disease is generally only found on post-mortem examination. In some cases the animal does not thrive, the appetite becomes diminished or lost, there is weakness, dulness appears, the breathing becomes laboured and quickened, there is at first a dry cough, which afterwards becomes paroxysmal and frequent. When the disease involves a large surface of the lung, the sides lift, and even the movements of the chest become discordant. Expiration is generally accompanied by moaning or grunting.

On percussion of the chest wall, one may detect areas of dulness or diminished resonance over those parts of the lung where the lesions are situated near its surface. In some cases the percussion note may be clearer and louder than normal, or even a musical sound, resembling that given off from a fine glass when struck, may be obtained.

On auscultation, the vesicular and bronchial murmurs may be increased in intensity, and accompanied here and there by sibilant râles.

The temperature is rarely high, unless the infestation is associated with some active infection, such as acute tuberculosis or septic metritis. There may be a nasal discharge, in which the fungus may be found on examination.

Course.—The disease is generally chronic, and runs a course varying from one to several months. It may, however, run a very rapid course, and terminate life within a week from the commencement of its clinical appearance.

Diagnosis.—It may be confounded with tuberculosis when this latter disease is not associated with it. A positive tuberculin reaction is a fair indication that such a case is one of tuberculosis, but a negative reaction in presence of a lung disease should not negative the presence of tuberculosis as that disease may be associated with aspergillosis. Detection of the Aspergillus in the nasal discharge affirms the nature of the malady. In order to confirm this it is sometimes necessary to have recourse to cultures in Raulin's fluid.

Prognosis.—This is always serious, especially when the disease is associated with tuberculosis.

Lesions.—The nodules in the lungs vary in number or may be innumerable; they vary in size from a hemp-seed to a lentil or even filbert. There are discrete patches of interstitial and vesicular emphysema, which give to the hand a crackling sensation. On section, the numerous dark red nodules may be seen in the surrounding normal tissue. The wall or fibrous or connective tissue capsule is sometimes covered with a grass-green fungal mass, containing a well-defined central nucleus, composed of mycelia, sterigmata, and spores. In acute cases the lung tissue surrounding the lesion is hepatised. Diffuse hepatisation is rarely observed.

In some acute phases in certain chronic cases interstitial hæmorrhages may occur in various organs of the body. They may be found on the skin, on the surface of an organ, or in various depths of it. These hæmorrhages somewhat resemble those seen in purpura hæmorrhagica. In the lung hæmorrhagic solidifications may be observed reaching up to the size of one's fist.

Treatment.—Turning out in the open, giving easily-digested and nutritious food, and arsenic and iodine or iodide of potassium in the food, or intratracheal injections of iodine, turpentine, paraffin, or salicylic acid suitably diluted, should be given a trial.

III. ASPERGILLOSIS IN THE SHEEP.

In 1891 Mazzanti found the lung of a lamb containing nodules scattered about the lung substance. They were from the size of a poppy-seed to that of a hemp-seed, surrounded by a ring of hepatisation, sometimes grouped together in threes or fours, forming a single mass, and containing a puriform material, in which were spores and mycelia radiating outward from the seat of the air-cells. The species of parasite was not identified.

IV. ASPERGILLOSIS IN THE HORSE.

This disease in equines was first described by Rivolta, who encountered it affecting the sinuses of the head and the lungs of a horse as far back as 1856. In 1862 Mitchell and Varnell recorded an outbreak of a fatal disease in several horses caused by the ingestion of oats covered with an Aspergillus. The chief features of the malady were a paralysis of the tongue and limbs, and sometimes a quickened breathing. Experimental inquiry demonstrated that the incriminated oats conveyed the malady. No microscopical examination of the lesions was made, or at least reported. In 1873 Rivolta described two cases occurring in a horse, and gave the disease the name of *Gutturomycosis*, and considered it to be due to a parasitic fungus which he denominated *Gutturomyces equi.* In 1876 Pech observed the affection in seven horses after eating mouldy straw. Zürn in the same year stated that he had observed aspergillary pneumomycosis in several horses. In 1880 Schütz described it. In 1881 Bassi encountered it in a horse and a mule. In 1884 P. Martin recorded a case of pneumomycosis in a four-year-old horse. In 1893 Goodall related a case of otorrhœa accompanied by epileptiform seizures in a horse; in the otorrhœal discharge he found a fungus, which he named *A. nigricans*, and to which he attributed the disturbance. In 1894 Thary and Lucet described an acute septicæmic form of pneumomycosis in a four-year-old mare. In 1902 Mazzanti published a case of gutturomycosis with stomatitis in a six-year-old horse. In 1903 J. N. Ries gave a detailed account of another case in a twelve-year-old horse.

It is very probable that disease caused by the *A. fumigatus* is far from rare in the horse, but that its true nature is seldom recognised or suspected. Some authorities consider the fungus gains an entrance to the lungs and other channels on foreign bodies, or develops only in the system of those weakened by overwork or previous disease. But experimental evidence is against this latter view, and in Mitchell and Varnell's cases the giving of aspergillosed oats to healthy horses caused disease and death in a few days.

Symptoms.—The clinical appearances of this disease may resemble those of a sore-throat, stomatitis, contagious pneumonia, bronchitis, pulmonary emphysema, pneumonia due to foreign bodies, bulbar paralysis, septicæmia, anthrax, or a profound anæmia.

In most of the observed cases there has been a great difficulty in swallowing food or water, the latter being returned by the nostrils or mouth. During an attempt to swallow either liquids or solids, spasm of the pharynx or œsophagus, sometimes followed by coughing and expulsion of the swallowed material, may be observed. The parotid region is sometimes hot and painful, and the head is extended. There is great depression, disinclination to move, and extreme emaciation. The breathing is difficult, and the abdomen is tucked up. There is usually no rise of temperature unless the lungs become involved.

When the lungs become implicated the following symptoms are present, viz.—a rapid short breathing, dilatation of the nostrils with hæmorrhagic discharge, pain evinced by moaning or grunting on percussion of the chest or on movement, muscular tremors, tumultuous beating of the heart, small, almost imperceptible, pulse, sweatings, cold extremities, injected conjunctivæ, and a high rise of temperature. In fact, there may be all the usual signs of a mechanical or gangrenous pneumonia.

In many instances the first manifestation of the malady is an ulcerative stomatitis, soon followed by dysphagia. In a few cases the disease manifests itself as a chronic bronchitis or pulmonary emphysema, with a prolonged course. In Bassi's case in the mule there was a profuse nasal hæmorrhage resulting from extension of the ulcerative process from the mucous membrane of the guttural pouch into the carotid artery. Some cases of gutturomycosis seem to arise from extension of an aspergillary stomatitis, the original or primary lesions being situated on the mucous membrane lining the mouth.

Diagnosis.—Microscopical examination of the nasal discharge or of the pharyngeal mucus is the only means of establishing a correct diagnosis by finding the mycelia, spores, or conidia or their remains, and thus excluding bulbar paralysis, strangles, influenza, and other obscure pharyngeal disturbances, probably of mycotic origin. In the chronic cases tuberculosis or glanders may be excluded by the use of tuberculin or mallein. From stricture, obstruction, or simple spasm of the œsophagus a differential diagnosis is possible by the careful study of the clinical history and a suitable exploration.

Lesions.—The lesions may resemble those encountered in gangrenous pneumonia, septic pleurisy, septic broncho-pneumonia, diphtheroid pharyngitis, septicæmia, or miliary tuberculosis.

In the septicæmic cases the pleural membrane, lung, and heart, are dotted all over with dark hæmorrhagic spots; the pulmonary pleura is raised by blackish patches of consolidated lung tissue, which are also scattered all through the lung substance, and vary in size from a pea up to a fowl's egg, or even to that of one's fist; the pleural cavities contain a quantity of blood-stained effusion. Similar nodules may be found in the liver and kidney. The centre of the nodules may at times be purulent or caseous, according to the age of the lesions. The kidneys are swollen to double their normal size, and friable. Scattered all through their substance are small reddish points varying in size from a pin-head to that of a sixpence. The circumrenal cellular tissue is infiltrated with a yellowish gelatinous fluid. On section of the kidneys a great amount of blood escapes, and the cut surfaces show numerous hæmorrhagic infarcts. The blood does not coagulate very well, but it is of a blackish colour.

The mucous membrane on the inside of the guttural pouches may be swollen, blackish, and contain muco-pus, or appear whitish and glistening, and showing one, two, three, or more, slightly raised irregularly circumscribed patches, varying in size from a sixpence to a two-shilling piece. These patches have a greyish-white or blackish appearance, and are more or less adherent to the underlying tissues, resembling the false membrane of diphtheria. The substance of these patches contains mycelia and spores of the *A. fumigatus*, also various bacteria and inflammatory products.

Microscopical examination of the lung lesions show emphysematous and hæmorrhagic interstitial changes, and also spores of the fungus. The kidneys show characteristic lesions of parenchymatous nephritis, and the presence of spores. The nerve fibres of the glosso-pharyngeal, pharyngeal branch of the pneumogastric, the hypoglossal, and of the superior cervical ganglion, have undergone degenerative changes, and are invaded by connective tissue new growth, and tufts of mycelia.

Disturbance of these nerves, or more probably their centres of origin, would account for those cases accompanied by lingual paralysis, dysphagia, pharyngeal or œsophageal spasm or paralysis, hurried breathing, and general paralysis.

Treatment.—Nothing short of swabbing the pharyngeal mucous membrane with tincture of iodine or nitrate of silver would be of any use in acute cases of pharyngeal aspergillosis. An electuary containing salicylic acid might be tried in mild cases. Spraying the throat by means of a tube passed up the nostril might also be given a trial. When the lesions are situated in the cavities of the guttural pouches, direct introduction of Lugol's solution or a saturated glycerinated solution of salicylic acid into the pouches by means of an antitoxin syringe should be adopted. When the lungs are involved, Dieckerhoff's intratracheal method of treatment may be advisable. In mild cases iodide of potassium and Fowler's solution of arsenic should be given in the drinking-water, or mixed with the food. Donovan's solution is also indicated as an alternative. If the weather is favourable, the horse should be turned out in the open. On no account should the animal be drenched with food or medicine, especially if there be any pharyngeal incompetence.

V. ASPERGILLOSIS IN THE DOG.

Although the dog is resistant to experimental inoculation of the spores, and susceptible to the toxin of *A. fumigatus*, he is at times subject to the development of the fungus in his system, and the disease results. Gotti, in 1871, described a mycosis of the external auditory canal in the dog. Zürn and also Spinola observed similar instances. Rivolta, in 1885, observed in the lungs, kidney, spleen, and left uterine cornu of a bitch, "encephaloid sarcomata containing filaments interlaced in various directions." Although no cultures of the organism were made, Rivolta gave it the name of *Muromyces canis familiaris*. There was a dry, frequent cough, lameness of one hind-limb, and profound emaciation. In 1886 Olsen and Gadde stated that the rabbit and cat succumbed to inoculation of spores of the *A. subfuscus*. Plant recorded the existence of pulmonary mycosis in the dog, and Stazzi, of Milan, described, in 1905, two cases of nasal aspergillosis.

Symptoms.—In one of Stazzi's cases of nasal aspergillosis due to the *A. fumigatus*, a six-year-old dog suddenly became restless, the eye had a vague, stupid expression, the head was carried to the right and low down, he was irritable and savage, hid himself in secluded places in the house, and sometimes shook his head furiously as if he had something in his ear; and in addition to this he was seized with choreic movements of the muscles of the cheeks, ears, and jaws, and had a paroxysmal and difficult cough. This state was rapidly aggravated, and in five or six days the dog refused all food, and was often seized with epileptiform convulsions. When Stazzi examined the dog at the end of that period, he found the temperature 101·8° F., the pulse was weak and frequent, the respiration quickened, the mucous membranes cyanotic, and the breath fœtid. This examination set up the fits as well as the hoarse cough, the latter resembling more a sob than a true cough. The dog scratched his muzzle with his front paws or rubbed it on obstacles, as if he desired to remove some embarrassing foreign body from his nostrils. The epileptiform convulsions occurred two or three times in the twenty-four hours which supervened before the dog died.

The essential lesions were found in the right nasal passage. The middle portion of the inferior turbinated bone had on it a brownish patch, covered with a layer of the dark green mould, having a strong musty odour. Microscopical examination revealed all the constituent elements of the *A. fumigatus*. Cultures on various media verified the fact, and intravenous inoculations in rabbits and pigeons demonstrated the great pathogenic and toxic action of that fungus. Stazzi found nothing abnormal in the ear, and carried out the necessary researches to eliminate rabies.

In the other case, in a three-year-old dog, there was a muco-sanguinolent discharge from the right nostril; the animal was restless, sneezed, and coughed in a paroxysmal manner, and continually scratched his nose. On examining the nasal mucus for the eggs of the suspected Linguatula, Stazzi found the *A. fumigatus* in the form of non-intersected filaments of 2 to 5 μ in thickness by 20 to 50 μ in length, as well as very small conidia. Antiseptic injections up the nostrils did not give any favourable results, the animal going from bad to worse until the right frontal sinus was trephined and irrigated. In the course of one of these irrigations a kind of resisting membrane giving off a slight mouldy odour escaped from the nasal cavity. On examination of this membrane the diagnosis was confirmed.

Probably the fungus is introduced into the nasal passages by fragments of wood, grass, straw, hay, grain, hay-seeds, etc. Obscure cases of chronic nasal catarrh which do not readily yield to nasal irrigations, even when containing iodine, are not rarely encountered. Probably, on further research by the canine clinician, some of these cases may be found to be due to the *A. fumigatus*. In some cases the nasal discharge may be slight or absent. Aspergillary rhinitis, more than any other inflammatory or neoplastic lesion of the nasal mucous membrane, determines in neurotic, irritable animals severe reflex phenomena, and epileptiform or rabiform seizures.

VI. ASPERGILLOSIS IN MAN.

Aspergillosis has been observed in man by Dieulafoy, Chantemesse and Widal, Potain, Rubert Boyce, Gaucher and Sergent, Rénon, and many others. It may appear in the form of a pseudo-tuberculosis, at times associated with the bacillus of tuberculosis. It occurs chiefly in pigeon crammers, hairdressers, and workers in grain or meal. The *A. fumigatus* has also been found in ear affections, post-nasal catarrh, traumatic keratitis set up by straw, hay, etc.

Public Health.—The observations of Dieulafoy, Chantemesse and Widal in 1890 clearly show the danger man runs in carelessly handling pigeons and poultry affected with aspergillary stomatitis (canker of pigeon fanciers). As the disease is common in all classes of domestic birds and in wild species in confinement in this country, veterinary surgeons should be on the lookout for cases of transmission of the disease from bird to man.

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BLASTOMYCOSIS IN THE GOOSE

BY HENRY GRAY, M.R.C.V,S.

THIS disease is due to a unicellular fungus—*Blastomyces* (*Cryptococcus*) *anseris*—belonging to the Blastomycetes, which are closely related to other yeasts or Saccharomycetes. Among the Blastomycetes, Guiart places the *Oïdium* (*Saccharommyces*) *albicans*, or the parasite causing buccal, œsophageal, and gastric thrush in very young animals and children bottle-fed on cow's milk (*vide* vol. ii.). The Blastomycetes are reproduced, as in the case of the yeasts, by budding and by asci, and may take on the form of a filament or of a yeast. They have been found in various lesions in man and animals, but occur usually as saprophytes on fruit, grain, and other vegetable structures.

The *Blastomyces* (*Cryptococcus*) *anseris* has been found in the fatty livers of geese crammed specially. They are enclosed in small sacs, with a sessile base, by which they are attached to Glisson's capsule, rarely, if ever, to the liver tissue proper. These sacs are more or less numerous, varying in size from a pea to a filbert, connected with each other by narrow channels, of a yellowish-white colour, fluctuate, and resemble in appearance a chain of echinococci cysts along the border of the liver. They contain a yellowish-white gelatinous material, which, on standing, becomes a pus-like liquid. The Blastomycetes are round or oval in shape, discrete or confluent in a gelatinous matrix. The parasites are refractile, and have a distinct membrane enclosing a nucleated mass of protoplasm. They may undergo budding, one or more small rounded bodies being visible at one or other pole, the parasites varying in size from 1·5 to 30 μ in diameter. They readily grow on glycerinated potato, glycerinated agar, and broth at ordinary room temperature. Inoculations of cultures on guinea-pigs and rabbits give negative results; in pigeons the results are indifferent, and not very trustworthy.

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THE SPIROCHÆTOSES

By H. GRAY, M.R.C.V.S.

Synonyms.—Spirilloses.

French : Les Spirilloses.

Definition.—A class of diseases due to the presence of a specific spirochæte in the system, the species of parasite varying according to the species of animal affected or the district in which the disease occurs.

GENERAL REMARKS ON THE SPIROCHŒTA (Ehrenberg, 1838).

According to Blanchard, all modern authors write "spirochæte," after Cohn (1875), which method of spelling he considers is faulty, as it conforms neither to the rules of Latin nor to zoological or botanical nomenclature.

The body of the spirochæte is excessively slender, spiral, and flattened The ectoplasm is distributed in the form of a slender undulating membrane, which surrounds the whole body in a spiral form. It has no flagella and no endogenous spores. A very long filiform nucleus occupies the axis of the body, with grains of chromatin distributed over its surface. Multiplication occurs, as far as is known, by longitudinal division.

According to Schaudinn, the spirochætes are closely related to the family of trypanosomes, and may in certain cases, as in that of the *Spirochœta ziemanni*, even be a stage in the life-cycle of a trypanosome. Numerous spirilliform organisms are known, but there is a great difference of opinion as to their true nature. Some of these resemble bacteria, but being very slender, it is difficult to make out their true structure. The genus *Spirillum* is, it is said, differentiated from the genus *Spirochœta* by the presence of an undulating membrane, blunt ends, and a chromatic substance extending the whole length of the body in the latter, characteristics which are absent in the former.

The spirochætes are widely distributed in Nature, existing in stagnant water, sea-water, decomposing organic matter, or as saprophytic parasites in man or animals. Some, however, have a high degree of virulence, and produce infective diseases running a more or less rapid course.

They have been found by Baruchello and Pricolo* in the pleural and pulmonary alveolar exudates in contagious pleuro-pneumonia of the horse; by Ganzer and others in dental caries, tartar, and saliva; by Jowett in grease and canker; by Beebe and Ewing,† and also by Mettam, in the vaginal lesions of the infective lymphosarcomata of the dog; by Lucet and also Regaud in lesions of epizoötic gastro-enteritis of the dog; by Bizzozero, who states that they always exist in the gastric cells of the dog; by Ball and Rouquet in the stomach and intestinal canal of the normal cat and dog; by Schaudinn and Hoffmann in syphilitic lesions (the *S. plicatilis* and *S. refringens*); in ulcerating surfaces of carcinomata, etc.; and by Gilruth in intestinal lesions in the pig.

Spirochætes have been found in the oyster (*S. Balbianii*) and in other molluscs, also in sea-fish; a new species in the chyle-stomach and hind-gut of bees by Fantham and Porter; in a great number of insects; in the intestine of the bird (*S. Eberthi*); in the rabbit (*S. Raillieti*); in the blood of mice [*S.* (*Treponema*) *muris or Laverani*]; in the blood of the rat (*S. muris* var. *Virginiana*); in the blood of the bat (*S. vespertilionis*); in monkeys, otter, gondi.

Cleland found a spirochæte, to which Bosanquet has given the name of *S. aboriginalis,* in the ulcerative granuloma of the pudenda of Australian natives. It is unlike the *T. pallidum,* the spirals being quite irregular. Spirochætes are also found in the camel, elephant, and in antelopes.

In all these instances they are only accidental, and not the cause of the processes they occasionally accompany. On the other hand, quite a number of spirochætes are now known to be the true cause of pathological processes. Such are the well-known *S. obermeieri,* which is the cause of relapsing fever in man, and the *S. pallida,* the causal factor of syphilis in man. For a very long time the spirochæte of relapsing fever was termed a "spirillum." Thiroux and Dufougere found a spirochæte in the blood of *Ceropithecus patus* in Senegal, and gave it the name of *S. pithecus.* It causes an irregular fever, and has only been transmitted to mice and rats. It is of the *S. Duttoni* type. Many authorities describe five types of spirochætes causing recurrent fever in man—viz., the European *S. obermeieri,* the African *S. Duttoni,* the American *S. novyi,* the Asiatic *S. carteri,* and the Algerian and probably Egyptian *S. berbera.* They are conveyed by ticks, body-lice, bed-bugs, and probably fleas. Schaudinn thinks the spirochæte of syphilis should be included in the new genus *Treponema* created by him, and termed the *Treponema pallidum.*

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* *La Clinica Veterinaria,* April 14, 1906.

† *Journ. Comp. Path. and Therap.,* vol. xix., part iv., December, 1906.

A spirochæte (*S. pertenuis*) has been discovered in yaws. The widely distributed spirochætosis of the fowl is caused by the *S. gallinarum* and its varieties. In the spirochæte of relapsing fever and that of fowl spirochætosis, Borrel and Zettnow have discovered flagella, and in consequence some authorities think these organisms should be classed as bacteria, and not as protozoa.

A spirochæte has been found to be the cause of Vincent's angina. Tick fever of man is due to the *S. Duttoni*, and the spirochætosis of geese to the *S. anserina*. Spirochætes have also been found in the Chinese buffalo by Heanley; in the pig by Gilruth; in the ox by Theiler; in calves in the Cameroons by Ziemann; in the sheep by Theiler, and also by Martoglio and Carpano; and in the horse by Stordy and Martin, and considered by their respective discoverers to be the cause of febrile and other symptoms, and of various lesions.

According to some authorities, the spirochætes of the goose, fowl, and cattle should be placed in the genus Treponema, in which the syphilitic and frambesian, or yaws, organisms are included. The distinguishing features between the genus Spirochœta and that of the Treponema are, the first has an undulating membrane, no flagella; and the latter, no undulating membrane, but flagella. The *T. pallidum* of human syphilis has been transmitted to the chimpanzee, orang-outang, gibbon, and macaque—all simian apes. A spirillum (*S. nigrum*) occurs in chronic ear suppurations.

Finally, Bordet * of Brussels, in subcultures of a bovine contagious pleuro-pneumonia culture made on a medium containing defibrinated blood, found the medium became black along the needle track, but saw no growth by the naked eye. Cover-glass preparations stained with hot Giemsa did not show the simple amorphous granules seen in the original culture, but perfect spirochætes, which were very slender and fairly long, having the spirals sometimes rather close together, were encountered.

This spirochæte of the lung-plague of oxen varies considerably in length, and is sometimes merely comma-shaped. On solid media the spirochæte is rather longer, but very slender. It is motile.

It is shorter and finer than the *T. pallidum* of syphilis, not so easy to stain, and more difficult to see.

But the observations of Borrel, Dujardin-Beaumetz, Jeantet and Jouan, do not agree with that of Bordet, who used in his experiments a culture obtained from Dujardin-Beaumetz's laboratory. These authors find on ultramicroscopical examination that the organism of bovine contagious pleuro-pneumonia is absolutely immotile, and very polymorphic, manifesting itself in the form of a diplococcus, tetragenus, morula, or streptococcal filament, embedded in a viscid matrix. The pressure of this matrix leads to the formation of pseudo-mycelial filaments. The bi- or tri-polar aspect is fairly frequent. These authorities propose to give the name of " asterococcus mycoides " to this organism in consequence of its structure being made up of a mucous sheath, and pseudo-mycelial filaments having many poles.*

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* *Bull. de la Société Royale des Sciences Médecine et Nat. de Bruxelles*, November, 1909.

In this section those diseases in the domestic animals due to spirochætes will be described. They are as follows :

I. Spirochætosis of the Fowl.
II. Spirochætosis of the Goose.
III. Spirochætosis of the Ox and Calf.
IV. Spirochætosis of the Sheep.
V. Spirochætosis of the Pig.
VI. Spirochætosis of the Horse.

I. SPIROCHÆTOSIS IN THE FOWL.

Synonyms.—Spirillosis in the fowl.

French : Spirillose des Poules.
German : Hühnerspirillose.

Definition.—An infective disease of the blood-stream, or a septicæmia of the fowl, due to several varieties of the *S. gallinarum*, and transmitted from the affected to the healthy fowl by the agency of diverse varieties of the fowl tick—viz., *Argas miniatus*, *A. persicus*, *A. victoriensis*,† and probably other ectozoa not yet determined.

Geographical Distribution.—This disease was first discovered in the fowl by Marchoux and Salembini, in Rio Janeiro, in 1903. It has since been encountered in Algiers, various parts of Australia, Bulgaria, Cyprus, India, Martinique, Rhodesia, various parts of South Africa, Soudan, Tunis, the German Cameroons, United States of America, and other parts. Probably it is still much more widely distributed, but not everywhere yet recognised.

Animals Susceptible.—It is naturally and experimentally transmitted to young chickens and susceptible adult fowls, and experimentally conveyed to geese, pigeons, larks, and other birds. It is doubtful if the rabbit, guinea-pig, and rat are susceptible. Experimental passages through chicken to chicken seem to increase its virulence for a time, and then this decreases, as in all natural infections or epizoötics. It is said that it cannot be indefinitely transmitted artificially from fowl to fowl. For it to be virulent the tick becomes the essential factor.

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* *Annales de l'Institut Pasteur*, March, 1910.
† *Veterinary Journal*, September, 1910.

Bacteriology.—According to Jowett, the pathogenic organism may be found existing in the blood of the fowl suffering from the natural infection, either in a free state, or as rounded bodies in the interior of the erythrocytes, together or separately.

1. The *Free Spirochæte* is a delicate, extremely thin, threadlike, spirally-formed organism. Its body is of uniform thickness except at the extremities, where it tapers gradually to a point. Examined in the fresh state, the organism is found to be very actively motile. In fixed and stained preparations the parasites are seen to vary greatly in size. Short forms are encountered, measuring 8 or 9 μ, with three or four spiral turns; others, and these are more commonly encountered, measure 16 or 17 μ, and possess seven, eight, or nine spirals. Even larger spirochætes are occasionally encountered measuring 20 to 25 μ, and in a few instances 30 μ. In the case of these long forms, however, it is sometimes rather difficult to determine whether they are really one parasite, or of two joined end to end. Depending much, apparently, upon the position assumed by the parasite during the process of fixation (previous to staining), it may appear—(1) as a regular and beautifully formed spiral, or (2) it may be looped in a circle, figure-of-eight pattern, or, in fact, assume any form one might imagine possible, with a long, extremely flexible, delicate, ribbon-shaped body, such as is possessed by this parasite.

Frequently two spirochætes are seen joined end to end. Whilst it is not unusual to see two spirochætes lying close against each other—approximated at one extremity, the others diverging at an acute angle—this is believed to represent a stage in the (longitudinal) division of the parasite. At certain stages of the infection spirochætes may be seen in which a number of transversely arranged unstained areas are present. Collections of the organisms in more or less intricately arranged and interwoven "tangles," or "clumps," are quite frequently seen. This phenomenon is not observed at the commencement of the infection, when the parasites are still scanty in the blood. Later, however, when they have increased in numbers, it is not uncommon for the spirochætes to form clumps, and afterwards to disappear from the blood-stream; this is the so-called "crisis." Thereafter the spirochætes may not again appear in the blood in numbers sufficient to be demonstrated by ordinary microscopic examination, and still the subject may die, although the parasite cannot be detected in its blood. On the other hand, in some of Jowett's experimental cases in *young* chickens, the parasites have increased progressively in numbers until death. In any case, however, they usually disappear somewhat speedily after that event.

2. The *Intracorpuscular Bodies* Jowett encountered in naturally as well as in experimentally infected subjects. As said above, they may coexist with the free spirochætes, or they may disappear before the latter; or, on the other hand, may not be seen until after the spirochætal crisis—*i.e.*, until the spirochætes have disappeared from the blood.

The intracorpuscular bodies encountered by Jowett were never more than four found in one erythrocyte. Usually only one or two are observed in one erythrocyte, and occasionally three or four. These "bodies" are generally rounded in form, and invariably are situated in the extranuclear portion of the erythrocyte. They are, perhaps, more often found at the end of the cell than in any other position; still, it is not rare to see them laterally placed. They may be situated quite close to the edge of the nucleus, or, on the other hand, may border the periphery of the cell. Very frequently they occupy a midway position.

The young, recently-formed bodies are uniformly stained (dark red or purple, with Romanowsky stains—the chromatin staining reaction in fact) throughout. Other bodies appear only partially stained, a portion of the body being purple coloured, the remainder unstained. Still others of the "bodies" are stained only at the periphery, the remainder (interior) of the "body" being entirely unstained. Finally, others are stained at the periphery, and enclose a number of granules. Occasionally Jowett has encountered a blood-corpuscle containing a collection of these granules, unenclosed apparently by any peripherally stained ring or capsule; this phenomenon, however, is rare. In size the bodies vary: the smallest measure 1 μ, or considerably less, whilst the largest may attain in measurement a diameter of 4 μ.

The other blood changes usually associated with the presence of the parasites are polychromasia, vacuolation of the cells (especially of the leucocytes), and, in short, the general blood changes one associates with anæmia. Such is the version given by Jowett* after his study of the Cape Town spirochæte. Dodd did not find the "after-phase" bodies in fowl spirochætosis in Queensland. Gilruth did not observe endoglobular forms in the Victoria outbreak.

According to Brumpt, the parasitic spirochætes in fowls differ according to locality. He subdivides them into—

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* Jowett, *Veterinary Journal*, April, 1911.

1. *S. gallinarum vel Marchouxi* (found in Brazil, Somaliland, etc.).
2. *S. neveuxi* (Senegal).
3. *S. nicollei* (Tunis).

Balfour believes the Soudanese strain of spirochæte at which he worked is a specific entity, and has proposed for it the name *S. granulosa penetrans*. Pigeons are insusceptible; gerbils are immune to inoculation. Chickens fed on ticks containing either spirochætes or granules, or granules alone, become readily infected. Ticks are infective for as long as three months after the last ingestion of infective blood. He thinks his form of parasite is identical with that of Jowett's.

Microscopical Demonstration.—The quickest method of demonstrating the spirochæte with an ordinary microscope and an oil-immersion lens is to place a drop of normal saline solution in which a little of the infected material has been rubbed up, on one end of a slide, and close beside it a drop of finely prepared Indian ink. Allow the two drops to run together, and then quickly spread the liquid across the whole of the slide, as in making a blood-film. This shows the spirochætes as colourless bodies on a black background. The ultramicroscope also readily shows them up.

Cultivation.—Levaditi* was successful in cultivating this organism in series. He used for this purpose collodium sacs placed in the peritoneum of the rabbit, an animal not very susceptible to the spirochætes. The sacs contained fowl serum previously raised to 72° C., and the spirilla rapidly multiplied and preserved their virulence at the end of eight passages.

The Infecting Ticks.—The American *Argas miniatus*, the more widely disseminated *A. persicus*, and the Australian *A. victoriensis*, are nocturnal parasites which feed during the night, and hide away in crevices, cracks, and behind objects during the daytime. During the night they leave their hiding-place, and crawl on to the birds, which they bite, and abstract blood, and at the same time inoculate their host with the spirochæte, which they have obtained from an affected bird. *Ornithodoros moubata* is said also to transmit the *S. gallinarum*, which may furthermore be capable of infecting the bird *per os*, indicating that the organism can pass through the mucous membrane of the alimentary canal. The *Dermanyosus avium*, or red-mite, is not a carrier. Fowl-lice (*S. menopon*) are capable of transmitting *S. granulosa penetrans* Balfour.

Occurrence.—Although this disease is most prevalent during the spring months of the year, it may occasionally be seen at any time of the year, especially in the case of an introduction of fresh birds into an old farmyard or run where ticks are plentiful. According to Bevan,* birds kept on an infected place become immune to the disease; but at the same time this immunity would appear to be of short duration, for it has been repeatedly observed that these apparently immune birds will succumb after the newly arrived birds have restarted the infection. And although a fowl may have recovered from the Soudanese type of spirochætosis (*S. gallinarum vel marchouxi*), it may react to the Tunisian or Senegal type (*S. neveuxi*), though it might fail to do so with the former type. Still further, fowls recovered from the Soudanese type are yet susceptible to the Tunisian type (*S. nicollei*), which, however, does not protect against itself. The duck, goose, Java sparrow, sparrow, and chaffinch, are susceptible to *S. neveuxi;* and while young fowls are also susceptible, adult birds seem immune to bites or to inoculations with *S. neveuxi.*

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* *Société de Biologie*, Séance du 7 Avril, 1906.

Period of Incubation.—Two or more days, judging by the appearance of the parasites in the blood, which may be infective long after a recovery.

Symptoms.—For the convenience of description the disease may be divided into two types, the acute and the chronic, as has been everywhere encountered.

Acute Type.—A very high fever (109° to 110° F.), as indicated by the thermometer inserted into the cloaca,† is the first warning that the disease is developing, but to the ordinary observer intense thirst is the first noticeable symptom. This is soon succeeded by an abstinence from food and an opening or ruffling of feathers. The animal becomes dull, disinclined to move, does not care to follow its fellows, and has profuse diarrhœa of a very offensive odour. Emaciation and anæmia become pronounced; the eyelids are closed, or semi-closed, and the bird rests on the ground, being so weak as to be incapable of standing. Death takes place in this somnolent state, or sometimes quite suddenly.

These symptoms do not always coincide with, or precede, the discovery of the parasites in the blood. As a rule, the parasites are in great numbers a few days after infection, when they rapidly increase towards the height of the malady, and gradually decrease as death approaches. Where recovery takes place, they rapidly decrease. In a few instances the spirochætes may be detected before any symptoms are observed.

Chronic Type.—This may result from the acute form, or arise independently. After an acute attack the parasites may not be detected in the peripheral blood, but recovery in many instances is not absolute, paralysis of some part of the body appearing after a variable interval, sometimes of weeks. The loss of power appears either in the legs, wings, or neck, but chiefly the former, the fowl being unable to walk, or with some difficulty. Paresis arises gradually, being first observable by the difficulty the bird has in using the claws, which are often doubled up as if from cramp. When the paralysis is complete, the bird appears bow-legged or knock-kneed, or its limbs double up under it when it tries to walk. If disturbed, it falls on the ground. When the neck is affected it is so twisted that the bird looks continually over its back. In some instances the paresis disappears completely of itself. In others there is gradual emaciation, and death then takes place without any parasites being discoverable. The elevation of temperature is not a marked feature. The chronic form may appear primarily, but no organisms can be detected in such cases.

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* *Journ. Comp. Path. and Therap.*, vol. xxi., part i., pp. 43-49, 1908.

† The average normal temperature of the fowl is 106·5° F.

Diarrhœa may appear, and the feathers around the cloaca becomes caked with fæces.

Course.—In chickens a few weeks old the disease runs a very rapid course, but in adult fowls it is less fatal. An acute case may terminate in death two to seven days after the appearance of parasites in the blood. An apparent recovery from an acute case may be followed by a relapse, ending in death in a few days, or by a slowly progressing chronic type. In young chicks it may rapidly take place before the parasites appear in the blood.

Lesions.—The most pronounced lesions after death are extreme anæmia and a boiled or macerated appearance of the muscles. In acute cases, when death takes place at the crisis, the liver and spleen are hyperæmic, and the latter organ may be considerably swollen. The liver often shows disseminated pin-head points of necrosis. The small intestines are sometimes congested, and occasionally show punctiform hæmorrhages.

The surface of the body often shows small subcutaneous hæmorrhagic points due to the attachment of the larval ticks. In the case of the body of the bird being covered with a number of larval ticks during life, there is scarcely any blood found in the system after death. On the contrary, where few or no ticks are observed during life, there may be no signs of loss of blood, but lesions resembling those of septicæmia.

Fatty degeneration of the heart and liver may be present, but these are considered to be foreign to the disease.

The parasites disappear soon after death, but occasionally they may be detected. Inoculation of blood or emulsion of spleen may transmit the disease for some hours after death.

In the chronic form anæmia and emaciation is most pronounced, and the spleen is often atrophied.

Treatment.—The aim of the poultry farmer should be to rid his premises of the transmitting agent of the spirochætes from bird to bird. This is not only more economical, but more successful in results. Therefore the premises should be thoroughly cleansed and disinfected so as to destroy all the ticks. One of the most economical disinfectants for this purpose is the "green creosote oil of commerce," which is used largely in the pickling of railway sleepers and other timbers. It is very cheap, and therefore may be used freely in painting or saturating the walls, egg-boxes, fowl-houses, and perches, after these have been scraped and perhaps washed. It is very effectual in destroying all kinds of parasites living in the fowl-house, and is, moreover, a powerful preservative of all timbers.

In the case of the fowls being actually infested with vermin, the birds should be thoroughly dipped in a bath of sulphuretted lime solution. But the weather should not be wet or cold when this is undertaken, for fear of chilling the birds.

As a *curative* when the birds are already infected, Uhlenhuth, Gross, and Bickel state that they have found 5-centigramme doses of atoxyl, given at the time of infection, or two or three days after, are capable of preventing infection or effecting a recovery, although the blood of the fowls so treated remained infective. Soamine, $\frac{1}{15}$ grain in 1 c.c. of sterile water, injected into the muscle, generally aborted the attack, and ultimately a recovery from the disease is brought about.

Ehrlich-Hata "606," or salvarsan, acts as a powerful spirillocide in spirochætosis of the goose, and is capable of rendering an infected animal immune when the precipitate is prepared according to Ehrlich's method. Balfour has tried it on fowls, and when given in sufficient doses he found it banished spirochætes from the peripheral blood, and in certain cases "cured" the bird. In other cases the granule phase supervened, on which the drug has very little, if any, effect. Abrin will kill *S. gallinarum*, and thus produce a vaccine which will protect canaries.

II. SPIROCHÆTOSIS IN THE GOOSE.

Synonyms.—Spirillosis in the goose.

French : Spirillose des oies.
German : Gänsespirillose.

History.—This disease was first discovered in 1890 in the Transcaucasus by Sacharow, who gave to the causal organism, which swarmed in the blood, the name of *S. anserina.* Gabritchewsky, in 1898, made a long study of the relationship between the spirillosis of geese and the recurrent fever of man, and obtained an immunising serum. Cantacuzène, in 1899, determined the method of destruction of the spirochætes in the blood, and brought forward fresh facts concerning the procedure of infection. In 1903 the disease was observed in Tunis. In 1907 Dschunkowsky and Luhs encountered it in the Caucasus, and assert it is more common than generally suspected.

Bacteriology.—The *S. anserina* is from 6·86 μ to 15·01 μ in length, and about 0·21 μ in width. The number of spirals is from two to seven; their length is from 2·00 μ to 3·05 μ, and their depth attains from 0·28 μ to 1·43 μ. This spirochæte has at each extremity a cilium, which varies in length. Sometimes there is only one, but more often both cilia are entirely absent. They are very motile, and move like a corkscrew on their long axis by spiral undulations, or, rather, by an apparently rigid movement of their elements.

The spirochætes are visible in the blood without previous staining. If obtained at the commencement of the infection, they often run into one another, forming very loose bundles of radiating spirochætes. They are difficult to stain.

To obtain a quick manifestation of the cilia and of pronounced staining, the blood should be smeared on clean glass slides, treated with hydrochloric acid and absolute alcohol, passed through the flame, and then dried in the air. The slide is then washed with a strong, but fine, jet of cold distilled water. It is then dried by means of blotting-paper. A few drops of Löffler's or of Bunge's mordant solution are poured on the slide, which is then heated, until boiling, by the flame of a Bunsen burner, and afterwards washed with a strong jet of distilled water, and finally dried with blotting-paper. The preparation is subsequently treated with a few drops of an aqueous solution of gentian violet or of fuchsin, heated to boiling-point for five or six seconds over a Bunsen flame, washed thoroughly with a strong stream of distilled water, and immediately examined when wet or after drying with blotting-paper, and mounted in Canada balsam. This procedure only requires one or two minutes, and the spirochætes are stained two or three times darker than with the usual stains for micro-organisms, the flagella being distinctly visible.

Zettnow's method (in which chloride of antimony and ethylate of silver are used), as well as the methods of Weidenreich, Hoffmann, and Halle, give very satisfactory results.

Besides the goose, the disease can be transmitted by inoculation to the turkey, canary, crow, magpie, sparrow, and lark ; but it is rarely fatal in them. Pigeons are refractory. Very young chickens are not very susceptible, and rarely die from inoculation.

The goose and duck are susceptible to experimental natural infection with *S. Nicollei* and also *S. neveuxi*.

Symptoms.—The birds become weak, have little or no appetite ; they are dull, and assume a sitting position, bunched up in a heap. Diarrhœa appears, and the mucous membranes and featherless parts are pallid. This goes on for a week or longer, and the birds die from exhaustion, after having manifested a temperature of 108° to 110° F. Some geese have very painful swellings of the foot-joints, but Dschunkowsky and Luhs have not observed these.

Lesions.—There is extreme emaciation of the carcass. The liver is hyperæmic, swollen, and of a brownish or yellowish-red colour. The spleen may be very much swollen to two or three times its normal size ; it is of a dark purplish colour, soft, and very friable. If the disease should have lasted six days, the liver is found to be undergoing fatty degeneration, and contains numerous more or less large necrotic centres, or small yellowish caseous granulations the size of a millet-seed. The spleen has also these yellowish necrotic areas. (These, however, have nothing characteristic about them, as they are encountered in many infectious processes in birds.) The intestines are very much congested or inflamed. The blood and visceral organs no longer contain the spirochætes.

Treatment.—Atoxyl is a specific against this disease. An injection of 0·3 to 0·5 of this agent made two hours before infection, or even at the time, generally prevents the development of the disease. It is also a curative when the disease is manifest even of three to five days' standing. Two doses of 0·3 to 0·4 of this substance, injected twenty-four hours after one another, prevent fatal results, although the blood may contain a very great number of parasites. On recovery very great or firm immunity is obtained.

The curative dosage of atoxyl is 0·10 to 0·15 per 1 kilogramme of the body-weight of the bird. A dose of 0·20 is fatal.

Although the organs of geese lose their infectivity shortly after death, they can, nevertheless, be used as a vaccine, which will confer a high degree of permanent immunity. The serum of immune geese acts as a preventive as well as a curative. This can be rapidly obtained by using atoxyl and infecting geese with spirochætes.

III. SPIROCHÆTOSIS IN THE OX AND CALF.

Synonyms.—Spirillosis of the ox.

French : Spirillose des bovidés.
German : Rinderspirochätose.

History.—In 1902 Theiler discovered in the ox, kept in his laboratory or confined in the vicinity of Pretoria, a spirillum, to which Laveran, in 1903, gave the name of *Spirillum Theileri.* Ziemann shortly after encountered it in calves in the Cameroons, and Koch in East African cattle. Diatshenko found a motile, straight, comma-shaped, or **S**-shaped organism, which he called *Spirillum tschichir,* in the fresh spleen of Caucasian oxen suffering from hæmoglobinuria. It was easily cultivable, and is evidently not the parasite discovered by Theiler. In 1904 Heanley observed a spirochæte in the blood of young Chinese buffaloes suspected of cattle plague or of septicæmia hæmorrhagica.

In 1904 Theiler demonstrated that his spirochæte was transmitted from diseased to susceptible cattle by means of adults and larvæ of the blue tick *Boophilus decoloratus* and of *Rhipicephalus evertsi.* This was afterwards confirmed by Laveran and Vallée. In 1904-05 Theiler was successful in transmitting the disease from affected cattle to other cattle not bred in the district, also to susceptible sheep, but not to the horse. From his experiments he concluded that the horse spirochæte was not identical with that of the ox, and that the ox spirochæte and that of the sheep were the same. In 1906 Dodd made several experiments, and arrived at the conclusion that the spirochæte of the horse, ox, and sheep should be considered identical—viz., *Spirochœta Theileri.* Balfour found similar forms, probably of intestinal origin, in Jackson's hartebeeste in the Soudan; Cummins in the goat; Schein in blood of calves in South Annam. Nuttall describes a blood spirochæte, named *S. boviscaffris,* in the African buffalo. It is very broad, tapers at both ends, and stained specimens show transverse achromatic bands.

Bacteriology.—The *Spirochœta* or *Treponema Theileri* varies in length, the longest reaching 20 to 30 μ; they sometimes have thin or pointed extremities, but others have the same thickness all through, about $\frac{1}{4}$ to $\frac{1}{3}$ μ. The organisms are easy to detect in fresh blood, in consequence of their movements. Magnified by $\frac{1}{12}$-inch oil immersion, they are found to be in general fixed to one or more red corpuscles. Sometimes they are rolled round a red or white corpuscle; and when totally free, they have characteristic undulations, until they affix themselves afresh on a globule.

They are stained by all the usual aniline stains. They are well stained by the Romanowsky-Laveran method and with Azur II.

The number of parasites in the blood vary from time to time. They may disappear to reappear several days later. They may often appear in association with the piroplasma of redwater or of Rhodesian tick fever. They undergo multiplication in the interior of the tick. The *Spirochæta* (*Leucocytozoön*) *ziemanni* becomes almost invisible after undergoing repeated divisions. Theiler thinks the spirochæte discovered by him will probably be found all over South Africa.

Symptoms.—The disease is generally recognised as a benign one, and the clinical symptoms are, as a rule, very slight. If infective blood be inoculated, the febrile reaction begins on about the fourth day, and about a day later the spirochætes are found in the blood, occasionally as early as the third day.

There may also be a slight anæmia, the blood-count in certain cases falling to about 3,500,000 per centimetre, accompanied by poikilocytosis.

Inoculations of triturations of infective larval ticks do not produce a rise of temperature before the fifteenth day, and spirochætes are not seen before the seventeenth day.

When the spirochætal infection is associated with a piroplasmosal infection, the symptomatology is usually that arising from the later infection.

In the *buffalo* Heanley, on post-mortem examination, found very few spirochætes, chiefly in the spleen and blood. They were not observed in an enlarged hæmorrhagic lymph gland. The spirochætes resembled those found by Theiler in the ox. No piroplasmata were found, although a trypanosome has been found in the Chinese buffalo in Hong-Kong.

A spirochæte (with undulating membrane and without flagella) has been found in tuberculous cattle. It was studied by Mezincescu in 1904, and named *S. pyogenes*. Another spirilliform organism of undetermined *genus* has been found in the vaccinal pustules of the calf, and provisionally named *S. vaccinæ* (Bonhof, 1905).

IV. SPIROCHÆTOSIS IN THE SHEEP.

Spirochætes were found in the blood of sheep in the Transvaal by Theiler in 1902 ; by Martoglio and Carpano in Erythrea in the blood of Abyssinian sheep in 1904. It has been denominated *S. ovina* by R. Blanchard and by Novy and Knapp. It shows from three to ten spirals, and is 10 μ to 20 μ in length, by 0·2 μ to 0·4 μ in breadth. The extremities are tapered, the organisms do not stain by the method of Gram, and have not been cultivated on any known media. No flagella, spores, or chromatin, have been discovered even by Romanowsky's method. Two spirochætes are often encountered joined together their whole length, and even fused together at one extremity, as if they had been produced by longitudinal fission. Martoglio and Carpino consider, however, it is produced by transverse division. Others consider both longitudinal and transverse division occur.

The parasite is found in the blood plasma, while the red blood-corpuscles contain an unrecognised organism. Death took place on the seventh day of the febrile reaction. Like the *S.* (*Treponema*) *Theileri*, the disease was not reproduced by inoculation of the blood.

The *S. Theileri* has been, however, transmitted from the ox and horse to the sheep by Dodd, who considers it, as has been before stated, to be identical in all three of these animals. Ziemann has found a spirochæte along with trypanosomes in the blood of sheep in the Cameroons.

Susceptible sheep show, after subcutaneous inoculation of infective defibrinated blood of the horse, a rise of temperature on the third to fifth day, and which continues intermittently for several days longer. During these rises the spirochætes are found in the blood.

V. SPIROCHÆTOSIS IN THE PIG.

In 1906, Sydney Dodd discovered in several young pigs in the Transvaal a slender spirochæte varying from 9 μ to 26 μ in length, but on an average from 14 μ to 16 μ in length. The extremities were pointed. Spiral forms predominated, but in the same preparation the organism may be seen curved in a semicircle without showing spirals, simply being like long straight threads. The spirals vary from two to six in number. The spirochætes were usually found singly, or in twos or threes ; sometimes in large clusters, containing twenty or more individuals.

They were not easy to distinguish in unstained specimens, owing to slenderness and motility. Although staining with any of the ordinary stains, Romanowsky's method, or any of its modifications, gives very good results. Giemsa's method is the best. They are decolorised by the method of Gram or of Claudius.

The organism was only found in the cutaneous lesions, but never in the blood, which, when injected into healthy pigs, did not reproduce the disease. The spirochætes, although found in the lesions during life, disappeared from them soon after death. The disease was transmitted by cohabitation even to those pigs that had previously resisted blood inoculations. Inoculations made with scrapings of the cutaneous lesions gave positive results in one out of two pigs. The spirochætes disappeared from the lesions undergoing cicatrisation.

Symptoms.—The most noticeable symptom was several superficial wounds on the skin, which increased in number until the whole body was covered with circumscribed superficial ulcers, averaging about $\frac{3}{4}$ to 1 inch in diameter. The edges of the lesions had no tendency to spread. The disease spread to in-contact pigs, and spirochætes were found in the lesions. The ulcers commenced to cicatrise after varying durations. The beginning of this cicatrisation was evidenced by desquamation of the epithelium, accompanied by blood extravasation, which, on desiccation, gave rise to a brownish scab. The scab and bristles after a time shed, leaving a glistening cicatrix. In those regions where the lesions had been very numerous, the skin became quite smooth and tense. But although the cicatrisation became complete after various periods, the pigs, instead of regaining their former condition, continued gradually to become emaciated, until before death they were almost skeletons. Out of fourteen pigs only two recovered, although one of these still remained very thin and anæmic.

Lesions.—The constant lesion was the anæmic condition of the blood. There were in several instances broncho-pneumonic lesions. In other instances the stomach or intestines were congested.

The question is, Is this disease due to spirochætes? These parasites have been found in various lesions in the pig by Cleland, Gilruth, and others. Cleland found them in "castration tumours" of the pig in Western Australia. Gilruth encountered them in ulcerating tumours on the cheek or knee in pigs in Victoria; also in submucous cysts in the large intestine. In this latter case the two pigs had a fluctuating temperature. In one instance the mucosa of the cæcum was affected for about an area of 8 inches with patches of inflammation, covered by diphtheritic false membrane. The large intestine along its whole length showed numerous circular, flattened, greyish nodules, each about the size of a small pea, there being from two to four present to the square inch. The disease was transmissible by cohabitation. Apparently the spirochætes were present accidentally.

Dodd's description of the disease, studied by him in Pretoria, somewhat clinically agrees with that described as "pig variola" by Szanté in Hungary and Poenaru in Bucharest.

VI. SPIROCHÆTOSIS IN THE HORSE.

The *S. Theileri*, or, according to Novy and Knapp, *equi*, was first discovered by Theiler in the horse in South Africa. Gustave Martin encountered spirochætes in a horse in Timbo, in French Guinea. The organism was found in the blood of a horse, showing all the clinical symptoms of the trypanosomiasis of that region. They were very numerous, and joined together around the red blood-corpuscles, to which they communicated a kind of Brownian movement. They were, although not unrolled, 12 μ to 15 μ in length, by 0·25 μ in breadth. Certain forms were undergoing transverse division, and had five or six instead of three or four spirals, the central part being thinner and paler. Inoculation of the blood of the horse to the fowl did not transmit the disease; to the sheep, during the first month, there were febrile attacks, and its blood showed trypanosomes, but no spirochæta. Dodd found the spirochætes in the blood of newly imported horses of Argentine inoculated with piroplasma in the Transvaal. The defibrinated blood of such horses, when injected into the ox and sheep, gave intermittent febrile attacks and the spirochætes, but the reverse has not yet been observed. Dodd believes the equine organism is identical with the spirochæta of the ox and sheep; but Theiler is of opinion it is quite different from the latter.

The zebra also harbours a spirochæte.

Stordy observed in 1906 a case of spirochætosis in an Abyssinian pony in Nairobi, East African Protectorate. The animal was dull, with hanging head, large swellings over the orbits, and acute œdema of the neck. The temperature was 97° F., that in the evening 99° F. By the following morning the œdema of the supra-orbital and neck regions had disappeared, but the œdema had accumulated between the forelegs, extending backwards towards the sheath; the animal was extremely dull and weak, eating and drinking but little. The temperature had fallen to 95° F., rising again in the evening to 98° F. By the third day rapid emaciation had taken place, the œdema between the legs and under surface of the abdomen remained persistent, and the temperature rose to 99° F. On the fourth day the temperature fell to 95° F., rising again to 98° F.; the animal would only occasionally nibble at its food, and stood resting its head on the manger through sheer weakness. On the fifth day the temperature was 97° F.; the animal was so weak that it propped itself up against the side of its stall. Emaciation was still more advanced, and the weakness gradually got profounder, and death occurred at 10 p.m. Shortly before the rectal temperature was close on 101° F.

Examination of the blood on the second and third days gave negative results. On the fourth day smears from the blood and œdematous fluid, stained with Leishman's stain, showed ten or twelve spirochætes in each slide of the peripheral blood, and a few in the œdematous material. The blood injected intravenously into a dog gave negative results.

Lesions.—The carcass was extremely emaciated; rigor mortis was very slight; the muscular tissue had a washed appearance; the œdematous swelling contained a gelatinous straw-coloured exudation; a large spleen-shaped clot of blood was found lying on the surface of the right lung; underlying this clot the pulmonary pleura was ruptured, and a huge infarction occupied almost the whole lobe; the left lung appeared normal; the stomach contained several dark-coloured blood-clots, but otherwise had the appearance of being healthy; no lesions in the spleen, liver, or bowels; the kidneys were enormously enlarged, pale in colour, and each weighed 56 ounces; the bladder was full of pale urine. No parasites could be found in any of the organs.

A number of ticks, *Rhipicephalus pulchellus,* were found under the anus and between the hind-legs. This variety is commonly found in cattle, horses, dogs, and game in the district from which this animal came. It is also a great source of annoyance to human beings. Also, around the anus, a few of another variety of tick, *Rhipicephalus evertsi,* were attached; it is a very common species in the Protectorate.

Jowett, in 1905, found in canker and grease of the horse a spirochæte which varies from 7 to 20 μ or more in length by ¼ μ or even less in breadth. Each organism has the same thickness throughout, but both ends taper. The number of spirals vary from five to ten, and occasionally more or less, and in some cases none whatever, the organism appearing straight. Tangles are common, and movement persists in wet films. Three forms of these spirilliform organisms are observed—a thin and a thick without flagella, and a thick showing flagella. According to Schaudinn, to whom the specimens were submitted, Jowett's spirochæte resembles the spirochæte found in ulcerous processes in man, especially in ulcerous carcinomata. It is well known that in the venereal granulomata of the bitch, the carcinomata of the mouse, and granular, ulcerous, or chronic suppurating wounds in other animals are also infected with spirochætes; but there is no reliable evidence to indicate what part they play in these chronic processes. Jowett has, however, never failed to find spirochætes in any case of equine canker and grease that he has examined.

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BRAXY

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Synonyms.—Morbus subitorius ovis; Bradsot; Brasot; Gastro-mycosis-ovis; Redwater; Whitewater; Sickness.

Definition.—Braxy is an acute infectious disease of the ovine species due to a specific bacillus, and characterised by a very short period of visible illness, a marked regional and seasonal distribution, and by being almost invariably fatal. It is really an infective gastro-enteritis.

Historical.—The disease was recognised as occurring in Scotland by Dr. Duncan,* and later by Stevenson and W. Hogg. Krabbe identified it in 1872. In 1882 the disease was considered to be a form of anthrax by Williams in his "Principles and Practice of Veterinary Medicine." In 1893 Steele concurred with this view in his "Diseases of the Sheep."

The first scientific account of the disease was written by Ivor Nielsen of the Norwegian Government, who defined braxy as a gastro-mycosis due to a specific organism introduced with the food into the abomasum. Later, Jensen and Braland published an account of investigations carried out by them.

The late Professor Hamilton, acting as pathologist to the Departmental Committee appointed by the Board of Agriculture to inquire into the disease, carried out an exhaustive investigation. The Blue Book published in 1906 contains an excellent report of his work.

In Ireland, Mason, acting for the D.A.T.I., has continued the work of Professor Hamilton. In conjunction with the author, he successively employed a preventive vaccine in five counties.

General Remarks.—The condition known as "braxy" is frequently confused with other diseases of the sheep. This fact requires no explanation when one considers the small amount of attention hitherto bestowed on ovine disorders. In this article the term "braxy" is reserved for a certain well-defined disease due to a specific bacillus, and this description is based upon the results of our own experience in Ireland. The period of visible illness is so short that it is usually necessary to rely on a post-mortem examination for diagnosis. In some parts of Scotland "braxy" mutton is used for human food, and regarded as a great delicacy.

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* Transactions of the Highland Agricultural Society, 1807, vol. iii.

Etiology and Bacteriology.—The disease is due to a specific organism—the *bacillus of braxy*. This organism was first described by Ivor Nielsen, occurring in small hæmorrhagic areas in the mucous membrane of the abomasum, and later by Hamilton in the peritoneal fluid taken from sheep dead of the disease. It is a facultative parasite occurring in slender rod-shaped forms, varying in length from 3 to 5 μ, and usually less than 1 μ in breadth. It is frequently prolonged into chains, consisting of from eight to ten individuals. The most constant seat of the braxy bacillus is the intestinal mucous membrane. It exists in almost pure culture in the peritoneal fluid taken from affected animals. Its presence has been demonstrated in the liver, kidney, and even in the blood after death. The organism sporulates freely when the peritoneal fluid is incubated for ten to twelve hours at 37° C. The spore is a highly refractile oval body, usually situated towards one end of the bacillary body. The organism is non-motile, strictly anaërobic, and shows rounded ends by all methods of staining. It stains instantly on being placed in contact with a 1 per cent. solution of any of the common basic aniline dyes; it is Gram-negative. The organism grows readily on all the common laboratory media under anaërobic conditions. The most characteristic growth occurs on glucose gelatine after incubation for about one week at 20° C. Small cup-shaped areas of liquefaction appear evenly distributed through the medium, and from the upper concave surface of these a series of wavy arms are thrown upwards.

The optimum temperature for growth is 37° C., but quite abundant growth can be obtained at ordinary room temperature. The organism grows more luxuriantly in glucose broth than in ordinary broth, and still more luxuriantly on the addition of 5 per cent. diluted alkaline serum sterilised under high pressure. This is the medium employed by us in obtaining large quantities of dense growth for the preparation of preventive vaccine.

Seasonal Distribution.—In affected areas braxy is prevalent during the months of October to January inclusive. Its appearance usually coincides with the first white frost. It never occurs at any other time throughout the year. During the non-susceptible period of the year the sheep's blood is highly bactericidal for the braxy bacillus, but from October to January this property is lost.

Regional Distribution.—The disease has been identified as occurring in the United Kingdom, Norway, Sweden, Denmark, Iceland, Faroe Islands, etc. In Ireland the disease exists to a large extent in the Counties of Wicklow, Antrim, Tipperary, Kerry, and Donegal. It is usually confined to old grazing pastures in hilly districts. These pastures soon acquire a reputation for being dangerous. It is a strange fact that in an affected area clean and foul pastures often lie side by side, separated only by a fence. Those pastures situated on the eastern aspect of a hillside are much more dangerous than those on the western aspect.

Influence of Age, Sex, Condition.—Weaned lambs are very much more susceptible than older sheep. It is rare for braxy to occur in animals over two years of age, or in sucking lambs. Males and females are equally susceptible. During a severe cold season the disease is more prevalent than in a mild season. The influence of frost is well known, and was at one time held to be the actual cause of braxy. It was thought the animals died from tympanites set up by the effect of frozen food causing paralysis of the rumen.

Sheep in good condition are more liable to become the subject of braxy than those in poor condition. Plethora must be regarded as a predisposing cause, probably acting as it does in the case of black-quarter.

Animals Affected.—In nature braxy is a disease confined to sheep. No case has yet been recorded as occurring in any other animal. All breeds of sheep appear equally susceptible, but the disease principally affects Cheviots, blackfaces, and the smaller breeds. Jensen succeeded in experimentally transmitting the disease to the calf, guinea-pig, rabbit, mouse, and pigeon.

Methods of Infection.—There can be no doubt that ingestion is the common natural method of infection. Sheep grazing on foul pastures swallow the organism with their food. During the non-susceptible season, however, the animals possess a high degree of natural resistance, and can resist infection. It has not yet been explained why the resistance should be lost during a certain season of the year, and the animal fall an easy prey to the disease. Experimentally braxy can be set up by the administration *per os* of a small quantity (6 c.c.) of young living culture of the causal organism during the susceptible period. A dose of 25 c.c. administered during the non-susceptible period proved harmless. The disease can be experimentally induced *at any time* by the subcutaneous injection of 1 c.c. of young living culture. In about eighteen hours the animal becomes lame on the infected limb, its temperature is raised to 106° or 107° F., and it shows great depression. Death occurs within thirty-six to forty-eight hours after inoculation. On post-mortem the usual lesions of braxy are noticed, together with great swelling and extravasation into the affected limb. The causal organism can be recovered and subcultivated from the peritoneal transudate.

A larger dose of culture is required to set up the disease in hoggets than in lambs, or if the injection is not performed in the susceptible period. A much smaller dose is sufficient to induce symptoms if a small quantity of acetic or formic acid is injected side by side with the living virulent material. Prolonged cultivation of the organism renders it almost avirulent. A growth subcultivated six times, and then allowed to remain at room temperature for one month, was used to inoculate a hogget. No symptoms were observed after subcutaneous injection of 10 c.c.

Period of Incubation.—The period of incubation has never been exactly determined. Experimentally, after subcutaneous inoculation, it has been found to be about forty-eight hours. After administration *per os* it was about four days. Probably in nature the period of incubation is considerably longer than four days.

Mortality.—In Ireland the disease is almost invariably fatal; cases of recovery are extremely rare. In affected districts from 10 to 50 per cent. of the total flocks become affected. As in louping-ill, sheep moved from clean to foul pastures are especially liable to attack.

Robertson states that 25 per cent. of the total flocks in the west Highlands become affected. S. Jonsson states that in 1870-71, 3 per cent. of the total flocks in Iceland died from the disease.

Prognosis.—Always very unfavourable.

Symptoms.—The disease is not infectious from animal to animal, but is always contracted from grazing on foul pastures.

It is very seldom that one has the opportunity of studying the symptoms of braxy from the onset of the disease to its termination. The period of visible illness rarely exceeds five or six hours, and usually the animal is found dead without any signs of illness being noticed. It is held by shepherds that a trained sheep-dog will follow an animal about to die of braxy, and by this method they can pick out suspicious cases.

The affected animal shows loss of appetite, dulness, and weariness in movement. Later the animal walks quickly with a short step, and may fall as if dazed. The temperature is raised to 107° F., the breathing is difficult, and the pulse is small and increased in frequency. Diarrhœa is usually present, and occasionally violent colic. Rarely sheep live thirty-six to forty-eight hours after the appearance of symptoms. The characteristic braxy odour becomes noticeable just before death.

Morbid Anatomy.—The onset of putrefaction is extremely rapid. Within four hours after death the carcass is completely emphysematous, air being distributed through the subcutaneous connective tissue, and blood-stained froth issues from the natural orifices. Later the skin over the abdomen takes on a green colour, the skin covering the remainder of the body being pink. The wool is easily pulled from the carcass, especially in cases occurring towards the end of the season. The odour given off from the carcass is absolutely characteristic of the disease. It bears no resemblance to the odour of putrefaction. The dogs in the neighbourhood are speedily attracted by it to the body, which they at once devour. The subcutaneous veins are congested with dark uncoagulated blood. The most uniform abnormality is the presence of a quantity (8 to 12 ounces) of peritoneal transudate. The fluid is always opaque, sometimes blood-stained, sometimes whitish in colour, hence the local names of "redwater" and "whitewater." It constitutes an almost pure culture of the causal organism. The lining membrane of the peritoneal cavity is more or less deeply congested, but never in a state of actual inflammation. The spleen is slightly enlarged, very soft; frequently the spleen pulp is almost liquid in consistence, and darker in colour than the normal. The liver is of a lighter colour than normal and very friable. It is in a state of cloudy swelling. The kidneys are much softer than normal, and contain brownish-red areas of necrosis.

The rumen, reticulum and omasum are quite healthy and filled with food. The abomasum appears deeply congested, even when viewed from the external aspect. When its wall is slit open this division of the stomach is empty of food, but contains a quantity of brownish mucus. The mucous membrane is thrown into deep folds, and is in a state of inflammation, with small hæmorrhagic areas dotted over it. Frequently the epithelium peels off in flakes.

If the heart is punctured, a whiff of gas escapes. When the carcass is not discovered for about twenty-four hours after death, the gas present may have burst through the skin.

(Written from accounts of 100 consecutive post-mortems performed for the D.A.T.I. sheep disease investigations.)

Differential Diagnosis—(*a*) *Black-Quarter.*—Braxy can be differentiated from black-quarter by the absence of a distinct muscular swelling, the difference of the characteristic odour of both diseases, and by an examination of the peritoneal fluid.

(*b*) *Anthrax.*—In a case in which anthrax is suspected, a microscopic examination of the blood after death will reveal the presence of the anthrax rods in large numbers.

(*c*) *Malignant Œdema.*—It is doubtful whether malignant œdema occurs in the sheep. It is certainly distinctly rare. In such cases a traumatic origin would suggest itself, and the presence of an œdematous swelling in the neighbourhood of a wound, taken with the absence of the characteristic odour and peritoneal fluid of braxy, would justify a diagnosis of malignant œdema. We have not observed such cases.

(*d*) *Louping-Ill.*—It is unnecessary to mention any differentiating points between this disease and braxy. They do not resemble each other in any way.

Curative Treatment.—Treatment is universally regarded as quite useless. Many shepherds believe prompt bleeding from the facial vein to be efficacious, and the method is largely practised. Others administer large doses of magnesium sulphate and other such remedies.

Prophylactic Treatment.—The internal administration of such drugs as carbolic acid, sulphate of copper, lysol, etc., about six weeks before the susceptible period, was believed by many to be protective. Sulphate of copper, administered in appropriate doses, is largely used as a preventive measure in the County of Antrim. Needless to say, we have proved this method as of no value. Many farmers adopt the plan of moving their sheep to lowland pastures during the susceptible period, and can prevent the disease occurring to any extent by this means.

Another improved measure largely used in Scotland as a preventive against braxy is as follows:

An aged pig is put to grass on foul pasture for three days, and at the same time given as much skimmed milk as it will consume. The fæces from the pig are gathered as soon as voided. One pint of dung is mixed with 15 pints of milk, and the mixture strained through muslin.

The sheep to be treated are allowed to fast for twelve hours, and one wineglassful of the mixture administered to each animal about the third week in September. According to Hamilton, the method gives good results. The pig swallows a quantity of the braxy organism from the foul pasture. These multiply in the intestines, are passed out and collected with the fæces. The sheep, therefore, really receive a certain dose of living organism *per os,* and this acts as a preventive vaccine. Nielsen treated the kidneys taken from animals dead of braxy in the same manner as black-quarter muscle is used to make Arloing's black-quarter vaccine. He obtained good results from the vaccine made in this way. The subcutaneous administration of small doses (0·5 c.c.)

of living virulent material was carried out by the Department of Agriculture in Ireland as a preventive method. The vaccination was performed during the first week in September. The method was abandoned in favour of administration of larger doses (4 c.c.) *per os* during the same week, followed a week later by the same dose of a second vaccine.

The method of preparation of the vaccine carried out at the Department's laboratory under the supervision of the author is as follows: The peritoneal fluid is collected from an animal just dead of braxy in sterile pipettes, one end of the pipette being sealed off in the flame, and the other capped with gutta-percha. The pipettes are then incubated for twelve hours at 37° C. Flasks containing glucose broth covered with olive oil are sterilised by heat. The contents of a pipette are carefully added to the broth under oil, and the whole raised to 80° C. for twenty minutes in order to kill any accidental non-sporing organism. After incubation for twenty-four hours abundant dense clumps of light greyish growth settle to the bottom of the flasks, and this, when proved to consist of a pure culture of non-sporing organisms, constitutes our first vaccine. A similar culture incubated for thirty-six hours constitutes our second vaccine.

The process is carried out with four lots of peritoneal fluid collected from cases of braxy occurring in different parts of the affected area, and the vaccines so obtained are mixed together before use. These vaccines must not be used later than September 21, or some of the vaccinated sheep will die as a result.

This method of prevention yields excellent results. By its use the mortality from braxy never exceeds 1 per cent. even in the most severely affected districts.

LOUPING-ILL

By B. HARVEY MELLON, M.R.C.V.S.
Late Bacteriologist to the Agricultural Branch of the Department of Agriculture and Technical Instruction for Ireland.

Synonyms.—Chorea paralytica ovis; Meningo-myelitis; Twitch; Trembling; Staggers; Dizzy; Shivers.

Definition.—An infectious neuritis of the ovine species due to the *Bacillus choreæ paralyticæ ovis* (Hamilton), and characterised by clonic spasms of groups of muscles, terminating in partial or even complete paralysis. It has a well-marked regional and seasonal distribution.

History.—Louping-ill appears to have escaped serious attention on the part of scientific investigators. The late Professor Hamilton of Aberdeen, acting for the Departmental Committee appointed by the Board of Agriculture, is the only pathologist who really devoted much time to the study of the disease. The Blue Book published in 1906 contains a valuable record of his work.

In Ireland, Mason has carried on a scientific investigation of louping-ill and braxy since 1905. He has successfully employed Professor Hamilton's preventive vaccines in five counties. At present these vaccines are prepared at the Department laboratories, and are being extensively used throughout the country.

General Remarks.—This disease has long been confused with other conditions, and, owing to the fact that veterinary surgeons are seldom called upon to treat diseases of the sheep, a great amount of ignorance prevails.

Louping-ill cannot be regarded as infectious from animal to animal; the disease is usually contracted from feeding on contaminated pastures.

Animals Affected.—Louping-ill may be regarded as a special disease of the sheep. The writer's experience of louping-ill is limited to the Cheviot, Roscommon, and blackface breeds; but all breeds of sheep appear equally susceptible.

Cases have been recorded occurring in calves, pigs, and geese. Mason informs us of a calf in which he observed the characteristic symptoms of the disease at Kippore, Co. Wicklow, in 1905. The peritoneal fluid was collected after death, and forwarded to Professor Hamilton, who pronounced it to contain an organism indistinguishable from the causal organism of louping-ill.

Regional Distribution.—The disease is located in certain well-defined areas throughout the British Isles. In Ireland the Counties of Antrim, Wicklow, Donegal, Kerry, South Tipperary, and Waterford are affected. No cases have been recorded as occurring outside these islands.

Seasonal Distribution.—In affected areas the disease makes its appearance with persistent regularity during the months of February, March, and April. Cases occur during May and June, but are not nearly so numerous. Sporadic cases are observed throughout the year.

Etiology.—Louping-ill is due to a specific organism—the *B. choreæ paralyticæ ovis* (Hamilton). The disease cannot be regarded as septicæmic, as it is impossible to demonstrate the bacillus in the blood of affected animals. Inoculation of blood taken from a sheep suffering from louping-ill does not set up the disease.

Bacteriology.—The principal habitat of the organism is the intestinal mucous membrane. It exists in the opaque peritoneal fluid taken from acute cases as a pure culture. In the so-called chronic cases the peritoneal fluid is usually translucent. It is often necessary to incubate the fluid for a few hours at 37° C. in order to show that the organisms are present to any extent. The organism has not yet been discovered in the cerebro-spinal fluid or in any other tissue except those already referred to.

The bacillus of louping-ill is a facultative parasite occurring in coarse rod-shaped forms, often prolonged into chains, consisting of fifteen to twenty individuals. It varies in length from 4 to 7 μ, and is about 1·3 μ in breadth. The ends appear rounded by all methods of staining. If the peritoneal fluid collected from a case of louping-ill is incubated for ten to twelve hours, the organism sporulates freely. The spore usually occupies a central position, but if situated at one end, it imparts an appearance to the bacillus which renders it liable to be mistaken for the tetanus bacillus. The organism possesses only feeble powers of motility except in comparatively young cultures. It stains almost instantaneously when placed in contact with a 1 per cent. solution of any of the basic aniline dyes. We consider methylene blue gives the most satisfactory results. Good results are obtained also when the methods of Gram or Claudius are employed.

The organism grows readily on all the common laboratory media under anaërobic conditions. The optimum temperature for growth is 37° C., but good growth can be obtained at ordinary room temperature. A considerable amount of gas is evolved during cultivation, giving off a faint odour of putrefaction.

The most convenient method of obtaining large quantities of growth, and the one employed by us in the preparation of our preventive vaccine, is as follows: The peritoneal fluid is collected in sterile pipettes from an animal within two hours of death, one end of the pipette being sealed off in the flame and the other end capped with gutta-percha. The pipette is then incubated for twelve hours at 37° C. Flasks containing glucose broth covered with oil are sterilised by heat. The contents of a pipette are carefully added to the broth under oil, and the whole raised to 80° C. for twenty minutes in order to destroy any accidental non-sporing organism. After incubation for twenty-four hours an abundant greyish growth is observed at the bottom of the flask, and this, when proved to consist of a pure culture of non-sporulating organisms, constitutes our first vaccine. A similar culture incubated for thirty-six hours constitutes the second vaccine employed.

Age Incidence.—In our experience sheep of all ages and classes are equally liable to infection, but some authors state that sheep during their first year of life are more susceptible.

Influence of Sex.—Animals of both sexes are equally susceptible.

Influence of Pastures.—The disease exists principally on old grazing-land in hilly districts. A large part of such land is untillable owing to its rocky nature, and soon acquires a reputation as a breeding-ground for the disease. It is a strange fact that a clean farm, known to be quite safe for grazing sheep, may be wedged in between two foul pastures.

Influence of Parturition.—The act of parturition is often succeeded by an attack of louping-ill of the acute toxæmic type. Parturition reduces the natural powers of resistance, and must be regarded as a predisposing cause.

Period of Incubation.—It is impossible to state definitely the exact period of incubation. A large dose of living virulent culture of the causal organism was administered to a sheep in a susceptible season of the year, and four days elapsed before the onset of symptoms was observed. It is possible that the period of incubation is considerably longer under natural circumstances.

Methods of Incubation.—There can be no doubt that ingestion is the common natural method of infection. The disease is essentially an intestinal one, and affected animals pass out enormous numbers of organisms with the fæces, thus infecting the pastures. Sheep grazing on these pastures during a susceptible season are liable to contract the disease. It has not yet been satisfactorily explained why sheep enjoy a high degree of immunity for seven months of the year, during which time the causal organism is freely ingested with the fodder without serious results. During the susceptible period the organism can pass into the peritoneal cavity, and it is only then that symptoms appear to ensue. Muzzling experiments carried out by Professor Williams and repeated by Professor Hamilton prove that ingestion is the common natural method of infection. It has been asserted by some authors that ticks play an important part in the transmission of louping-ill. In our opinion it was abundantly proved by Professor Hamilton that ticks do not commonly act as carriers. We have frequently observed louping-ill where ticks are unknown.

Microscopical examination of ticks taken from infected sheep always fails to reveal the presence of the organism. Subcutaneous injection of mashed-up bodies of ticks collected from diseased animals usually fails to transmit the disease.

Louping-ill has been experimentally induced by subcutaneous inoculation, into the thigh, of living virulent culture, or peritoneal fluid collected from a case of the disease. In about twenty-four hours great swelling and œdema of the infected limb take place, with hæmorrhage into the muscular tissue and transudation into the peritoneal cavity. Death ensues within thirty-six hours, and the organism can be subcultivated from the peritoneal fluid. The subcutaneous injection of a small quantity of acetic acid and living virulent culture causes death in from twelve to fourteen hours.

The subcutaneous injection of cerebro-spinal fluid, beaten up cerebrum, liver substance, or blood, fails to set up the disease.

The administration *per os* of a small quantity (4 c.c.) of living virulent culture to a sheep during the susceptible period invariably causes a fatal attack of louping-ill, whereas the same dose can be safely administered during the non-susceptible season.

Mortality.—In a louping-ill district from 10 to 50 per cent. of the total flocks become affected. The mortality amongst affected animals is about 95 per cent. The effect of moving sheep from clean to foul pastures is very disastrous, and may lead to the loss of the entire flock.

Morbid Anatomy.—The lesions of louping-ill vary considerably, depending on whether the animal has died from the acute toxæmic or the chronic form. Some authors describe cerebro-spinal alterations, such as hyperæmia and thickening of the meninges, with considerably increased amount of cerebro-spinal fluid, especially in the region of the choroid plexus. The spinal cord is said to have been softer than normal, and to contain pin-point hæmorrhages. We have never observed such lesions during a post-mortem study of 120 cases of the disease.

ACUTE TOXÆMIC TYPE.—Putrefaction sets in shortly after death, blood-stained fluid may issue from the natural orifices, and tympanites is well marked. The skin does not alter in appearance until at least twenty-four hours after death. The wool is not easily pulled off. The odour is not characteristic, and strongly resembles that of ordinary putrefaction. The peritoneal cavity contains an opaque greyish fluid, varying from 6 to 12 ounces in volume, and swarming with the causal organism. The peritoneum is congested, sometimes deeply congested, but never inflamed. The remaining organs and tissues of the body appear quite normal.

CHRONIC NERVOUS TYPE.—In animals dead of this type of louping-ill it is impossible to discover a single abnormality by the naked eye, except for the presence of a peritoneal transudate. The fluid is quite clear and straw-coloured ; it contains the causal organism of the disease. Death is apparently due to a bacterial toxin acting similarly to the tetanus toxin.

The toxin of louping-ill differs from that of tetanus in that it is an endotoxin. If the liquid obtained after a culture has been passed through a filter is injected subcutaneously into a sheep, no symptoms are set up.

Symptoms.—Two distinct types of the disease are met with :

1. ACUTE TOXÆMIC TYPE.—The first symptom usually observed is that the animal jumps wildly into the air, and shows a considerable amount of hyperæsthesia. It then stands quietly, whilst its muscles tremble violently. The temperature is raised to 105° or 106° F., breathing is laboured, and the pulse is small and hard, and the appetite lost.

The animal walks with staggering gait, and steps too high, appearing to be blind. Later, symptoms of ataxia appear, it becomes seized with a convulsion, falls to the ground, the muscles in a state of clonic spasm, the head extended, and the limbs projecting outwards. These convulsive spasms recur at rapidly lessening intervals during the next few hours, until paralysis of the hind-quarters supervenes.

The animal then lies continuously on one side in a helpless condition, paralysis soon extends forwards, and death occurs within twenty-four hours. The entire period of illness varies from forty-eight to seventy-two hours, during which the animal appears to be under the influence of a powerful toxin.

2. Chronic Nervous Type.—This form is much more common than the preceding in this country. The first symptom observed is that the sheep carries its fore-limbs too high, and the head is extended or carried to one side, usually the left side. The pupils are dilated, and the animal appears very nervous. Attempts at grazing are made, but in reality the animal eats little. Later, inco-ordination of the movements of the hind-limbs is noticed; the animal may even stagger and fall. Convulsive fits never occur, but trembling of groups of muscles, especially of the head and neck, may take place. Occasionally paresis of the hind-parts supervenes, but usually death takes place from exhaustion and starvation. An animal may be kept alive for two months by hand-feeding. Death usually occurs in from two to four weeks. The temperature remains normal during the course of the attack.

Sequelæ.—Recovered animals usually exhibit some nervo-muscular trouble, such as torticollis, chorea, etc.

Differential Diagnosis—*Gid or Sturdy.*—This condition is due to the presence of *Cœnurus cerebralis* in some part of the cranial cavity, so that the resulting symptoms will depend upon the portion of brain substance affected—*i.e.*, if the parasite is lodged on the right cerebral hemisphere the sheep tends to gyrate to the left in a circle, whilst if the parasite is lodged in the cerebellum, we see loss of equilibrium. Louping-ill is easily differentiated, therefore, from gid, as it would in that case be quite impossible to select any portion of the brain as the *locale* of a morbid centre.

Tetanus.—In the case of tetanus a traumatic origin generally suggests itself, the disease usually occurring after such operations as castration and docking. The spasms are tonic in type, as opposed to the clonic spasms of louping-ill.

Braxy.—It appears unnecessary to describe any differentiating points. The disease has no relationship to louping-ill.

Kidney Staggers.—The disease has been identified by the author as a slowly progressive sclerosis of the lumbar portion of the cord, giving rise to partial paralysis of the hind-parts. The animal may live on indefinitely, and the hind-parts only are affected.

Treatment.—Curative treatment is quite useless.

Preventive Treatment.—Various drugs have been used as preventives before the onset of the susceptible period. Carbolic acid, lysol, etc., in appropriate doses have been tried, but are now generally known to be useless. The subcutaneous injection of small doses (2 c.c.) of living virulent culture (method of preparation, see p. 1253) during the month of January has been attempted on a large scale by the D.A.T.I., but have now been discarded in favour of the administration *per os*. The dose of first vaccine employed is 4 c.c., added to 4 ounces of water. The second vaccine is administered one week later. On no account must vaccination be carried out after January 31, as fatalities are bound to follow if this precaution be neglected. We have now adopted a method by which the vaccine employed contains four strains of the louping-ill bacillus. The results obtained by this method of prevention are excellent. We have never observed a higher mortality than 0·5 per cent. of vaccinated sheep in the most badly affected louping-ill districts.

HEARTWATER

BY J. SPREULL, F.R.C.V.S.,

Government Veterinary Inspector, Natal.

Definition.—A specific febrile disease affecting sheep, goats, and cattle in South Africa, and due to an ultravisible virus transmitted by the Bont tick—*Amblyomma hebræum* (Koch).

This disease received its name from its most prominent post-mortem lesion—namely, a more or less marked effusion of yellow plasma into the pericardial sac, this lesion being fairly constant.

History and Distribution.—Heartwater used to be a veritable scourge in many districts of the Cape Eastern Province, rendering sheep and goat farming almost impossible; but within the past ten years the systematic dipping of cattle (the chief hosts for all species of ticks) has made an end of the disease, for the progressive farmer at least. At one time whole districts were in the grip of the disease, and many farmers were ruined by it; to-day, owing to the advent of dipping, these districts are again full of small stock, and heartwater is no longer dreaded. This disease is also common in the bush-veld of the Transvaal, in some parts of Zululand, Natal, and the Transkeian territories. It probably spread southwards from Zululand along the coastal belt, invading only such areas as were congenial to the bont tick, by which alone the disease is transmitted in Nature.

The first bont tick found in Lower Albany was seen on a cow which came from Zululand in 1840, and gradually, as the species became numerous (about 1860), small stock did badly; but it was a good few years after this date before the connection between the disease and the tick was even suspected.

Species Affected.—Heartwater is very fatal indeed to small stock, especially when young, but its relative susceptibility varies in the different breeds. The Angora goat is undoubtedly its greatest victim; next comes the ordinary Boer goat, then the merino sheep and its crosses, and the Afrikander. Imported sheep of numerous breeds have also been known to succumb, but the Persian sheep, though susceptible, almost invariably escapes with a febrile reaction only. Its crosses show a varying susceptibility, their resistance weakening in direct proportion as the merino blood predominates. Cattle are less susceptible than sheep. Amongst wild antelopes the springbuck is known to be occasionally affected. The horse is insusceptible even when inoculated with large quantities of virulent blood.

ETIOLOGY.

This disease has been the subject of much study and experiment. Amongst others, Dr. Edington, Lounsbury, Dr. Theiler, Dixon, Robertson, and the writer, have made investigations into its nature. Edington was the first to convey heartwater to cattle by inoculation. Lounsbury transmitted it by ticks, first to sheep and goats in 1899 and to cattle in 1902, and his brilliant work in this direction has subsequently been amply confirmed. He showed that the tick is normally non-infective, but that when it fed on a diseased animal it picked up the infection, carried it through its moulting period, and transmitted it to its next host, if a susceptible one. The infection does not pass through the egg, as is the case in redwater. If a larva pick up the infection, it transmits it as a nymph; if a nymph pick it up, it will pass it on as an adult. More strangely still, Lounsbury proved that when a larva picked up the infection and fed on an insusceptible animal as a nymph it was still capable of infecting its next host when it fed as an adult tick (in this respect differing from the infection of East Coast fever). Before going farther, then, it will be necessary to consider the characters of the bont tick, also called the "tortoise-shell tick." The word "bont" comes from the Dutch, and is nearly equivalent to the English word "variegated." The male is very handsome, ovate in shape, broadest posteriorly, almost flat, and about $\frac{1}{5}$ inch long. The so-called "shield" covers his back. It is hard and shiny, and shows numerous mosaic-like patches of grey, green, black and gold tints. The legs are brown, with paler bands at the joints. The rostrum is very strong, and holds so firmly to the skin that when the tick is forcibly pulled off it generally removes a piece of skin with it. The males do not swell out with blood after feeding.

The adult female is dark green or very dark brown in colour, the shield covering the front part only of its back, and showing gay colours, which readily identify it with its mate. When gorged, she becomes as large as one's thumb-nail, red-brown in colour, with a bright red band round the posterior margin.

The adult tick attacks all classes of farm stock, including ostriches, wild antelopes, dogs, and even man himself. It is generally found on parts bare of hair or wool, and about the genitals, udder, and teats. In small stock it often causes lameness by attaching itself between the toes. The larval and nymphal forms, which have none of the adult's gay colours, and look to the naked eye like those of other species, attach themselves anywhere upon their hosts.

When fully fecundated and gorged, the female bont tick falls to the ground, and in a few weeks' time (two to twelve weeks, depending upon the state of the weather) begins to lay her eggs. Egg-laying lasts from three to nine weeks. The eggs are glistening, nearly transparent, reddish-brown bodies, to the number of several thousands (10,000 to 20,000), adhering to one another by some sticky substance. In summer they may hatch in three months, but in cold weather it may take twice as long; the average is from four to six months. The larva is six-legged, and $\frac{1}{40}$ inch long; it waits on the grass tops till a host brushes it off; then, becoming attached, feeds itself to repletion, in six to nine days falling off to moult. It is now $\frac{1}{12}$ inch long. It hides in the ground, and in one to three months or more goes through the first moult, and emerges as a nymph. The latter is eight-legged. On finding a host, it gorges itself, in four to eight days becoming plump and round, bluish-grey in colour, and $\frac{1}{4}$ inch long. It now falls to earth, and in four to twelve weeks, or longer, goes through the second moult, from which emerge distinct male and female adults. These again seek a host, where pairing takes place, both sexes being attached to the skin the while. The female seeks out the male only after he has fed for several days; her sojourn on her host lasts ten to twenty days. After copulation, she fills out and falls off to lay her eggs; but the male may remain on his host for many weeks, and probably mates again.

The life-cycle of this tick, therefore, will take nine months for its completion at the very least. The warmer the weather and the more abundant its hosts, therefore, the sooner will its life-cycle be completed. In each of its three stages the tick is capable of great vitality. Larvæ may live entirely without food for seven months or more until a host is secured, nymphs six months, and adults twelve months or more.

Tick life is most active in summer and autumn, and consequently heartwater is most rife in these warmer seasons. Spells of very hot weather, however, seem more deadly to the unfed tick than the cold of winter.

The range of the bont tick is a somewhat limited one. It seems to require a good deal of moisture and warmth for its development, for it is never found in high altitudes or in parts where dry climatic conditions prevail.

Some of Lounsbury's later experiments showed that the infection of heartwater could be lost in ticks which were forced to undergo their moult at very low temperatures; yet if warmed up for a few days in an incubator before placing them on a host they would become able to transmit the infection. These results explain the erratic appearance and disappearance of the disease as met with sometimes under natural conditions.

BACTERIOLOGY.

Microscopically, no visible organisms have been found. Their existence, however, is readily proved by the virulence on inoculation of the blood, spleen pulp, thoracic and other effusions. The poisons of the infective agent are very powerful, and produce a severe intoxication, which usually brings about the death of the subject in a violent paroxysm. The organism of heartwater, although it has not yet been demonstrated microscopically, will not pass through the Chamberland F. filter-candle.

MORBID ANATOMY.

The animal's condition is but little affected by this disease, its course being almost always too short. The attitude of the carcase may to some extent suggest heartwater: head thrown back over the shoulder, saliva at the mouth, tongue protruded, the earth around churned by the galloping movements of the limbs prior to death. On skinning off, slight injection of the subcutaneous veins may be noticeable; there may be a little lymphy material beneath the brisket. On opening the chest, a large quantity of clear yellow sero-albuminous fluid may be seen, or it may be absent. When present, it is very typical; collected in a vessel, it will coagulate quite firmly almost immediately. The heart sac may also attract attention at once from being full of a similar fluid, but again this fluid may be scanty or wanting. Sometimes both pericardial and pleuritic fluids are plentiful; sometimes either is excessive and the other almost wanting. If the animal has been some time dead before the autopsy is held, these fluids are frequently found blood-stained. There is no true pleurisy or pericarditis. The heart itself shows a few petechiæ or ecchymoses outwardly or within its cavities, chiefly the left ventricle. The blood is bright red in colour, and clots readily. The lungs are frequently normal, but always where effusions are plentiful their dependent lobes will show interlobular œdema, most marked at their fringes, and resembling the rivers on a map. The trachea and bronchi, on section, are found full of the same effusion worked into a white froth.

In the abdomen a little effusion may be seen occasionally. The first three stomachs are normal, the abomasum alone showing slight inflammatory œdema, a congested mucous membrane, or at times a multitude of tiny petechiæ on an otherwise normal mucous membrane. The small bowel may be congested or nearly normal, showing only a few ecchymoses here and there; the large bowel, usually normal, is injected or inflamed in those cases where diarrhœa was severe during life. The liver is normal or slightly congested; the gall-bladder is full of normal bile. The spleen is nearly always enlarged; its pulp is softened, and a few petechiæ are frequently seen under its peritoneal covering. The lymphatic glands are little altered, except for œdematous changes. The peritoneum is normal; the kidneys may be slightly congested; the bladder and urine are normal.

The brain and its membranes are usually congested, and the spinal fluid is frequently increased in quantity.

The lesions found at a heartwater post-mortem are apt to vary greatly, even typical ones being absent. When the effusions were wanting, the South African farmer was wont to diagnose "gall-sickness." At all times, if the animal is killed early in the fit or before it, the distinctive lesions are almost entirely wanting.

PERIOD OF INCUBATION.

When induced by tick infection, the disease develops in from eleven to eighteen days, one infected tick being sufficient to convey it. If the tick or ticks feed on the skin of the body, the disease incubates a day or two sooner than when they feed on the region of a hind-limb. When susceptible small stock move on to infected pasturage, they generally break out with heartwater in from fourteen to twenty days. By intravenous injection of a huge dose of virulent blood, Edington reduced the incubatory period to five or six days. A dose of 5 to 10 c.c. generally takes eight to ten days, and a smaller dose a day or two longer. By subcutaneous injection this period is much lengthened, fifteen to twenty days being quite common, and in one case the writer noted an incubatory period of twenty-six days. Naturally, the disease reaction is apt to become milder as its incubatory period is lengthened.

SYMPTOMS.

These are best seen in the goat. Although there may have been high fever for several days, the first symptom noticeable is a stiffness in the gait. The affected animal falls behind the flock whilst at pasture. If driven, he will quickly show respiratory trouble, the sides heaving, the nose being poked forward, and the tail dropped or quivering. Next he will lie down and refuse to rise. If left undisturbed, he will remain quiet, but gets up again after a time. He now appears dull; rumination has ceased, but he will generally feed a little frequently up to shortly before death. Many cases show nervous disturbance from the first, bleating at intervals, and shortly going into a fit or series of fits. This is easily accelerated by driving or otherwise hustling them. During a fit it will be noticed that the tongue projects a little from the mouth, the latter is kept half open, there is a noticeable retraction of its angles, slight salivation and chewing movements begin; the goat bleats plaintively, and the tongue is kept constantly moving out and in through the lips; the eyes squint and rotate in the sockets; the nose is often brought round towards the chest. If left alone, these symptoms may pass off somewhat; the goat stands up again, and may walk on slowly, but must not be driven. If hurried, another and worse fit will come on, and death often rapidly supervenes. In this stage the goat lies flat on one side; often there is marked opisthotonos, the head being thrown upwards on to the withers, and we observe violent bleating, chewing movements, slobbering with the tongue, squinting, and quivering of the muscles, generally accompanied by galloping movements of both fore and hind legs. This fit may end suddenly, or may last for hours, the animal's moanings and plight exciting one's pity to a marked degree. It is very seldom indeed that a goat in this state recovers. Sometimes they die comatose. Towards the end there is generally more or less tympany, due to decubitus. Fæces and urine are passed at frequent intervals; both generally appear normal, but the former may be diarrhœic and occasionally blood-streaked. Diarrhœa, though not frequent, is seen in some rapidly fatal cases. Sometimes the attack is so sudden that the goat is not noticed sick at all, but is found dead in the kraal or left behind on the pasturage.

In a period of some weeks after recovery from a severe attack, both sheep and goats will cast their fleece.

The fever reaction is peculiar; it generally rises abruptly from normal to 105° or 106° F., or even higher, remains there for a day or two, temperatures of 107° to 108° F. being frequent; then it will drop just as suddenly to subnormal shortly before death.

Symptoms in Cattle.—Adult cattle seldom show any symptoms; indeed, it may be assumed that these have become immune from exposure to infection as calves. On farms where heartwater was rife in small stock, young calves, soon after going to pasture, frequently died in a fit somewhat resembling that seen in the goat, but less typical, squinting and violent twitching of the eyelids being the most noticeable symptoms. Very often such deaths were put down to hairball, poisoning, or gall-sickness, and the true cause was not even suspected.

In small calves, then, the first symptom noticeable would be fever (106° to 107° F.), staring coat, dulness and depression; then a slight frothing from the mouth, and very frequently stiffness of the limbs and a staggering gait. Later, as the fit threatened, there would be chewing movements, licking with the tongue, eyes staring, squinting, and showing the sclerotic veins highly injected, with intermittent spasms all over the body. Next the calf falls down, sprawls about, and finally lies stretched out on his side, bellowing piteously, struggling with all four legs; the eyelids blink violently, and he dies comatose.

Incubation after tick infection lasts twenty to twenty-five days in cattle, and death follows within four days of the animal being noticed sick.

Lesions in Cattle.—Acute abomasitis is observed, the mucous membrane and rugæ being much swollen. The bowels show swollen œdematous mucous membranes, with hæmorrhagic patches. The lungs are often œdematous; there is froth in the trachea and bronchi, and yellow fluid in the chest in varying amount. Fluid may also be present in the heart sac, petechiæ on the external surface of the heart, and probably in the left ventricle, but these are not constant. The liver is congested, the gall-bladder full of clear yellow, green, or brown bile. The lymph glands are swollen and congested. The spinal canal and brain cavity contain an increased quantity of fluid, the brain parenchyma being frequently sodden.

COURSE AND PROGNOSIS.

The course of this disease is nearly always very rapid, and the mortality exceedingly heavy. Once having gained entrance to a pasturage, the infection remains permanently upon it unless the methods to be described later are efficiently carried out. Susceptible small stock brought on to such a pasturage die off very rapidly; losses of 50 per cent. within a few months have been known amongst merino sheep, and in young angora goats quite 80 per cent. might be reached. By-and-by the remnant of the flock acquires immunity, but odd cases are apt to crop up from time to time, and the disease always exacts a heavy toll from each season's lambs and kids.

DIFFERENTIAL DIAGNOSIS.

Heartwater in small stock is readily recognised during life by the very high temperatures and the peculiarity of its fits. The post mortem, too, is very frequently typical; its clear yellow effusions, coagulating rapidly upon exposure to the atmosphere, distinguish it at once from those anæmic conditions due to fluke, wireworm, etc., where a watery, colourless, incoagulable effusion may also be seen in the chest, heart sac, and abdomen.

TREATMENT.

No medicinal treatment has so far proved of any real merit, though farmers' recipes used to be legion. The best course to pursue is to isolate all sick animals, pen them singly, and leave them entirely undisturbed in a cool shady situation. The trypan blue injection method has been tried in cattle, but was found useless.

IMMUNITY.—Heartwater, as already remarked, has been a subject of experiment for many investigators. The work was undertaken to elucidate many points in its etiology and nature, but more especially with the idea of finding some inoculation method which would produce a workable immunity. The task was found to be an exceedingly difficult one, and finally, when success seemed to be wellnigh at hand, the manner in which dipping began to achieve the destruction of tick life diverted the attention of everyone to a more radical and surer way out of the difficulty, and practically removed all necessity of finding a protective inoculation.

The earliest investigations proved that the disease was not directly contagious, and that small stock could only be run upon infected veld when so thoroughly greased as to make them repellent to all tick attack. When the tick was excluded, then nothing save the inoculation of viruses obtained from the sick or dead animal succeeded in transmitting the disease. The blood of a sick animal was found to be highly virulent when fresh, but in three days or less it becomes inert. The addition of 5 per cent. glycerine or 0·25 per cent. carbolic acid rapidly destroy it, as does also desiccation. Robertson found that seven minutes' exposure to a temperature of 52° C. killed its virulence, and lower temperatures failed to give the attenuation which he sought. Bile was found to be non-virulent, and to convey no immunity. On the other hand, recovered sheep, goats, and cattle possess a serum of antitoxic value, which, after fortification, increases greatly in potency; yet all attempts to balance such serum with a dose of virulent blood simultaneously injected into experimental goats, so as to pass them through a mild heartwater sickness, gave more or less erratic and uncontrollable results. Serum from hyperimmunised horses was equally unsuccessful. The investigations, however, elucidated many interesting points which deserve mention here. It was shown that the disease could be conveyed by inoculation into the vein, under the skin, or into the thoracic cavity, by means of the blood, spleen pulp, or effusions of a sick animal. In this way the disease can be passed from any affected animal to sheep, goats, or cattle, and from one to the other at will, in endless succession apparently without any attenuation—in fact, by intravenous injection the virulence appears constant, a dose of 5 c.c. or more almost invariably destroying every susceptible animal. Fresh blood, defibrinated or citrated, is the most convenient for such experimental injections. If tapped at the height of the fever reaction, it is at its highest pitch of virulence. In such acute cases the blood was proved to be virulent from twenty-four to forty-eight hours prior to the onset of fever. In cases where recovery ensued, the blood at the height of the fever was apparently as strongly virulent as in that of a goat which succumbed; at later dates, however, the writer found that the blood had somewhat attenuated, and now transmitted an infection of decreased intensity, from which recovery would ensue. Blood taken from the reacting goats ten days or more after the fever reaction had ceased very often produced a similar elevation of temperature in a third, fourth, fifth, and sixth series of goats. Blood taken at fifteen, twenty, and twenty-five days after the temperature had regained normal was found to be of decreasing toxicity, and after a month or so it proved quite inert. This bears out the experience of the disease in Nature: immune animals do not act as a reservoir of the virus, and ticks fed on such animals cannot pick up the infection. Similarly, in his experiments Lounsbury found that ticks feeding on recovering animals picked up less than lethal infections, and, after moulting, transmitted such strains of lessened virulence, thus duplicating the experience of the disease upon the veld. By adding a less than lethal dose of virulent blood (1 c.c. and 1·5 c.c.) to a dose of " recovering " blood and injecting the two simultaneously, the writer produced very severe reactions and sickness, ending in recovery, and it almost looked as if finality had been reached; but when a small flock of yearling merino sheep and angora goats inoculated by this method was run upon infected veld for twenty-one months, some unexpected things happened. The sheep, all of which were inoculated once only, did remarkably well, and appeared to have acquired a high degree of immunity, only 12½ per cent. of them dying of heartwater. Most of the angora goats were inoculated once only. Those which had had two or three of such experimental inoculations appeared to be satisfactorily immunised, as also some whose one inoculation had produced a lengthened and severe reaction. No goat whose fever reaction lasted less than eight days could be said to have acquired a high degree of protection. During the twenty-one months in which the flock grazed on the infected veld one-third of the goats died, and several others were sick twice over, making, when the inoculation disease is counted, no less than three recoveries within two years from that peculiar complaint. On the other hand, 80 per cent. of a small lot of control goats died of this disease. To cap the veld infection, two goats and three sheep were inoculated with 5 c.c. of virulent blood into the vein: one goat died on the seventeenth day after; the other sickened and recovered. Of the sheep, one was sick and recovered, one was fevered only, and the third resisted this severe test completely.

Discouraged by these results, the writer turned to the simpler method of subcutaneous injection of virulent blood. For full-grown goats, 5 c.c. is frequently dangerous; sheep will stand rather more, but yearling merinos often succumb to a primary injection of 4 c.c. On the other hand, as much as 20 c.c. has sometimes been injected without killing or even making the animal (sheep or goat) visibly sick. With this great variance in susceptibility, much circumspection had to be used, but finally the following doses were fixed as being workably safe:

	First Vaccine.	Second Vaccine.	Third Vaccine.
	c.c.	c.c.	c.c.
Kids and lambs	1·5	1·5	2·0
Goats and sheep (2-tooth)	3·0	3·0	4·0
,, ,, (4-tooth)	3·5	3·5	5·0
,, ,, (6 to 8 tooth)	4·0	4·0	6·0

The danger from this method is greatest after the first and second vaccines; the third can safely be made much larger than either of the two preceding it. A fourth vaccine adds greatly to the protection conferred, and can with safety be much larger than the third. These injections must not be practised at a less interval than one month. Sheep can be put through the process safely, but angora goats are always more difficult to handle. After three vaccines practised as above, an experimental infection with pathogenic bont nymphs was resisted completely by a number of two-tooth merinos, some of which had given no very tangible thermal reaction to the vaccines. After the tick test, large doses of virulent blood injected into the vein were resisted *without reaction.* In practice a farmer might utilise this method whenever a sick animal cropped up to serve his purpose; to apply it systematically there would always be the necessity of keeping up a chain of live animals to furnish the sick blood for injection at the proper intervals. The method, therefore, has to-day little more than an academic interest.

PROPHYLAXIS.

Now that we know by what means heartwater is transmitted, preventive measures readily suggest themselves, and chief amongst these is—

1. *Dipping.*—The dipping of small stock in an arsenical dip strong enough to kill ticks was found to be effectual in checking the disease for a time. Perhaps a weekly immersion in Watkins-Pitchford's three-day dip might prove permanently successful upon badly infected pasturage, but few would be found willing to dip sheep or goats so frequently. From the point of view of greater tick destruction, much more rapid and satisfactory results would be effected by dipping the cattle of the farm. In practice a fortnightly immersion has succeeded where the dip was strong enough to destroy the adult tick. The five-day or so-called "laboratory dip" of Natal would succeed even better, and in a much shorter time, for by its use neither the larval nor nymphal forms of the tick would ever escape. Quite apart from heartwater, the dipping of cattle has been found to be a paying proposition, as it is saving to-day many calves which in former times used to succumb to redwater, anaplasmosis, and hairball; while heifers now come to calving with udders and teats intact which beforetime were often sloughed off by suppurating sores, caused chiefly by the bont tick.

Although no other prophylactic method can compare with dipping, still, two others have been practised with some degree of success:

2. *Burning of Grass.*—This should be done in late autumn, when many ticks are to be seen on the heads of the long grass. It can only partially succeed, however, as ticks during moulting and egg-laying lie secure upon the ground. For many other reasons, also, grass-burning is objectionable.

3. *Starving of Ticks.*—This can only be done where the farmer has fenced paddocks, and can afford to keep them free from all stock which would act as hosts for at least fourteen months. Owing to the presence of hares, wild antelopes, and birds, complete success would not be secured; besides, no farmer with plenty of stock could afford to rest more than one paddock at a time.

As a palliative measure, change of pasturage used to be greatly in vogue. When heartwater caused losses in a flock, they were moved off to new grazing; if the new grazing contained no bont ticks, then the disease stopped for good and all. The Boers in the Transvaal low veld knew this well, and when troubled with the disease they promptly moved off to high veld. In our more settled Eastern Province it was a common experience that some parts only of a farm were infected, the flats and valleys more particularly, whilst higher ridges and hillsides remained clean or nearly so. Intelligent farmers often succeeded in keeping down their losses by frequent changes of grazing during the summer months from the flats to the tops and back again after a short interval, and that, too, without in the least understanding the reason for their success. If no veld free from bont ticks is available, the affected flock can still be saved, the mortality being checked by two changes of grazing, each lasting three to four weeks. In this way bont ticks feeding on a sick sheep would fall off to moult and be left behind without a host to infect in their next feeding stage. This is a method on all fours with that employed so successfully against East Coast fever.

BLUE-TONGUE

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Synonyms.—Catarrhal fever, or Malarial catarrhal fever (the latter is the name originally given to this disease by the late Mr. D. Hutcheon). In the Cape Province it is often styled simply " Fever."

Dutch : Blauwtong and Bekziekte (literally, " mouth-sickness ").

Definition.—A specific disease affecting sheep in South Africa, characterised by fever and a catarrhal inflammation of the mucous membranes of the mouth, lips, tongue, upper air-passages, and alimentary canal, frequently also by inflammation of the laminæ and coronary band of the feet. Severe cases sometimes end in suppuration and casting of the hoofs, and patchy shedding of the fleece is also frequent.

SHORT HISTORY AND GENERAL REMARKS.

Although this disease must have been known at the Cape since its early settlement, very little notice of it was taken until within quite recent years. Its nature was but little understood until it became the subject of investigation by the Government veterinarians in 1901, and subsequently Messrs. Dixon, Robertson, Theiler, Watkins-Pitchford, and the writer, have all contributed their share to the common knowledge with which this disease is now surrounded, and which will be here set forth.

Blue-tongue is always most prevalent under conditions of warmth and moisture, whilst during very dry seasons it may appear only in a mild form, causing few outbreaks and very little loss. In some parts, however, it appears year after year, causing heavy losses, especially in the wetter seasons. In such localities it would appear to possess some special virulence of type. It is now considered certain that the infection is carried by some biting insect, probably a mosquito ; and as outbreaks always occur in summer or autumn (roughly December to April), and disappear soon after the first frosts, this seasonal incidence lends great force to the above belief. Sheep kraaled in low, damp situations, or near a pond, dam, or stream, are most liable to suffer, whilst those on a hillside escape; but altitude is not everything, for the disease is met with on plateaux 4,000 feet or more above sea-level. It is the local elevation which counts for safety; sheep on low, hilly situations remain healthy, when those in the valleys hard by are suffering from the disease. Moreover, the dipping of a flock will often check the disease in their midst, the fleece evidently becoming in this way repellent to the transmitting insect. Farmers, too, have found it inadvisable to shear their sheep at such a season.

Shedding of small stock overnight will also prevent this disease; and as kraaling does not do so, the presumption is that the roof overhead in some way prevents the attack of the mosquito—most probably by producing in the confined atmosphere of the shed just sufficient smell of ammonia (arising from the manure) to make it objectionable to the insect. Something similar has been noted in stables in respect of the transmittal of horse-sickness. One of Watkins-Pitchford's experiments goes a very long way towards proving the likelihood of a fly as the transmitting agent. In 1902, during the blue-tongue season, he kept sheep in gauze-covered pens on the banks of a river; these remained healthy for three months, whilst control sheep tied up outside contracted blue-tongue freely. The protected sheep were allowed to graze out in the open in the daytime, only being returned to the gauze pens every afternoon. Sick sheep were placed with them, and some even died inside the pen, but the test sheep remained healthy. Later, they were inoculated to prove their susceptibility, and they reacted promptly. In the following year he repeated this experiment, and with exactly the same result. Thus it was shown that the infection of blue-tongue is apparently never transmitted in the daytime, and that sick sheep do not infect healthy ones either by contact or by their natural discharges. It is not known whether the mosquito can feed upon a sick sheep, and then at once transmit by feeding upon another susceptible one. Another of Watkins-Pitchford's experiments appears to show this transference as a mechanical one. Four inoculated sheep were penned in the open with four clean ones, there being no disease around at the time. The contacts became sick—one on the nineteenth day, one on the twenty-first, one was fevered erratically from the tenth day, and the fourth proved immune. In spite of this result, such direct transmission seems unlikely in most instances, for the disease often crops up in a bad season in places where it has not been known for years. It seems more probable that the causal micro-organism passes some phase of its existence within its insect-carrier, and this belief is further strengthened by the fact that mosquitoes and biting flies have not been found capable of travelling any great distance under normal conditions. There may be a reservoir of the virus somewhere in Nature, possibly in an aquatic animal, but so far it has not been found. No infection can be obtained from the blood of immune sheep; when these contract a second attack, it is always due to a fresh infection.

DISTRIBUTION.

This disease is well known in most parts of South Africa, particularly in the Cape, Natal, Transvaal, and Rhodesia.

BACTERIOLOGY.

No organism has yet been found microscopically in the blood or tissues of a sick animal, or isolated from them by bacteriological methods. When virulent blood or its serum only is passed through the Berkefeld filter, the filtrate is found to be infective for susceptible sheep upon inoculation; consequently the causal organism must belong to the ultravisible class. The virus is possessed of considerable powers of resistance *in vitro*. Watkins-Pitchford kept virulent blood in sterile bottles, and found it still infective up to seventy-five days, and sometimes even up to ninety days.

SPECIES AFFECTED.

The pure-bred merino is the most susceptible, especially when young, the disease becoming less severe as the adult stage is reached; but there is a great range in the susceptibility of individuals of the same breed. A few merinos show a nearly complete immunity; others contract abortive forms only. Cross-breeds, both of the Africander and the Persian, suffer in a considerable degree, but less severely than the merino. It may be that the increased resistance of the adult sheep is due to partial immunity acquired from a previous attack. Lambs do not suffer till after they are weaned. Pure Africander and Persian sheep suffer much less than the merino.

Although only the sheep is known to suffer from naturally contracted blue-tongue, the writer found that goats (both the angora and Boer goat) could harbour the infection after inoculation with virulent blood. Generally no febrile reaction at all resulted from this injection, yet the goat's blood tapped five to ten days later would reproduce the disease with all its symptoms upon inoculation into a susceptible sheep. Ten angora goats were so inoculated, from goat to goat in series, and the blood of the tenth reproduced the disease when injected into a sheep. Blood taken from these goats at dates later than ten days after injection might prove virulent for sheep, or might prove uncertain. After twenty-one days it generally gave a negative result.

In a similar way young calves could harbour the blue-tongue infection just as the goats did, and their blood retransferred to susceptible sheep reproduced the typical sickness.

Robertson failed to infect either the horse or the dog.

MORTALITY.

In natural outbreaks the mortality varies very greatly. In some seasons a 5 per cent. loss would be relatively heavy, as the majority of flocks might entirely escape. In others, especially in areas notorious for its frequent or annual recurrence, 10, 20, or even 50 per cent. of a flock might succumb. Apart from the death-rate, however, the farmer's loss is a heavy one, because of the severe loss of condition in the sheep which recover, and the deterioration in their wool. By subcutaneous inoculation of virulent blood, about 20 per cent. of fatalities would generally result if yearling merinos were used.

MORBID ANATOMY.

As a rule, death ensues from debility, and the mouth and other lesions have generally healed by that time. It may happen, however, that the blue line around the coronets is distinguishable, or perhaps an unhealthy-looking wound inside the cheeks and in contact with the molars will assist one in diagnosis. For the rest, all that will be seen is an emaciated carcass.

In severe cases the tongue is stripped of its epithelium, and may be gangrenous at the tip. The dental pad and inner surface of the lips are raw and excoriated, and sores are found inside the cheeks and in the region of the molars, the latter being usually the last to disappear. The throat and base of the tongue are healthy, and only in very severe cases do we find the hard palate affected. The mucous membrane of the nasal septum and frontal sinuses is generally much congested. The œsophagus is healthy, but the rumen often shows inflamed patches, more especially affecting the pillars and the rumeno-reticular margin, and there may even be a slight ulceration at these parts. In cases where vomiting was frequent, lesions of the paunch are sometimes lacking on post-mortem.

The reticulum is sometimes a little inflamed, but the omasum is almost invariably healthy and its contents soft. The abomasum may be severely inflamed, or only mildly so. Sometimes its fundus, and at other times the pyloric end, is most affected, and occasionally the mucous membrane is very œdematous in the latter regions. There may be tumefaction and erosions of the rugæ, with one or two ulcers at the pylorus; or, again, the stomach may seem nearly healthy except for a host of punctiform petechiæ, which give its mucous membrane a peppered appearance.

The small bowel is often inflamed throughout, generally most so at the duodenum, and it contains an abundance of sticky mucus. Very often the inflammation is found in patches, or there may be slight congestion only. When diarrhœa was severe before death, the small intestine is found acutely inflamed with zebra markings, and Peyer's patches are inflamed and prominent. In such cases the large bowel is inflamed even to the rectum, but generally this part of the alimentary canal is much less affected than the small bowel.

The mesenteric lymphatic glands are more or less inflamed and œdematous. The spleen is as a rule considerably enlarged, soft, and pulpy; it is, however, often firm, and seldom very dark in colour. The liver is congested, but otherwise normal; the bile is normal, and the gall-bladder generally full.

The kidneys are congested, and may be marbled on their external surface from hæmorrhage into their cortex; sometimes they are œdematous, and the capsules strip off easily. The cortex may be of a brownish colour on section. The bladder is generally full of urine, which is always clear.

In the chest we find a slight amount of yellow fluid occasionally, and a little is often present also in the pericardial sac. The heart is usually congested, and in all severe cases shows ecchymoses under its endocardium, and even deeply into the muscular structure; its cavities are generally full of dark, fairly firm clots. The lungs are usually healthy, but may be more or less œdematous; when much vomition has taken place, the dependent parts are found of a dark red colour, hepatised or collapsed, and œdematous, while the trachea and bronchi are full of froth, containing particles of the vomited ingesta, and their mucous membrane is in an acutely inflamed condition.

The subcutaneous tissue of the jaw and under-surface of the neck may contain a little colourless or pale yellow watery effusion.

PERIOD OF INCUBATION.

The incubatory period of this disease as produced under natural conditions has not been determined, but experimentally it is two, three, four, or more clear days—the average being four.

SYMPTOMS.

In a typical case the onset of the malady is marked by fever, which is often severe, reaching as high as 107° F.; but temperatures of 104° to 106° F. are the more usual, and sometimes fever is almost absent, although the symptoms develop in the usual manner and at the proper time. There is nothing very typical about the fever reactions; they are often very erratic, the morning readings especially being lower than one might reasonably expect.

The fever period generally extends from five to seven days, shorter or longer periods being frequent, and there is always a marked drop when the eruption takes place. Although the fever symptoms appear so early, it is not before the seventh to the tenth day that the distinctive lesions in the mouth show themselves. A careful observer, however, would note that the sheep looked thin and did not feed as well as usual for one or two days preceding the eruption. Examination of the mouth may then detect a slight rosiness of the mucous membrane, or occasionally an abnormal blueness, but very often such change is unnoticeable.

The first thing that generally attracts attention is a slight frothing at the mouth and swelling of the lips, particularly the upper lip, with a catarrhal discharge from the nostrils. Frequently the sheep makes a smacking movement with the lips and tongue. If the mouth be examined—a proceeding which the sheep most actively resents—its lining membrane will be found to be somewhat blue in colour, and the lips may be sore along their free margins. Next there is an œdematous infiltration into the mucosa of the dental pad, and also the part immediately behind the incisors; this causes an abnormal whiteness of these parts. In mild cases this condition lasts two or three days; the loosened epidermis is rubbed away by feeding, and recovery rapidly takes place. In a severe case, however, the surface of the pad becomes raw, sores appear inside the lips and cheeks, and even on the bars of the hard palate; the tip of the tongue becomes sore by shedding of its epithelium, and may even be gangrenous.

In very severe cases the tongue becomes badly affected, and may be almost wholly stripped of its epithelium and of a dark blue colour. In a few cases this organ becomes extremely œdematous, quite fills the mouth, making swallowing almost impossible, and there is a copious salivary discharge from the mouth. In such cases the tongue, especially on its lateral aspects, where the mucous membrane is thin, presents a glistening yellow or amber colour, resembling a huge blister. In all such cases the lips become greatly swollen by a serous infiltration into their subcutaneous and submucous tissues; while the very profuse nasal discharge dries upon the nostrils, diminishing their apertures, and causing stertorous breathing. Sores are frequent also on the skin of the nostrils and muzzle. There is occasionally an œdema of the lower parts of the face, and especially of the skin and tissues under the jaw, extending, though very rarely, for a distance down the under-surface of the neck.

Despite the soreness of the mouth, the sheep have usually an inclination to feed; yet loss of condition is rapid, even when the mouth lesions do not for long interfere with feeding.

Occasionally diarrhœa sets in, and this is always very fatal, blood being passed in the fæces in the later stages. As a rule, the fæces are almost normal, except for the presence of a more or less abundant slimy mucus.

The mouth lesions generally last from three to five days, but heal less rapidly than they form. Very soon the appetite returns, the feet become tender, and there is a bluish or red line around the coronets, which are swollen and painful on pressure, especially at the heels. Sometimes the skin of the pasterns is swollen and inflamed. Most commonly both fore-feet are affected; sometimes all four, both hind, or a fore-foot and a hind one at the same time.

The severity of the foot lesions is not always in the same degree as the soreness of the mouth. Cases with only slight soreness of the mouth often become very foot-sore. This symptom generally appears from the fourteenth to the seventeenth day after inoculation, and passes off in a few days; but it is aggravating while it lasts, as it causes the sheep to lie a great deal and refuse to feed; or, if hungry, they feed in a kneeling posture, and do not move about enough to fill themselves. The gait of such foot-sore sheep is very characteristic, and resembles that of a foundered horse. It is only in the worst cases that the feet become tender whilst the mouth is still sore. In some cases the foot trouble is very mild even after marked mouth lesions.

There is great thirst during both the febrile and eruptive stages. When the sheep begin to feed well again, vomiting may supervene, more especially if they have had free access to water. The ingesta are voided by the nose, and in many cases inhaled into the lungs, giving rise to a very fatal pneumonia.

In young animals, more especially if they be in poor condition, this disease runs a very rapid and fatal course, often with diarrhœic complications. Emaciation is severe; the sheep lie persistently, although the feet are not yet affected; the nose is brought round into the flank, and sometimes the neck becomes twisted (wry neck), death ensuing within eight to ten days after inoculation. In such cases the whole skin of the body may show an abnormal rosy hue; the wool can be pulled out in tufts, prostration is extreme, the animal's strength collapses, and, after a short period of coma, death supervenes. Fortunately, such very severe cases are exceptional.

After severe attacks recoveries are very slow, the sheep falling into a state of extreme debility, and the neck at times remaining twisted for a very long time. Forcible reduction of the twist shows that there is neither pain nor stiffness of the part, but sheer muscular atony alone. As a rule, the disease has run its course and complete recovery has taken place within twenty-four days after inoculation. In inoculation cases the eyes are seldom affected, and shedding of either hoofs or fleece is very rare and only partial. Such symptoms are more frequent in outbreaks caused through natural infection.

It will thus be seen that clinically the disease may be divided into *acute*, *subacute*, and *abortive* forms. There is no true chronic form, although death occurs oftener from the resultant debility which follows upon an attack than at the stage when the symptoms are at their height. By inoculation we find that many animals possess a high degree of resistance, and we thus recognise the abortive forms. Such forms range from those showing only an irregular temperature to those developing more or less marked fever reaction, and others still which, in addition to the fever, show for a few days a distinct reddening of the oral mucous membrane.

DIFFERENTIAL DIAGNOSIS.

Blue-tongne is not difficult to diagnose. The season of the year, locality, history of the outbreak, and sequence of the mouth and foot lesions, should differentiate it from foot and mouth disease. There is also the well-marked blueness of the oral membranes, white dental pad, and lack of eruption on the coronets. In "geeldikkop" (literally, "yellow-thick-head") the whole head, face, and ears swell up; the subcutaneous effusion is markedly yellow; the skin tends to dry and crack, that covering the ears often undergoing dry gangrene; and there are no foot lesions. In foot-rot the lesions give off fœtid discharges, mostly on the solar surface of the claws or in the interdigital space, and there are no mouth lesions.

CURATIVE TREATMENT.

The affected sheep should be removed at once from the flock, and placed in a shed or small shady paddock, and preferably upon green lucerne. Even severe cases will recover if not continually over-dosed with fanciful remedies, disturbed by dogs, or driven long distances when in a weak state. In most outbreaks the mortality does not exceed 10 per cent. of the affected animals; hence many remedies have been extolled which, after all, can have but a feeble or negligible effect upon the course of the disease. For the mouth lesions weak antiseptic and astringent lotions, such as alum or chlorate of potash, may be used. A mixture of Stockholm tar and sulphur is also a favourite remedy. The chief aim should be to provide a liberal, soft, succulent diet for animals whose lips and tongues are swollen and painful, and who yet often retain a considerable degree of appetite, and this especially in the later stages, when the mouth is practically healed, but the feet so tender that the sheep can scarcely get about. Milk or gruel may with advantage be given to bad cases by means of a funnel and rubber tubing. Sick sheep appear to greatly relish a lick of common salt, and this also should be provided. Often they will drink large quantities of water, and in this state they should not be driven, as it will bring on vomiting, which is frequently followed by a fatal inhalation pneumonia. When the muzzle is greatly swollen, the discharges often become dry and occlude the nostrils, and these should be sponged clean at intervals. Where the sheep, in addition, suffer from grub in the head, this trouble will be particularly frequent. When the tongue and lips are greatly swollen, a slight scarification will assist recovery. The feet may occasionally require a little trimming.

EXPERIMENTAL MODES OF INFECTION.—The blood, uncoagulated, citrated, or defibrinated, blood-serum, spleen-pulp and effusions, taken at the acute stages, are all virulent. It seems to matter very little by what means these are inoculated—subcutaneous, intravenous, intratracheal, intrathoracic, and intra-abdominal injections all reproduce the disease alike.

The dose does not very markedly affect the character of the resulting sickness. In one case a single drop of fresh citrated blood introduced under the skin produced fatal sickness. Some sheep will withstand comparatively large doses of this virus without showing more than a febrile reaction. As a rule, the degree of sickness produced depends more upon the susceptibility of the subject inoculated than upon the apparent virulence of the attack in the sheep from which the blood is taken.

Watkins-Pitchford claims to have produced blue-tongue by drenching susceptible sheep with virulent blood, but this means failed in the hands of Theiler and Dixon.

Dixon first demonstrated that the blood could be preserved in the fluid state and still retain its virulence. To the freshly drawn blood, citrated or defibrinated, he added an equal bulk of a solution composed of equal quantities of glycerine and water containing $\frac{1}{4}$ per cent. of carbolic acid. Blood preserved in this way proved virulent for very long intervals, though there is an undoubted attenuation after a time. In this respect blue-tongue blood resembles that of horse-sickness, and greatly differs from that of heartwater. Even attenuated blue-tongue bloods can be so preserved, and Theiler has taken advantage of this in the manufacture of his vaccine. The blood of sheep recovering from blue-tongue gradually becomes less virulent, but in some subjects this attenuation proceeds much more slowly than in others. In general, the blood remains fairly virulent for about thirty days after the fever has abated. In one case of a severe type the writer found the blood still virulent fifty days after the fall of fever. This was probably quite exceptional, and, anyhow, at later dates the blood of an immune sheep proves quite innocuous, thus showing that the immune animal does not form a reservoir for the virus. The immunity produced by one attack of blue-tongue is only relative; re-inoculations with virulent blood sometimes produce marked fever reactions, sickness, and even death. An imported ram has been observed to suffer from naturally transmitted attacks in several consecutive seasons. On the other hand, once immune, a sheep can have its immunity greatly strengthened by repeated injections of large quantities of virulent blood. This fortification method, similar to that employed for the production of an antitoxic serum against rinderpest, was initiated by the writer in 1901, and with immediate success. With a strong antitoxic serum, on the one hand, and a preserved virulent blood on the other, the conditions necessary for a sero-vaccination method for the production of immunity in susceptible sheep were easy. Later, it was found that fresh sheep could be vaccinated in this way, and rapidly fortified thereafter for use as serum-producers.

PROPHYLAXIS.

From what has already been written, it will be seen that the prevention of blue-tongue is under some conditions fairly easy. Such a precaution as that of "kraaling" in some elevated situation overnight may be possible; dipping, too, may be employed at fairly short intervals; or the stock may be placed overnight in sheds, if such are available. On low-lying farms, however, especially in the case of large flocks, these precautions, though good in themselves, would hardly be attainable; suitable sheds are expensive, and frequent dipping for many reasons undesirable. The necessity for a protective inoculation was there, and a safe method had only to be discovered and made known to the farmers, so as to create a demand for it. In January, 1902, the writer put his method into practice in the field. It was used at first in infected flocks, and proved at once successful. The method consists of the simultaneous injection of 2 c.c. preserved virulent blood and 4 c.c. serum; these can be mixed in the correct proportions just before use, and a dose of 6 c.c. of the mixture is injected into each sheep. The inoculated sheep pass through a mild attack of blue-tongue, mostly with fever only. If the serum is potent, only a few very susceptible sheep will show symptoms at all, and these recover rapidly. The best season for inoculation is from October to December—that is, from one to two months in advance of the earliest date at which an outbreak would be likely to appear.

THEILER'S METHOD.—Dr. Theiler, Director of Veterinary Research, Union of South Africa, has now produced a vaccine consisting of an attenuated blood. He found that after many passages of the virus through sheep in his laboratory the virulence of the inoculation disease gradually diminished. After ten or more such passages, a modified disease was produced, in which there was no mortality; yet an immunity was conferred on the animals so treated equal to that following upon an attack induced by the natural infection. This method was introduced in 1907, and of late years has been largely used, and with very satisfactory results; and as it is easier and less expensive to produce than any serum could be, it has quite supplanted the writer's method. The immunity produced by this method lasts twelve months or more. The vaccination process reduces the sheep slightly in condition, so it is well to apply it in spring-time, when the grass is green and succulent. At this season there will be least set-back, and a strong immunity will be brought about just prior to summer, when veld infection is greatest. Ewes should not be inoculated during pregnancy.

WATKINS-PITCHFORD'S METHOD.—H. Watkins-Pitchford, F.R.C.V.S., F.R.S.E., late Bacteriologist to the Government of Natal, has recently introduced another inoculation method, also consisting of attenuated blood. In this case the attenuation is produced by heat. He says: "The blood is drawn from sick sheep under aseptic conditions, is collected and defibrinated without contact with the air. The virulent blood is then submitted in a specially arranged incubator to a temperature from 112° to 117° F. for a length of time, and it was found that by adjusting this exposure the degree of virulence could be controlled almost to the point of extinction."* The writer is not aware that this method has been put to any considerable use in the field as yet. Perhaps by producing in this way vaccines of different strengths it might be made the most suitable method for use against the widely differing types of infection which prevail in different localities.

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* "Report on Sheep Disease known as Blue-Tongue," *Natal Agricultural Journal,* 1908.

SOUTH AFRICAN HORSE-SICKNESS

By Lieutenant-Colonel H. WATKINS-PITCHFORD, F.R.C.V.S., F.R.S.E.,
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Synonyms.—African horse-death ; Perreziekte ; Paardenziekte.

Nature of the Disease.—South African horse-sickness may be described as a specific exudative equine malady. No causal organism has yet been demonstrated in the disease, which, though typically microbic in its general characters, must at present be classed with those other specific diseases in which an ultramicroscopic organism is presumed—rather than proved—to exist. All efforts of the bacteriologist to cultivate the organism outside the animal economy have failed hitherto, a fact which, together with the insusceptibility of small laboratory animals, has rendered experimental research into this disease a matter of much difficulty.

HISTORY.

Records of the disease are fragmentary, and date back only to the introduction of the horse by Europeans. The earliest recorded visitation of the disease is in the year 1719, when it assumed an epizoötic character, sweeping throughout Cape Colony and destroying large numbers of horses. The year 1801 was known to early settlers as the year of " the great horse-sickness."

Since the time of the first known introduction of horses by Europeans, now some two hundred and eighty years ago, the disease has levied a heavy annual toll upon South Africa, in some years mild in its incidence, and in others denuding a whole countryside of its horses. Edington cites an outbreak of this latter sweeping form of the disease which in 1854 destroyed 64,858 horses in Cape Colony alone.

DISTRIBUTION.

The disease is not confined to South Africa proper. Balfour (of the Wellcome Laboratories, Khartoum) has found the disease to exist on the Nile, while others describe its existence in countries far north of the Equator.

Some years ago I was able to obtain, through the kind assistance of the Colonial Office, a number of Consular Reports showing, on the east Coast, the prevalence of the disease as far north-east as Zanzibar, and as far as St. Paul de Loanda on the north-west. The Consul-General from Old Calabar (north of the Equator) reports the prevalence of the disease "throughout the territories of the Protectorate," and from Angola the official report states that the disease is "most deadly, and horses die shortly after being landed."

The term "South African horse-sickness" is therefore a misnomer, although the disease appears strictly confined to the African Continent, and assumes its epizoötic form near and south of the Line. Vast stretches of country exist where the presence of the horse proper is impossible, and from which he would seem to have been totally eliminated by the disease in question.

The horse is, of course, an equine closely allied to the zebra (with its three species, *Equus chapmani*, *E. burchelli*, and *E. zebra* or *quagga*, Burchell's and the common zebra), and South Africa has been, as we all know, from time immemorial the home of this species of Equidæ. It seems, therefore, not a little strange that so closely allied a genus as our domestic horse should have found existence in a natural condition apparently impossible in the subcontinent. The laws governing the questions of immunity and susceptibility are at present obscure, an apparently slight racial difference being sufficient often to constitute an impassable barrier.

CLIMATIC INFLUENCES.

As would be supposed from the consideration of the geographical distribution of the disease, warmth and humidity are recognised factors in its spread, conditions which obtain especially during the African summer and autumn months. Coastal districts, low-lying swampy lands, river valleys, etc., are especially liable to the ravages of the disease during the summer months, while in some localities—subtropical in character—the malady is to be found at all times of the year. Where outbreaks of an epizoötic character occur in the higher lands of the interior —"high veldt"—such outbreaks are found to be dependent upon the heavy rains of summer, and appear to have a direct relation to the level of the subsoil water.

The disease is found to disappear with the advent of the cold weather and light frosts of early winter, in those districts in which such seasonal influences obtain.

ETIOLOGY.

While the association with warmth and humidity has long been noted, the essentially paludal nature of the disease has escaped recognition until recently. Many and strange theories have been brought forward to account for horse-sickness, such as miasma, honey-dew, oppressive atmosphere, " bitter and sticky dew," gossamer-down, etc.; these have all been alleged seriously and under due authority to be responsible for the disease. Perhaps the most prevalent opinion—widely existing up to the present day—is the agency of the dew in the production of the disease, grass and herbage " contaminated " with dew having been held—even by many scientific men—to be concerned intimately with the etiology of the malady. The restriction of all grazing until the dew evaporated from the grass, and the early stabling of horses in the evening, has been a procedure tending to safety long recognised by the South African horse-owner, while it has been widely asserted that grazing throughout the night in dangerous localities and seasons could be safely permitted, provided the animals were stabled again before dawn.

This crepuscular or twilight influence, together with the essentially paludal nature of the disease, led the writer to suggest the agency of flying insects (such as the mosquito), and to attempt the proof of the suggestion. The outcome of the experimental observation showed conclusively that horses could safely live in the deadliest localities and seasons, provided protection against mosquito attack was afforded either by stabling in gauze-covered boxes, in smoky stables, or—though less certainly—by the application of strong-smelling or pungent dressings to the animal's coat. Dew, grass, miasma, etc., were sufficiently disproved by cross-experiments undertaken at the same time. South African horse-sickness, therefore, is transmitted, like malaria of man, by the bite of a flying insect.

PERIOD OF INCUBATION.

The space of time elapsing between infection and the onset of febrile symptoms varies within considerable limits, dependent upon the degree of infection, point of entry of the virus, physical resistance of the animal, and other factors of varying nature. The average period of incubation under normal circumstances, however, may be placed at from six to eight days.

The writer has seen the termination of the period of incubation as early as the third day in cases where a latent and indefinitely suppressed condition of artificial infection has been made to assume the active stage through the imposing of a second infection upon the first. Conversely, horses have been inoculated intravenously—in the winter and under conditions of exact scientific observation—and have not commenced to react until the thirteenth week after infection.

The introduction of the virus directly into the blood-stream anticipates by some twelve hours the accession of febrile symptoms produced by subcutaneous inoculation. When the virulent material is introduced into the stomach the period is somewhat longer, and where such introduction is made into the rectum (in the form of an enema), the incubative period is often considerably prolonged, terminating with curious frequency on or about the fortieth day, and giving rise to a lengthy but non-fatal febrile attack.

As stated above, however, the period of incubation of the disease may be placed at about seven days.

SYMPTOMS.

The symptoms of this disease may be conveniently classed in three divisions—(*a*) *Acute or fulminating*; (*b*) *subacute or chronic*; (*c*) *ambulant or suppressed.*

Of these, the only form likely to arrest the attention of the casual observer is the acute.

1. **Acute Form.**—In this case the disease generally assumes a sudden and critical stage, attended or closely followed by the rapid development of a condition of suffocative œdema of the lungs, which terminates the animal's life within a few minutes. This manifestation of the disease generally follows active exercise, especially under the saddle. After or during a sharp trot or hand-gallop—during which no untoward symptoms have appeared—the horse suddenly becomes sluggish. An unusual heaving of the flanks warns the rider to dismount and slacken his saddle-girths. The distress rapidly increases; a slight frequent cough leads to the appearance of a nasal discharge of a clear serous fluid, slight at first, but rapidly becoming copious as the cough increases in frequency. Distress becomes great as the embarrassment of heart and lungs increases. The pulse is with difficulty appreciable; great uneasiness, rising and lying down, wide distension of the nostrils, and the ejection of the serous discharge in the form of small whipped-up flakes of foam, staggering and collapse, rapidly ensue, and the animal succumbs—a death painful to observe—from asphyxiation, the nasal discharge becoming copious, and forming a large mound of foam round the muzzle of the animal as it lies upon the ground *in articulo mortis.*

This train of symptoms is often developed with great rapidity, five minutes sufficing to complete the clinical course from commencement of objective symptoms to the fatal issue. Often, however—especially where signs of sickness have been observed early and exercise prevented—the above clinical picture is more slowly developed, and is merged into the subacute form.

2. **Subacute Form.**—Dulness and inappetence will often prompt the horse-owner to use the clinical thermometer, and so detect an elevation of the bodily temperature of some three or four degrees. Injection of the conjunctiva, together with a deep-seated blotching of the same and a slightly increased respiratory action, will probably be the only other detectable symptoms during the course of the subacute form for several days and until near its close. A soft cough, infrequent at first, becomes developed coincidently with a critical fall of temperature, marking the commencement of the exudative stage, which, when established, rapidly becomes more acute as the pressure of the effusion into and around the heart and roots of the lungs increases the congestive condition. Death, with development of more acute symptoms, *ut supra*, follows more or less rapidly after the establishment of the signs of effusion. The animal may survive, however, some eight or ten hours with a normal, or more frequently a subnormal, temperature, picking food listlessly and hanging its head between the paroxysms of coughing until equilibrium becomes disturbed and consciousness is impaired. The struggle to maintain a standing posture generally precipitates the fatal issue, and progressive asphyxiation closes a painful death scene.

Under this heading must also be included the form of disease known to the early Dutch settlers as " dik-kop " (" thick-head "). This subacute form affects—as its title denotes—more especially the head, and though disturbance of temperature exists, the main objective symptoms are confined to the head and neck, and consist in the outpouring of serosity, due to a transudation from the vessels, into the loose areolar tissues of the supra-orbital fossæ and muzzle, extending through the jugular space down the neck, chiefly in the region of the great vessels. The mechanical impediment thus afforded to the return of blood from the head produces a venous congestion of the buccal and lachrymal membranes and a dull, semicomatose condition, from which it is often difficult to rouse the animal. Acute symptoms often supervene, but slow and uncertain recovery frequently takes place from this phase of the disease.

3. **Ambulant or Suppressed Form.**—This form of horse-sickness frequently exists unsuspected. The conjunctival membrane, however, will often show a marked degree of blotching and suffusion in horses which have become infected either naturally or artificially. A distinct elevation of the temperature exists in many of these cases, but, provided no fast

or excessive work is performed, the animal shows no sign of illness or distress. Such suppressed symptoms generally pass—as would be expected—without attracting the serious notice of the horse-owner, and these subacute attacks generally subside—unless mismanagement evokes acuter symptoms—within a few days of their onset. The writer has strong reasons for concluding that such intermittent or mild attacks, by their frequent recurrence, tend to produce a considerable degree of immunity, or, more strictly speaking, to increase the resistance naturally possessed in varying degrees by horses. Cases have occurred in the course of the writer's experimental work where horses, after inoculation with a mild form or strain of the disease, have developed the subacute form, and have continued to react energetically for many weeks without other symptoms than a continued hyperpyrexia. In one instance an animal reacted thermically for twelve weeks without intermission, showing no symptoms of specific invasion other than the continuous elevation of temperature and the gradual emaciation attendant upon the same. When reinoculated some months subsequently with virulent material, this animal failed to react, showing thereby the accession of a more or less complete immunity. The writer is further of opinion that the above ambulant or suppressed cases constitute an animal a " bacillus-carrier " for the perpetuation of the disease, and such an animal is able, under suitable conditions, to furnish the means of transfer of the disease to other horses in its more acute form.

Other natural reservoirs of the disease must, of course, exist in localities where the disease is enzoötic, but outbreaks often occur apart from such natural factors, and can only be explained upon the above supposition, to which a parallel would not be wanting in other pathological directions.

MORTALITY.

This in the acute cases is 100 per cent. Recovery from the acute form is very rare. Cases are occasionally met with where a horse, though already discharging froth from the nose and " turned out to die," survives after a lengthy and critical illness, but the mortality of such cases may be put at or about 100 per cent.

Where the effusion into the pericardial and pleural cavities is slight in quantity, a better prospect of recovery is held out ; and if on the sixth or seventh day of the disease the temperature falls, while the character of the pulse improves, a favourable termination to the case may be looked for. Such instances, however, are comparatively rare, probably not exceeding 15 per cent. under favourable circumstances.

Recovery from the mild or ambulant form is the general rule, especially where the premonitory symptoms of elevation of temperature and injected membranes warn the owner of the necessity for precautionary measures.

MORBID ANATOMY.

The post-mortem appearances are striking, and not likely to be confused with those of any other disease. The most characteristic lesion—of such constancy as to be looked upon almost as pathognomonic—is an intense degree of injection of the villous portion of the stomach. The appearances are often so marked as to irresistibly remind the observer of the intense inflammation produced by arsenical poisoning. The appearances of the stomach, however, are not due to an inflammation of its structures in the strict pathological acceptance of the term ; in fact, the main features of an inflammatory process are absent. Pain is generally absent, and an animal will often continue to feed slowly till within a few minutes of death. The intense pain of gastritis is conspicuous by its absence. Although the intensity of the appearances seems to warrant the use of the term "hæmorrhagic" as applied to the gastric mucosa, no leakage of corpuscles from the vessels of the villous loops can be noted, nor are inflammatory or congestive changes present microscopically. The specific exudate, so characteristic of the disease in its incidence on other organs, is absent from the stomach.

The intestines—large and small—participate generally, to a greater or less degree, in the same obscure pathological condition mentioned above, being streaked and injected, sometimes over extensive areas, but without loss of surface or escape of blood elements.

In some experimental cases coming under the observation of the writer, the specific lesions of the disease appeared to be expended solely upon the gastro-intestinal tract, and no involvement either of heart or lungs existed sufficient to determine the fatal issue.

While absence of lesions from lungs and heart is sometimes, though rarely, found, it is upon these organs that the stress of the disease generally falls. In the typical pulmonary form, an exudation of clear amber-coloured serosity occurs, infiltrating and dissecting out the lobules of the lung and enveloping the bloodvessels ramifying between the same. Often the effusion is so great in quantity as to separate large areas of visceral pleura from the underlying lung structures, and by its pressure to leave the actual mould of the ribs and intercostal spaces clearly defined upon the lung surface.

No true pleurisy exists, though loss of surface occurs to a limited extent on the visceral layer in some cases of excessive exudation. The parietal pleuræ are always normal.

The cut surface of the affected lung exudes froth and serosity, both from the air-passages and interstitial spaces, but its specific gravity is always less than that of water.

The heart almost invariably shows hæmorrhagic blotchings upon its left ventricular wall, often to such an extent as to change the entire surface into a deep mulberry colour streaked with brighter-coloured blotches and stripes. The right heart is much less often affected, and never to anything like the same degree. The epicardium generally shows blotches and petechiæ, especially in the region of the cardiac grooves and upon the fibro-fatty tissue of the great vessels entering and leaving the heart, and it is in this latter situation that the exudative process appears to commence, and by its presence to establish the vicious cycle of circulatory derangement and impaired drainage which seems to be responsible for much of the mechanical embarrassment.

The pericardium often contains as much as a couple of litres of clear serosity of an average specific gravity of 1011, the composition of which appears identical with the effusion thrown into the pleural cavities, and in quantity varying from 2 to 6 litres, or even more. Tapping of the effusion in either pleural or pericardial cavities does not appear to act otherwise than as a temporary palliative. Restriction of drinking-water or extensive bleeding is not attended by greater routine success.

Œdema of the legs, sheath, etc., is absent, as is effusion into the abdominal cavity. Occasionally the effusion will be found in the subarachnoid space and coagulated round the nerve-trunks as they emerge from the spinal cord. In one case noted pressure symptoms and evidences of nerve involvement during life were the only symptoms noticeable, while on post-mortem examination the only lesions observed were the involvement of the brain and cord in the region of their serous coverings.

The blood changes are insignificant, its specific gravity remaining normal until the onset of excessive effusion raises its density. Coagulation-time is not noticeably affected. Cell changes are not marked. In experimental cases a fatal issue can generally be prognosticated from the falling in number and disappearance of the eosinophile cells, while, conversely, their increase, together with a general rise of the white cell count, denotes a favourable issue.

Urine and body secretions generally appear to remain unaffected.

TREATMENT.

Treatment is of little or no avail when once pronounced symptoms have developed. The use of bold doses of arsenic by the mouth has given better results in the experience of the writer than any other line of treatment. Cardiac, respiratory, or general stimulants, purgatives, depletives, excitants, all seem powerless to avert or modify the usual fatal issue. Rest is imperative in treatment, while warmth seems indicated, although experimental cases of a critical nature have frequently survived the cold of the winter's night without increase of noticeable symptoms.

PROPHYLAXIS.

The prevention of the disease has long baffled the scientific observer. Koch, Danyz, and Bordet (of the Institute Pasteur), Edington, and many others, have endeavoured to devise means of rendering the horse and mule immune to the disease. Theiler of the Transvaal—who has had wider experience of the experimental side of the question than any other worker—has devised successful means for the immunisation of the mule by serum-therapy, and mules are now efficiently protected in large numbers in South Africa every year.

The writer's system of protection by means of efficiently safeguarding the animal's stable, either by light gauze windows and doors, or by the generating of smoke in the stable by means of burning stable-litter, is also widely in vogue in South Africa, and confers efficient protection, which, however, is confined only to the stabled animal.

The problem at present engaging the notice of South African workers in this disease is the effective immunisation of the horse, and this is proving a more difficult problem than the production of immunity in the mule, between which animal and the horse a considerable difference of susceptibility exists.

There is every reason to hope, from the present rate of progress, that this problem will be solved in the near future, and a great disability thus removed from the South African horse-owner.

EPIZOÖTIC LYMPHANGITIS

By CAPTAIN A. OLVER, F.R.C.V.S., A.V.C.

Synonyms.—Farcin de Riviere; Farcin curable; Farcin d'Afrique; Japanese farcy; Sarraja (Arabic name).

Definition.—A contagious, inoculable disease, characterised by inflammation, suppuration, and ulceration of superficial lymphatics or of mucous surfaces; by a benign course, absence of systemic disturbance, low mortality, and a slight tendency to recurrence. It is caused by the *Saccharomyces farciminosus* of Rivolta, and the lesions, both in the cutaneous and nasal forms, somewhat resemble those of glanders-farcy.

Species Affected.—Epizoötic lymphangitis is usually met with in horses, asses, and mules only; but in Japan, where the disease is extremely prevalent, cattle are also reported by Tokishige to develop atypical lesions, in which the specific organism can be demonstrated under the microscope. This observation has not, however, been confirmed in other countries, where bovines appear to have resisted all attempts at experimental infection.

History.—A form of benign farcy has been known to exist in Southern Europe since the early part of the nineteenth century under the name of "farcin de Riviere."

No diagnostic difference, however, between this disease and true glanders-farcy was definitely established until, in 1873, Rivolta announced the discovery of the specific organism of epizoötic lymphangitis, which he then called the *Cryptococcus farciminosus*. Prior to this discovery, Pehrson in 1865, and Chenier in 1881, had, however, declared that this disease was distinct from true farcy, and the latter proved that inoculation of material from either condition never produced the other.

In 1883 Rivolta, in collaboration with Micellone, published a further contribution to the study of the malady, in which they definitely stated that the specific organism could be found in every case of epizoötic lymphangitis, and announced that they had succeeded in infecting susceptible animals experimentally both by cutaneous and subcutaneous inoculation.

In 1897 Tokishige published a detailed description of the disease as met with in Japan, and announced that he had succeeded in cultivating the organism on potato, agar, gelatine, and peptone bouillon. These observations were confirmed by numerous observers, notably Bassi, Marcone, Fermi and Arusch, Baruchello and Nocard, all of whom concurred in the classification of the organism among the blastomycetes.

More recently, Sanfelice has again repeated the experiments of Tokishige, Marcone, and Baruchello, and arrived at the same conclusions.

Geographical Distribution.—Epizoötic lymphangitis has been reported to exist in many countries in Europe, Asia, and Africa, and, owing to its liability to confusion with glanders, it is probable that it occurs in other countries than those recorded.

It is still very prevalent both in Italy and Spain, where it is said to decimate the mules in certain districts. In France, during the early part of the nineteenth century, it was very prevalent in the south-east, and was carried thence to the north by canal and river horses. Since 1850, however, it has gradually disappeared, largely owing to restrictive measures against glanders, with which it was at first confounded. It was at one time widespread in Sweden, and is said still to exist in Finland and certain districts of Russia.

The British Isles are now free, but the disease was introduced with horses brought back from South Africa after the conclusion of the Boer War of 1899-1902. It was, however, mainly confined to military animals, and was finally stamped out in the summer of 1904 without ever having become enzoötic.

In Japan it is so prevalent that every horse is expected to develop an attack at an early age, and is considered more valuable after recovery, as it is then held to be immune.

In China it was met with during the operations for the relief of the Legations in 1900-01, and appeared to be prevalent throughout much of the country; but glanders being also rife, exact information on this point was difficult to obtain.

Numerous outbreaks have been reported from time to time in British India, Burmah, and the Dutch East Indies, and the disease caused considerable trouble during the Thibet Expedition of 1903-04.

In Africa it has been known to exist for many years in Egypt and Algeria, and more recently cases have been reported in Senegal, Northern Nigeria, and the West Coast. During the Boer War of 1899-1902 it was spread throughout South Africa by animals imported from Italy and other infected countries, and is also stated to have been in existence there before the war.

Finally, it is very prevalent in Abyssinia, and is constantly introduced into the Anglo-Egyptian Sudan from that country.

Etiology.—The lesions of epizoötic lymphangitis, in whatever form, are the result of the proliferation of the causal organism, the *Saccharomyces farciminosus* of Rivolta, which, in the great majority of cases, is found in pure culture in unopened abscesses. This parasite, in the form in which it is most commonly met with in the animal body, takes the shape of an oval or elliptical cell, 3 to 5 μ in length, rounded at one end, and coming to a sharp point at the other. Each cell is provided with a strongly refractile double contour, which is a very diagnostic feature, and can be most easily seen when the smear, after fixing, is moistened with a droplet of distilled water, saline solution, or glycerine and water, and examined under a cover-glass with a magnification of 800 to 1,000 diameters. Fixing should be by alcohol, or alcohol and ether, rather than heat, and Canada balsam should not be used for mounting, or the refraction of the double contour will be lost.

A strongly refractile granule can usually be seen in active Brownian movement within each parasite when examined in the fresh state, and mounted in water as described above. Many of the Saccharomyces are included within mono- or poly-nuclear leucocytes, which are usually present in the pus in large numbers. Others are seen singly or in pairs, either free or included in leucocytes, while occasionally a group may be met with, massed together, where they have been set free by the rupture of the containing leucocyte, the nucleus and débris of the body-substance of which may be still visible.

When a number of parasites are included within a leucocyte in this way, some of them do not present the typical form described above. They are somewhat smaller and more rounded, and the refractile double contour is not so well marked. A group of this kind often presents an external form suggestive of a mulberry, the organisms being closely associated in an irregular mass.

As a rule, no other organism than the Saccharomyces is met with in pus from a freshly-opened abscess, but in discharge taken from a granulating ulcer numerous bacteria are to be seen. Occasionally, a few pyogenic cocci may be found in the interior of an abscess, but they do not appear to have any marked influence on the course of the disease.

Staining.—In order to confirm the diagnosis arrived at by examining unstained smears, some form of staining is necessary. For purely diagnostic purposes soda methylene blue is a useful stain, the smears being stained for a few seconds only, washed in water, and mounted in 2 per cent. saline solution or in glycerine and water for examination. The organisms do not all take this stain equally, but the capsule is only faintly coloured, while the cell contents are usually stained a deeper blue, so that the parasite can be seen as a sharply-defined oval stained a dark blue, and surrounded by an almost clear zone. Parts of the cell contents take the stain more than others, sometimes suggesting the presence of a nucleus, but a definite nuclear body can never be defined.

For a more detailed examination of the parasite the following method gives good results, provided care is taken to stop decolorisation before it has proceeded too far—viz.: Make a film on a glass slide by spreading out a minute portion of pus as thinly as possible. Dry and fix with alcohol and ether in equal parts. Stain for five minutes with Ziehl-Neelsen carbol-fuchsin, heating the slide sufficiently to cause steam to rise, and adding fresh stain when necessary to prevent drying up. Wash in water, and immerse for two minutes in the following—viz.: Iodine, 1; iodide of potassium, 2; water, 200. Wash and dry, and decolorise in pure aniline oil until a pink tinge remains in the thicker parts of the film only. Wash in xylol to stop the action of the aniline oil, and examine under a magnification of 800 to 1,000.

An aqueous solution of methylene blue may be used as a contrast, but this does not appear to give any advantage over the simple stain. Stained in this way, the capsules usually retain the carbol-fuchsin better than the cell contents, and many of them appear to be empty. In others, stained cell contents are visible, and are sometimes massed at one end or side of the capsule, but not as a definite nucleus.

In many of the empty capsules only a part of the original oval remains, resembling a half-moon in shape. These are probably the remains of dead organisms.

Occasionally, a cell may be seen with a distinct projection from one end of the capsule, and these projections are generally considered to be the buds of new cells, which are being formed from the old by a process of gemmation.

Other methods of staining which may be employed are the Claudius, Gram-Weigert, and the Gram-Nicolle, but they do not appear to give such good results as the above.

Artificial Cultivation.—Tokishige was the first to cultivate the Saccharomyces, which he did on potato, agar, gelatine, and peptone bouillon, and to note the formation of secondary and tertiary hyphæ as well as spherical bodies and granules in artificial cultures.

Numerous other workers have since repeated these observations, and Sanfelice, more recently, has succeeded in cultivating the organism under the microscope in a layer of agar on a glass slide covered with a cover-glass, and ringed with vaseline to prevent evaporation. In the growths so obtained he was able to observe the formation of hyphæ.

Growth in all artificial media is always slow, and never abundant. It takes place better in an acid than an alkaline medium, and does not appear to be much influenced by temperature within wide limits, growth being equally good either at room temperature or blood heat.

On agar, the growth takes the form of greyish-white colonies, which are raised above the surface of the medium, and are difficult to detach or break up. When fully developed, the surface of the growth becomes wrinkled, but vegetation is always slow, and the colonies may be scarcely visible after thirty days' incubation.

In fluid media, a flocculent growth falls to the bottom, and forms a thick white deposit. On potato, the growth resembles that on agar, but is more rapid, and the colonies are of a light brown colour.

Growth takes place mainly on the surface of gelatine, which is not liquefied, and the colonies, after prolonged incubation, resemble yellowish-white grains of sand.

Classification.—Considerable doubt has arisen from time to time as to the proper classification of this organism. Rivolta, Tokishige, Marcone, Baruchello, Fermi and Arusch, Nocard and Sanfelice, have all placed it among the blastomycetes, basing their conclusions on the appearance of the organism, which closely resembles a yeast cell, although it is much smaller, and does not cause fermentation in sweet wort; on the forms assumed in artificial cultures; and on the results of inoculation experiments on animals. Canalis, on the other hand, placed it among the coccidia, Piana and Galli-Valerio among the sporozoa, and Gasparini has endeavoured to prove that the organism should be classed with the protozoa. Ducloux, and more recently Thiroux and Teppaz, have described it as a leucocytozoon, the last two writers claiming that a karyosome with distinct chromatin affinity can be seen when the organisms are stained by Laveran's modification of the Romanowsky stain, but other observers have failed to detect anything in the nature of a karyosome in Saccharomyces stained in this way. They also suggest that the disease is transmitted by some biting insect, but the whole weight of clinical experience strongly supports the generally accepted view that infection—in the cutaneous form at least—almost invariably takes place through a wound, and it is constantly observed that no fresh cases of the disease occur unless there has been some lesion of the integument.

The results of inoculation experiments on susceptible animals also support the same view, particularly those of Sanfelice, who succeeded in reproducing the disease in a mule by subcutaneous inoculation with an artificial culture of the organism containing both hyphæ and cells, and in recovering typical Saccharomyces from the resulting abscess.

Resistance of the Virus.—The Saccharomyces are very resistant to ordinary disinfectant solutions. They are said to resist the action of a 5 per cent. solution of carbolic acid, and clinical experience tends to prove that the disinfectant solutions commonly in use for washing wounds are quite ineffective in preventing transmission of the disease by means of sponges or other dressing material. They are stated, however, by Rivolta and Micellone to be destroyed in a few minutes by a temperature of 80° C.

Clinical Symptoms.—In its most typical form the symptoms of epizoötic lymphangitis closely resemble those of glanders-farcy. An animal, hitherto perfectly well, is usually noticed to have developed somewhere on the surface of the body one or more subcutaneous nodules, usually disposed in a chain along the course of an inflamed lymphatic, and varying in size from a pea to a walnut. The hair covering these nodules falls out, leaving small circular patches of polished skin, under which abscesses develop and burst, giving exit to a peculiarly thick, tenacious pus, which is rather cream-coloured than yellow, and of the consistence of Camembert cheese. Applied to a glass slide, it tends to roll on the surface rather than to adhere to it, and it is frequently streaked with blood. Very soon after the opening of an abscess the discharge takes on quite a different appearance. If the coat is long, the surrounding hairs become matted together by a yellow exudate; and if this is removed, a small ulcerous opening will be found discharging a yellow, oily fluid, in which the Saccharomyces are not usually so numerous as in the original creamy pus. On closer examination these abscesses will be seen to be situated at irregular intervals along the course of an inflamed lymphatic vessel, constituting the chaplet or rosary, which is supposed to be characteristic of the disease. After they have opened, they take the form of crater-like ulcers, which tend to heal by granulation from the bottom. The inflamed lymphatic can be felt under the skin, resembling a hard cord, and extending from the original seat of infection towards the lymphatic glands, through which the lymph of the part is passed.

These chains of abscesses may appear at any part of the body, depending entirely on the original seat of infection, usually a wound, which, however, may have been very slight, and may have completely healed long before the abscesses were noticed.

As is to be expected from the frequency with which they are wounded, the limbs are a common seat of the disease, particularly on the insides of the forearms and thighs, but there does not appear to be any predilection seat. When lesions are extensive on any of the limbs, there is usually considerable swelling of the whole leg, resembling that of ordinary non-contagious lymphangitis, but with less pain and lameness than in an acute case of that disease.

The distribution of lesions may vary in different countries, owing to some difference in the conditions under which the animals live. For instance, in South Africa, in the majority of cases, the limbs were affected as the result of kicks and treads; and eye cases, except as a secondary infection, the result of rubbing an open sore, were very rare; whereas in the Sudan, where the disease was very common among donkeys and mules, the regions of the spur vein and upper part of the forearm were more commonly affected, owing to the habit the animals have of biting each other on these parts. Primary eye cases, too, were comparatively common, probably due to infection conveyed by flies from an infected wound. In South Africa it is probable that the eye-fringes, which were almost universally worn, prevented infection of the mucous membrane of the eye in this way.

In Japan the external genitals are said to be a very common seat of infection, probably owing to the fact that the ponies are not castrated, and attack each other in this region.

In Russia the nasal mucous membrane is said to be the most frequent seat of the disease, and in British India it is said to be a common sequel of castration.

All parts exposed to galling, such as the top of the withers, shoulder, thighs, and head, are correspondingly liable to infection. If the disease is not successfully treated at once, the lesions tend to extend—more rapidly in some cases than in others—until the neighbouring lymphatic glands are much enlarged, and the skin over the infected area may be enormously thickened, and studded with abscesses and ulcers in various stages of development.

Until quite late in the disease there is no evidence of systemic disturbance, as a rule, the animals feeding well and maintaining their condition until they are destroyed as incurable, or some important internal organ has become extensively diseased. Cases have, however, been described in which systemic disturbance was marked from the outset, or even before any external symptoms had appeared, and it is probable that this does occasionally occur. In less typical cases the disease is not always easy to diagnose, and the microscope should be constantly used to detect the presence of the specific organism.

Nothing more may be noticed than a wound which does not heal satisfactorily, or a dirty discharge from one or both nostrils, or from one or both eyes, usually with some tumefaction of the membrana nictitans, or a minute quantity of discharge may be detected in an old scar, or a small, crater-like ulcer inside the ala of one nostril, strongly suggestive of a glanders ulcer. Yet, in the discharge from any of these, the Saccharomyces may be found in abundance, and such an obscure case may be the source from which infection is kept up in a stud for an indefinite period.

A damp climate is considered to favour development of the disease, and this seems to hold good in the Sudan, where, in the wet region of the south, the disease assumed a much more virulent type than in the dry region of Khartum and Omdurman, where it appears to die out spontaneously, although fresh cases are constantly introduced with every batch of donkeys and mules imported from Abyssinia.

The proportion of cases in which mucous surfaces alone are involved appears to vary in different countries. In South Africa such cases were rare, while in the Anglo-Egyptian Sudan quite 10 per cent. of animals attacked were so affected, usually on the conjunctiva. Tokishige mentions several cases, and Pallin states that in some 7 to 10 per cent. of his cases the mucous membranes were affected. In his experience, apparently, the disease, in the majority of cases, was located in some part of the respiratory passages, usually the nasal mucous membranes.

Cases in which the conjunctiva only is involved are frequently very slow in developing compared to other seats of the disease, and until one has had some experience of this condition there is nothing in the appearance of the lesion to suggest epizoötic lymphangitis.

The first symptom usually noticed is a clear, rather thick mucous discharge, with little or no suggestion of pus. Frequently a small, pale pink, macerated mass of granulations may be seen, either on the membrana nictitans or at the commissure of the eyelids, or nothing may be visible until the eyelids are everted for examination. The eye itself is usually not involved, but the disease frequently extends to the adjacent skin of the face.

Occasionally, cases are met with in which subcutaneous nodules have formed, but have not gone on to abscess formation. They persist as hard, painless nodules under the skin, and cause no inconvenience unless pressed upon by some part of the harness. According to Tokishige, this is the usual form taken by the disease in Japanese cattle.

Morbid Anatomy.—If the skin is cut through and a corded lymphatic exposed in the early stages of the disease, it will be seen that the lesion is almost entirely one of the lymphatic vessel, which stands out distinctly as a hard, white cord, about the thickness of a goose-quill, and with abscesses along its course at intervals. When cut into, the walls of the vessel will be seen to be very much thickened and inflamed. The lumen varies considerably in size, being dilated in places and constricted in others, and the contents consist of a variable quantity of blood-stained lymph or pus. Very soon, however, other tissues become involved. A large quantity of yellow, gelatinous exudate and new fibrous tissue is formed, and the muscles, tendons, ligaments, and joints, eventually become involved in the inflammatory process. The whole limb then becomes uniformly thickened with a mass of young, lardaceous, fibrous tissue, and the capsules of joints and sheaths of tendons and ligaments may be so altered as to permanently interfere with the animal's usefulness, even when a cure has been effected.

The disease tends to spread from the periphery towards the heart, and the lymphatic glands through which the lymph of the part is passed become enlarged and embedded in a large quantity of gelatinous material, and ultimately, if not removed, are liable to form centres of suppuration.

The lesions of the nasal mucous membrane take the form in the first instance of well-defined, crater-like ulcers, but these tend to coalesce as the disease progresses, until the epithelium of the mucous membrane involved is to a great extent destroyed, and is commonly replaced by pinkish, fungous masses of granulation tissue.

The ulceration may be confined to the thickness of the membrane, or may have extended into the substance of the septum nasi and turbinated bones, causing extensive necrosis of these tissues. In such cases the breath becomes fœtid, breathing is interfered with, and there is usually some dirty discharge, generally from both nostrils. Well-marked snoring, however, such as may rapidly supervene or even be the first symptom in glanders, is not a prominent symptom. The submaxillary glands are frequently enlarged and may suppurate, but this is not constant, and there appears to be little tendency for them to become firmly adherent to the jaw, as is said to be the case in glanders.

In other cases the mucous membrane of the pharynx, larynx, and trachea may be similarly involved, and Tokishige states that lesions may be found in the lungs in the form of lobular pneumonia. He also states that greyish nodules, resembling those of glanders, may occasionally be met with, and mentions having found lesions in the liver, spleen, and bones. A case is also on record in which a considerable lesion of the mucous membrane of the bowel was found to be due to the presence of *Saccharomyces farciminosus*. Such cases are, however, very rare, and could only be recognised on post-mortem examination.

Differential Diagnosis between epizoötic lymphangitis and glanders, or any other condition, should present no difficulty, owing to the ease with which the causal organism can be detected under the microscope, and little reliance should be placed on clinical symptoms, owing to the many different forms which this disease may assume.

Where there is a possibility of glanders existing as a complication, the Mallein test may be resorted to, but otherwise the presence of the Saccharomyces is conclusive, and these may at times be met with in the discharge from a wound or scar presenting none of the recognised symptoms of the disease. It should, however, be borne in mind that the organisms are sometimes rare in the discharge from an ulcer which has been open some days, and in which healing has commenced, so that it may be necessary to examine the discharge on more than one occasion before the animal can be declared free. In such a case, moreover, the presence of the Saccharomyces is liable to be masked by numerous other organisms which have developed since the abscess burst, and which are not found, as a rule, in pus from a freshly-opened bud.

Clinically, the main points which characterise this disease, as compared with glanders, are—

1. The entire absence of fever and systemic disturbance in the great majority of cases.
2. The creamy character of the pus from a freshly-opened abscess.
3. The free granulation and tendency to healing of the ulcers.
4. The comparatively slow progress of the lesions in the nasal cavities, and their tendency to granulation and formation of fungous growths rather than destruction of tissue.
5. The greater tendency to suppuration of the submaxillary glands and the less tendency to induration and the absence of fixing of that gland to the maxilla when enlarged.
6. Less discharge from the nostrils than would be the case with equally extensive glanderous lesions, both nostrils being usually affected.
7. Non-reaction to Mallein.

A form of ulcerative lymphangitis has been described in France by Nocard, which must not be confounded with the disease under description. The lesions are said also to resemble those of farcy, but they are due to a specific bacillus, are easily curable, and do not cause induration of the lymphatic glands. Animals so affected do not react to the Mallein test. (See p. 220.)

Prognosis.—Under favourable circumstances for energetic treatment and thorough after-treatment, prognosis is favourable where cases are seen before the disease has made much progress and the lesions are in a favourable position for operation. Probably 90 per cent. of such cases should be cured. Where, however, the lesions are so placed that radical operation is almost an impossibility, the outlook is not so favourable. For instance, cases in the hind-leg, in which the popliteal glands are involved, or cases in which the external genitals or any of the mucous membranes were the primary seat of the disease. In any event, the fact should always be taken into consideration that there is a distinct tendency to recurrence for months after the case is apparently cured, and a long period of isolation is therefore necessary in every case.

Treatment.—Energetic treatment should be undertaken at the earliest possible moment, and should aim at limiting extension of the disease towards the nearest lymphatic glands. Provided that facilities exist for maintaining the operation wounds in a moderately aseptic condition during after-treatment, the best means of securing this end appears to be by complete extirpation of the whole of the diseased tissue, under an anæsthetic wherever possible.

To be successful, operation must be very thorough; and if the whole of the diseased tissue is removed, the resulting wound will heal very rapidly.

In a certain proportion of cases where the fore-limb is extensively diseased, it may be necessary to remove the whole of the prepectoral lymphatic gland, and care must be exercised in doing so to avoid opening the jugular vein.

All cases, however, are not suitable for this form of treatment, and a less thorough method must then be resorted to. This remark particularly applies to cases in which the mucous membranes are the seat of disease, when little can be done beyond ordinary antiseptic treatment, careful measures being at the same time adopted to prevent the spread of the disease.

When the lesions are cutaneous, each bud may be opened with the actual cautery and dressed with a powerful antiseptic, such as tincture of iodine, or perchloride of mercury in methylated spirit (1 in 250). In any case it is advisable to limit extension towards the lymphatic glands as far as possible by cutting through the lymphatic vessel above the infected area.

It is quite unnecessary, and indeed harmful, to apply the actual cautery or any caustic dressing to these operation wounds. In the great majority of cases they heal perfectly satisfactorily under thorough antiseptic treatment, and healing must necessarily be more rapid where no unavoidable tissue destruction has been carried out.

In cases where there is a great deal of gelatinous exudate and new fibrous tissue surrounding the diseased lymphatic, treatment is more difficult; but a good deal of such tissue may be removed successfully with the knife, in suitable positions, and satisfactory healing follows.

An operation for the total extirpation of diseased tissues should not, however, be undertaken, as a rule, unless the lesions are circumscribed in extent and well defined.

In all cases the greatest care should be taken to protect every wound or abscess from the attacks of flies.

Internal treatment by means of various preparations of arsenic, mercury, iodine, and quinine, have been extensively tried, but the results have not been encouraging.

Prophylaxis.—In this country, and under all circumstances where facilities for the thorough isolation and treatment of cases do not exist, immediate destruction is to be recommended on account of the contagious nature of the malady, the length of time required for treatment and subsequent isolation, and the tendency of the disease to recur.

Where treatment is to be undertaken, most thorough arrangements must be made for the isolation and treatment of the sick, the isolation of the convalescent, the daily inspection of the healthy in infected studs, and the disinfection of infected stables, clothing, stable utensils, men's clothing, etc. For this purpose heat, where it can be used, is probably most suitable, as the parasite is said to be destroyed by a temperature of 80° C. The difficulty with which the organism is destroyed by antiseptic solutions should be made known, and in the case of large studs strict instructions should be issued that no material or antiseptic solution which has been used for dressing one wound is on any account to be used for another. Sponges should be totally interdicted for this purpose, and tow freely supplied, so that the piece used for each wound can be discarded and subsequently burnt, a fresh piece being taken for the next case.

In this connection it should not be forgotten that a wound may be infected and infective before showing any symptoms of the disease. The writer has demonstrated this fact with the microscope on many occasions, and once found that an operation wound which had been apparently healed for many months had been responsible for keeping up infection in the particular unit to which the horse belonged, long after the disease had been stamped out elsewhere. The cicatrix of this scar used occasionally to become slightly sore at one particular spot about the size of a pea, and the farrier, with the best of intentions, used to apply a little antiseptic solution from his dressing-bucket with a sponge, and subsequently dress other wounds with the same sponge and antiseptic solution. The result was that a series of cases occurred in this particular unit after the most drastic measures had been taken to exclude all possibility of infection from known cases of the disease, which measures had been entirely successful elsewhere.

Little is known as to the length of time the organism can survive outside the animal body, but measures for the disinfection of stables, fences, yards, middens, and all harness, saddlery, grooming-kit, etc., which have been exposed to the chance of infection, should in every case be very thorough.

Great care should be taken to exclude the possibility of an apparently cured animal acting as a carrier of the specific organism. Such an animal might be responsible for keeping up infection in a stud for an indefinite period should it remain undetected.

From the nature of the disease it appears probable that ordinary flies, by settling on wounds, play an important rôle in conveying the infection from one animal to another, and for this reason every endeavour should be made to protect wounds under treatment from their attacks. Wherever possible, *all* wounds should be covered; and where this is not possible, some dressing should be used which will tend to keep flies away. The writer has found a dressing composed of creosote, turpentine, and linseed oil useful for this purpose, as well as being a very satisfactory wound-dressing.

The frequent cases of infection of the conjunctiva which occurred in the Anglo-Egyptian Sudan appear to have been the result of infection by flies, and it seems probable that eye-fringes are, consequently, useful in protecting animals from this form of the disease.

Wherever an outbreak is being dealt with, the microscope should be constantly in use, not only to verify suspicious cases, but also to examine discharge from apparently healthy wounds which are in any way exposed to the possibility of infection.

Owing to the long incubation period of this disease, said to vary from eight days up to as much as nine months, a period of at least six months after the cure of the last case should be allowed to elapse before a stud is declared free.

In the treatment of cases measures should be taken to prevent the animals from rubbing the wounds with their muzzles or eyes, which they are liable to do owing to the itching caused by the disease.

Epizoötic lymphangitis is scheduled as a contagious disease under the Diseases of Animals Acts in Great Britain and Ireland.

APPENDIX

ANTHRAX

The Presence of Anthrax Bacilli in the Blood during Life.—According to Friedberger and Fröhner,* anthrax bacilli can be found in the blood only a very short time before death. This is explained as follows : " When the bacilli first penetrate from the local infected part into the blood, they fail to increase in it to any marked extent, on account of healthy blood having an unfavourable action on them. Consequently they disappear from it, and become deposited in the internal organs, especially in the spleen, lungs, and liver, where they freely multiply. It is only after the anthrax vegetation has made a certain amount of progress in these internal organs that the blood loses its germicidal properties and becomes a good nutrient material for the bacilli, which then increase enormously in that fluid " (Frank and Lubarsh). Hence a bacteriological examination of the blood during life very frequently gives a negative result, because the bacilli chiefly congregate in the internal organs. An exact diagnosis can, as a rule, be arrived at only after death, in consequence of the fact mentioned, and also because the symptoms (when observed) resemble those met with in other diseases.

Caulton Reeks is of opinion that the bacilli are present in the blood-stream prior to death, but not for a sufficient period to afford assistance in diagnosis. In a case of anthrax in the horse, he examined the blood and the gelatinous exudation from the swellings in the throat during life, but failed to demonstrate the presence of bacilli. He also observed a steer in the last stages of the disease, and made, within three minutes of the final struggles, film preparations from one of the peripheral veins at the base of the ear. These preparations, when stained and examined, were swarming with anthrax bacilli. He concludes that the disease commences in the intestinal bloodvessels, and from these invades the general blood-stream, so that it is safe to state that the bacilli are certainly in the blood-stream during life, but only reach the external vessels and capillaries on the approach of death.

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* " Veterinary Pathology."

Period of Incubation.—This was one of the points at issue in a recent law case, in connection with the alleged existence of anthrax in a number of cattle that succumbed to a mysterious disease; hence we deem it advisable to state what is known on the subject. According to Friedberger and Fröhner, "there have been no conclusive researches as to the duration of the incubation of anthrax in the usual form of infection of horses and cattle from the intestine." Feeding experiments carried out on sheep with spore-bearing bacilli have shown that the animals, without exception, died within two days after the feeding. In some feeding experiments carried out on horses and cattle, it has been shown that the incubation period in these animals appears to be from two to three days. Clinical observations on animals grazing on pastures in an infected locality gave similar results.

Anthrax in Birds.—The inoculation experiments of Feser and Œmler demonstrated that small birds, especially when young, can be readily infected with anthrax. The larger birds are less susceptible, but birds of prey were found to be immune. Under ordinary circumstances birds do not contract the disease, but during an epizoötic they may become infected by feeding on the flesh and blood of animals dead of anthrax. Cases are recorded in which farmyard poultry have become infected in this manner. It has been demonstrated experimentally that birds can be rendered far more susceptible to the disease by cooling the body by partial immersion in cold water. The symptoms observed in birds vary according to the type of the attack. In the peracute form death may occur suddenly, or after a few hours' illness, with staggering, tremors, violent convulsions, and a sanguinolent discharge from the mouth, nostrils, and cloaca. In the acute form the course may be twenty-four hours; marked depression is present, with weakness, dyspnœa, sanguinolent diarrhœa, drooping wings, ruffled feathers, lividity of the mucous membranes and of the comb and wattles. Dark-coloured swellings appear on the comb, wattles, conjunctiva, tongue, palate, and webbed portion of the feet (Friedberger and Fröhner).

Gloss-Anthrax.—In addition to the note on p. 46 in connection with the above affection, the following is worthy of insertion: In July, 1912, Caulton Reeks met with a case in a steer in which the symptoms conformed with those generally attributed to gloss-anthrax. The tongue was greatly swollen and protruded from the mouth, and on its surface were two large vesicles of a dark livid colour. There was a large amount of swelling in the submaxillary region, and marked dyspnœa, approaching asphyxia, was present. The animal was reported dead the next day. A bacteriological examination of blood taken from one of the ears gave negative evidence of anthrax. Preparations from the exudate of the submaxillary swelling, the post-pharyngeal glands and from the emphysematous lesions in the tongue were examined. The pharyngeal-gland preparations showed occasional short bacilli, with several long, filamentous-looking structures. The preparations from the tongue showed an almost pure culture of the bacillus of malignant œdema. This observer is of opinion that some, at any rate, of these cases of so-called gloss-anthrax are in reality cases of malignant œdema infection.

With this view we are in entire agreement, and we consider that cases of this nature urgently require investigation, as they are by no means uncommon. Why they should be regarded as anthrax, in the absence of a bacteriological examination, is one of those mysteries in veterinary nosology which cannot be explained. Until it is demonstrated that the lesions in the tongue and pharyngeal region really depend on anthrax, and are either a local manifestation of the disease or an associated condition with the generalized affection, the term "gloss-anthrax" should be eliminated from veterinary nosology.

Public Health.* — Anthrax in man always results from infection, either through the skin, the intestine, or the lungs. The chief source of infection is through wool, hair, and hides, and also through wounds inflicted during the skinning and handling of anthrax carcasses, or in conducting post-mortem examinations. Hence workers in wool and hair, also tanners and butchers are specially liable to the disease.

Two chief groups of the disease are recognised in man—viz., *external* and *internal* anthrax.

External Anthrax occurs under two forms—viz., *malignant pustule* and *malignant anthrax œdema.*

In *Malignant Pustule* inoculation usually occurs on the hands, arms, or face. The lesion takes the form of a small papule, which soon becomes vesicular, and around this inflammatory induration occurs. Within thirty-six hours a dark brownish eschar covers the seat of inoculation, and in the vicinity a series of small vesicles may be observed. Marked œdema is present, the inflammation extends to the lymphatics, and the lymph glands in the vicinity are involved. Unless energetic treatment be adopted, a fatal termination will result in from three to five days. In mild cases recovery may take place without severe constitutional symptoms. The eschar gradually sloughs out and healing occurs.

Malignant Anthrax Œdema.—In this form papules and vesicles are absent. There is extensive œdema, which generally succeeds to the constitutional symptoms, and the regions affected are the eyelid, the head, hand, and arm. Gangrene may result in consequence of the intensity of the œdema, and a considerable surface is involved; the constitutional symptoms are very grave, and death usually occurs.

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* For the above information we are indebted to Osler's "Practice of Medicine."

Internal Anthrax—(*a*) *Intestinal Form.*—This is believed to occur from eating the flesh or drinking the milk of animals suffering from anthrax; it is also said to result from external anthrax if the bacilli are carried to the mouth. Vomiting, diarrhœa, moderate fever, and pains in the legs and back, are observed. In acute cases dyspnœa, cyanosis, and convulsions or spasms of the muscles, are present, hæmorrhage may occur from the mucous membranes, and occasionally small phlegmonous areas or petechiæ are observed on the skin. The spleen is enlarged, and the blood is dark and remains fluid for a long time after death. An epidemic of the disease has been reported by Butler and Karl Huber, in which twenty-five persons were attacked after eating the flesh of an animal suffering from anthrax, and six succumbed in from forty-eight hours to seven days.

(*b*) *Wool-sorter's Disease.*—In this the infective material has been swallowed or inhaled with the dust. Hair and wool imported into Europe from Russia and South America have been responsible for the majority of the cases. The bronchi are chiefly affected, but a pulmonary form of the disease is also described. Cerebral symptoms are most marked in some cases, and in four instances the cerebral capillaries showed the presence of large numbers of anthrax bacilli.

In very acute cases death may occur within twenty-four hours, with symptoms of profound collapse and prostration. In other cases the course is protracted, and delirium, diarrhœa, and unconsciousness occur. In some instances the internal forms of anthrax are associated with the external lesions of the disease.

Rag-Picker's Disease is a local anthrax of the lungs and pleura, with general infection.

Prophylaxis.—Rigid disinfection of hides, hair, wool, etc., should be carried out. Under the Diseases of Animals Act it is now illegal to open the carcass of an animal suspected to have died of anthrax. But, nevertheless, cases occur from time to time in which butchers contract the disease from skinning or dressing the carcass of an animal affected with anthrax. The infection takes place either through an abrasion of the skin of the hands or arms, or by a wound accidentally inflicted by the knife.

Under modern methods of veterinary inspection anthrax should be a comparatively rare disease in man, so far as infection from diseased carcasses is concerned, but, unfortunately, the presence of the affection in animals is often overlooked, and death is ascribed to other causes. In those cases of equine anthrax which are so often mistaken for acute enteritis special care is necessary in order to prevent infection to knackers, who skin and open the carcasses without suspecting the real nature of the disease.

EPIZOÖTIC CEREBRO-SPINAL MENINGITIS IN HORSES.

Serious outbreaks of the above disease have occurred in some of the Western States of America during 1912, especially in Kansas and Nebraska. Up to the time of writing nothing definite has been determined with reference to the etiology and pathology of the affection. Some observers attribute the Kansas outbreaks to the ingestion of certain moulds which are occasionally to be found on the provender of animals; hence the disease was termed "forage (mould) poisoning."

We learn, however, from the October issue of the *American Journal of Veterinary Medicine* that numerous cases of the disease have been reported on animals that had been fed on the best of forage, while considerable evidence was forthcoming from certain laboratory workers that the disease is a specific one.

The losses in the State of Kansas alone were enormous, and it was estimated that 20,000 horses succumbed to the disease. Colorado and Wyoming instituted a quarantine against horses from Kansas. The disease always exists to a limited extent in Kansas and Missouri, and has in two previous years been very prevalent in the former State, when it was commonly referred to as "cornstalk disease" and "blind staggers.' The etiological factor was believed to be a mould growing on partially worm-eaten corn. In the recent severe outbreak in Kansas the autopsies revealed, in a large number of cases, the presence of parasites—viz., the larvæ of sclerostoma and other strongyles—in extraordinarily large numbers, in the affected animals, but these were also found in horses that succumbed to other affections, and even in apparently healthy animals. In the December issue of the *American Journal of Veterinary Medicine* we find that the outbreaks of the disease suddenly ceased, so that it is doubtful if sufficient evidence was obtained to positively prove or disprove any of the theories advanced. A writer in the December number of the above journal suggests that the disease may depend on the presence of the larval form of sclerostoma in the cerebral bloodvessels, causing cerebral embolism, and states that the latter condition was observed in all the horses affected in the Kansas outbreaks.

Bostrom (State Veterinarian of Nebraska), in a report on the Kansas-

Nebraska outbreak, gave the following account of the disease :* "Horses of all ages were attacked, but chiefly animals between two and six years old. The disease is very fatal; not more than 5 per cent. recover. The course is rapid; most of the cases die within forty-eight hours, some succumb as early as twelve hours after the first symptoms are manifested, and a few linger for seven days or more. The symptoms observed are attributable to derangement of the central nervous system even in the earliest stages—viz., somnolence, dulness, muscular tremors of the face and neck, grinding of the teeth, paralysis of the muscles of deglutition, and generally of those of one side of the head and neck; later on muscular inco-ordination of the limbs, falling down and remaining flat on the side without manifesting any evidences of pain. In some cases rabiform symptoms are observed. The visible mucosæ are injected, and petechiæ are well marked, especially on the membrana nictitans. In the early stages the temperature varies between 103° and 106° F., and the pulse and respirations are almost normal. Later on the temperature falls, the respirations are accelerated, the pulse increases in rapidity, and finally becomes imperceptible. The lesions observed are "inflammation of the brain, spinal cord, and also of the meninges, hæmorrhagic infarcts in the lungs and petechial hæmorrhage in the spleen, and yellowish infiltration of the connective and adipose tissues of the body."

Other observers state that in typical cases of forage-poisoning due to worm-eaten, mouldy corn, the first observable symptoms are an inability to appreciate distance and a certain degree of violence in movements. The animal strikes the door-sill violently with one forefoot when entering the stable, runs against the manger with considerable force when approaching it to feed, and seems to eat in a voracious manner. In from three to twelve hours, depending on the acuteness of attack, the horse runs against objects such as a tree or a pillar of a gate; he constantly moves in a circle if left at liberty, always towards the one side; the gait is staggering; the sense of sight is completely lost; the animal becomes violent and unmanageable, and may become dangerous to the attendants by biting or striking in a vicious manner; the temperature is only slightly elevated; the pulse quick and weak; the respirations rapid, stertorous, and laboured. In cases not treated in the early stages death occurs in 90 per cent. in a period of from twelve to seventy-two hours from the manifestation of the first symptoms.

The lesions found in cases that die within thirty-six hours from the commencement of the attack are those of intense cerebral meningitis. In more prolonged cases there is an increased amount of fluid in the ventricles of the brain, and in instances lasting for more than three days several ounces of serum escape from the brain on section of the organ, and the cerebral structure shows evidences of softening in parts. The medulla and its coverings show similar lesions. No lesions can be detected in the spinal cord or its meninges.

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* *American Journal of Veterinary Medicine*, October, 1912.

Bibens, of Garfield, Kansas, reports recoveries from the disease, if treated in the early stage by ligation of the carotid arteries, and states that 50 per cent. of those operated on recover.

FOOT AND MOUTH DISEASE.

Since the section on the above disease was written outbreaks have occurred in various parts of England, and in 1912 the disease appeared in Ireland as well, starting as a small outbreak in a country district in Co. Dublin. The source of infection could not be traced, but as the disease had not existed in Ireland for at least thirty years previously, it is clear that the infection must have been introduced by some means from another country.

Minor outbreaks occurred during 1912, chiefly in the midland counties of Ireland, but no case was reported from the province of Munster. By the employment of stamping-out measures the disease was kept under control, and at the time of writing no fresh cases had been reported.

A peculiar feature in connection with recent outbreaks is that no foot lesions were observed.

A Departmental Committee was appointed by the President of the Board of Agriculture on November 17, 1911, to inquire into the circumstances of the recent outbreaks of foot and mouth disease, and to consider whether any further measures can be adopted to prevent their recurrence. The Committee commenced taking evidence on January 25, 1912, and thirty-two witnesses were examined.

With regard to the means by which the virus of the disease may be imported, "the Committee recognised that it was impracticable, even if it was not impossible, to erect an impregnable barrier against the introduction of the disease, but it was agreed that the risk was greater with certain articles, those most open to suspicion being:

"1. Hay and straw.

"2. Milk and milk products.

"3. Hides and skins, heads and feet, carcasses of calves in skins, vaccine seed lymph.

"4. Hoofs, horns, bones, and other animal offals.

"5. Persons and their clothing."

A Scientific Committee has been appointed to investigate the nature, origin, and means of transmission, of foot and mouth disease, and this work will take place in India at an early date.

In view of the importance of a correct differential diagnosis of foot and mouth disease, and from the fact that differences of opinion may occur with reference to isolated cases, we deem it advisable to draw attention more fully than we have done on pp. 784-787 to affections simulating foot and mouth disease.

The late Sir George Brown, Director of the Veterinary Department of the Privy Council, described a form of stomatitis or sporadic aphtha in Great Britain in 1885, which could be, and in fact was, mistaken for foot and mouth disease.

Bang in 1899, and Stribolt and also Andersen in 1901, described a pseudo foot and mouth disease occurring in cattle in Denmark.

In 1904, Mohler,* of the Bureau of Animal Industry, United States of America, described a mycotic stomatitis which has often been mistaken for foot and mouth disease. The affection has been designated sporadic aphtha, aphthous stomatitis, buccal ulceration of cattle, ulcer of the tongue, simple benign or non-contagious aphthous fever, mycotic aphthous stomatitis, and sporadic aphthous stomatitis.

The disease is characterised by inflammation and ulceration of the buccal mucous membrane, and may extend secondarily to the feet, and more rarely to other regions of the body. As regards etiological factors, the affection is attributed to the ingestion of forage containing fungi or moulds, and it is probable that more than one fungus is responsible for the production of the malady, as a number may act together. Various authorities have incriminated or suspected the *Uromyces* and the red and black rusts of the Trifoliaceæ. These fungi cause very severe irritation of the buccal membrane, producing either a catarrhal or an aphthous, and eventually an ulcerative, stomatitis. The fungus of colza, etc. (*Polydesmus exitious*), acts as a severe irritant to the mouth and feet of cattle, causing a severe inflammation, and sometimes giving rise to symptoms simulating those of foot and mouth disease. Some authorities have incriminated the fungi of the Graminaceæ (*Penicillium* and *Puccina*) as a cause of stomatitis. Mohler has failed to transmit the disease by direct inoculation. The affection generally manifests itself when a dry, hot period is preceded by rains, which are favourable conditions for the development of moulds and other fungi.

The first noticeable symptom of the disease is a difficulty in the prehension of food; rumination is suspended; there are frequent movements of the lips (smacking of the lips), with the formation of frothy saliva on their borders; sometimes the saliva falls from the lips in the form of drops. The buccal mucosa is red and hot, and in exceptional cases one may observe small vesicles, but these quickly become eroded, and small ulcers are formed, varying from 3 to 25 millimetres in diameter. When several of these ulcers coalesce, they form large irregular patches with a hæmorrhagic border and a depressed, suppurative surface, containing a brownish or yellowish material, which is soon replaced by granulations. These ulcers are usually situated on the gums in the vicinity of the incisor teeth, on the dental pad, inside the lips, and on the summit of the tongue; but they are also found inside the cheeks, in the interdental space, and on the back of the tongue. Erosions and exfoliations occur on the muzzle, and brownish adherent crusts form and extend around the lips and nostrils.

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* Abstracted from Circular No. 54, Bureau of Animal Industry, August 10, 1904.

Lesions on the limbs are observed in some cases. The region of the pasterns becomes slightly tumefied and painful; this may extend to the fetlock, but never beyond the hock or knee. Fissures may be found in the skin of the coronet, while those occurring in the interdigital space may run on to suppuration. In such cases the animal assumes the recumbent position, and is with difficulty made to rise. When forced on to his feet, he shakes each foot as if trying to remove some foreign body from between the claws. In some instances the teats and udder become the seat of superficial erosions, fissures, and crusts, and the secretion of milk may be diminished or may completely cease. Rapid emaciation occurs, owing to the difficulty in feeding. Slight fever may be present, but it is of short duration, and the pulse is increased in frequency. The disease is generally not of a serious nature. As soon as the cause is suppressed and rational treatment is adopted, recovery takes place in less than a week. Many mild cases recover spontaneously. In the grave or complicated form, when early treatment is not adopted, death may result in six to eight days. But even under these conditions the mortality does not exceed 5 per 1,000. The duration of the disease varies from seven to fifteen days, the average being ten days.

The affection can be mistaken for foot and mouth disease. But mycotic stomatitis occurs from time to time in herds widely separated from one another; it only attacks a few animals in each herd. The erosions are very rarely preceded by vesicles, vesicles are never present on the feet or on the udder, and, lastly, the disease cannot be transmitted by inoculation.

In the pseudo foot and mouth disease observed by Bang, Stribolt, and Andersen in Denmark, the disease occurred in isolated sheds, and attacked nearly all the cattle confined therein. The early symptoms were lameness, fever, loss of appetite, and diminution of milk. The lesions occurred in the mouth and on the limbs. The mucous membrane of the mouth was covered with small vesicles in the region of the lower jaw; slight salivation and nasal discharge were also present, and the skin of the neighbouring parts was covered with small circumscribed yellowish crusts. Later on the mucous membrane presented surfaces covered with a yellowish croupous exudate on the dental pad, gums, lips, and even on the fauces.

The skin of the limbs, especially the hind ones, was swollen from the coronet up to the knee or the hock, and showed a serous exudate which agglutinated the hairs, and on drying formed a layer 1 to 2 millimetres in thickness. The exudate proceeded from a generalised oozing from the skin, and no vesicles existed. In some instances these morbid changes extended to the inside of the thighs, to the udder, and occasionally to the abdominal wall. The interdigital spaces were not involved, simply showing a more or less marked infiltration. In some cases the inflammatory affection of the coronet was so severe that the coronary cushion became detached by a purulent secretion.

The definite cause of the affection was unknown. It seemed to be associated with feeding on cotton cake.

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BANG: Nogle Bemærkninger om Kvægsygdomme, der Kunne forveksles med Mund-og Klovesyge. Mannedsskrift for Dyrlæger, Band xi., 1899, p. 157.

STRIBOLT: En Mund-og Klovesyge lignenende Sygdom. *Ibid.*, Band xiii., 1901, p. 130.

ANDERSEN: *Ibid.*, p. 182.

Some Further Tests with the Causative Agent of Foot and Mouth Disease.—Siegel (*Berlin. Tierärztl. Wochenschr.*, vol. xxviii., No. 39, 1912, pp. 713-718)* gave a report of results obtained with eighteen bovines, which were either young bulls or young heifers, obtained from establishments where no foot and mouth disease had existed during the past year. The biology of the *Cytoryctes cocci* was further studied. The organism was cultivated on artificial media, and in seventeen out of the eighteen animals infected, either subcutaneously, intravenously, or by contact, the classical symptoms and lesions of foot and mouth disease were produced.

The Transference of Foot and Mouth Disease by Blood-sucking Insects.—G. Terni (*Zentralbl. f. Allg. Path. u. Path.-Anat.*, vol. xxiii., No. 1, 1912, p. 15)† stated that the *Stomoxys calcitrans* are the only insects which pass the winter in barns. They convey foot and mouth disease by sucking the blood of an infected animal during the febrile stage.

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* Translation in the *American Journal of Veterinary Medicine*, December, 1912.

† *Ibid.*

JOHNE'S DISEASE.

Since the discovery by Twort* of a medium on which it is possible to grow the specific bacillus of Johne's disease, and the fulfilling of Koch's postulates by Twort and Ingram with regard to this disease, a great deal of work has been done and published subsequent to the writing of the article on Johne's Disease. A brief outline of the chief work and references to the full papers are given below. The dimensions of the bacillus are now usually given as 1 to 2 μ, a little smaller than the size mentioned (p. 198).

HALFDAN HOLTH. *Zeitschr. f. Infekt. parasit. Krankh. u. Hyg. d. Haust.*, vol. xi., No. 5 (communicated March 21, 1912), pp. 378-387.

This author, working in the laboratory of Professor Jensen at Copenhagen, gives a brief account of the nomenclature, etc., of the disease, and a notice of the failures of other workers, previous to Twort, to devise a suitable medium on which to cultivate the bacillus. With cultures received from Twort early in 1911, and with strains isolated by himself on Twort's medium and modifications of it, he obtained negative results from the inoculation of rabbits and guinea-pigs. He inoculated calves, which had previously passed the tuberculin test, subcutaneously and intravenously, with no immediate result, and three months later the animals were tested with ordinary tuberculin without effect. Three weeks later, again, they were tested with avian tuberculin, and gave " typical reactions." Finally a " tuberculin " prepared from cultures of Johne's bacillus was used, and in one case this gave a typical tuberculin reaction.

The morphological and staining characters of the bacillus, Holth found to agree with those given by Twort and Ingram.

From experiments on guinea-pigs, with the object of testing whether Johne's bacillus is capable of conferring immunity against tuberculosis, the author concluded that a slight degree of immunity was established, but the results need further proof.

MEISSNER (Hanover) AND KOHLSTOCK (Bromberg). " Immunisierungsversuche beim Chronischen Infektiösen Darmkatarrh " (*Berlin. Tierärtzl. Wochenschr.*, No. 25, 1912).

With the object of obtaining a reagent with which to immunise animals against chronic pseudo-tuberculous enteritis, the authors devised a method of obtaining large quantities of Johne's bacillus in an almost pure condition. This was done by scraping the mucosa of the intestine (of an animal affected with the disease), in which the bacilli were very numerous. After treating with antiformin (Uhlenhuth's method), and after various processes of washing and centrifuging, the sediment was dried, and found to consist very largely of Johne's bacilli. To this powder the name "enteritidin" was given. Sero-diagnostic experiments with enteritidin did not give satisfactory results. Experiments testing its power of immunising animals against Johne's disease were more successful, and in certain cases an immunity was established.

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* Proceedings of the Royal Society, September, 1910.

Further experiments by these authors with regard to the time during which the disease may remain latent in naturally infected calves showed that the period is a very long one—possibly as long as two years in some instances.

(In conjunction with Trapp, Meissner has published some of the most valuable work regarding experimental inoculation with this disease—*Mitteilungen des Kaiser Wilh. Instituts f. Landwirts. in Bromberg*, Band ii., Heft 3, March, 1910.)

TWORT, F. W., AND INGRAM. "Some Further Researches on Johne's Disease" (*Veterinary Record*, September 14, 1912).

In this paper were given further results regarding the testing of naturally infected and innoculated animals with diagnostic vaccines prepared from pure cultures of Johne's bacillus. Three naturally infected animals inoculated intravenously with a vaccine prepared from a pure culture grown on glycerine peptone beef broth, containing a glycerine extract of the *Bacillus phlei*, showed rises in temperature from normal to 105·0°, 106·1°, and 104·8° F., in one, four, and five hours respectively. Similar results were also obtained with artificially inoculated animals (calves).

These authors have also induced one strain of this bacillus to grow on an ordinary glycerine peptone beef broth medium, and also on glycerine liver broth, without the addition of any dead acid-fast bacilli or their extracts.

They have also succeeded in inoculating two goats with the disease from pure cultures. To a small dose of the diagnostic vaccine referred to above, these animals reacted well (normal to 106·4° and 105·4° F. in nine and ten hours respectively), the vaccine being in this case administered subcutaneously. On post-mortem examination the animals showed characteristic lesions of Johne's disease, from which the bacillus was again recovered in pure culture.

TWORT, C. C. "The Agglutination and Complement Fixation Reactions in Animals experimentally inoculated with Johne's Bacillus, with Special Reference to the Relation of this Bacillus to Other Acid-Fast Bacilli" (*Centralb. f. Bakt.*, Band lxvi., Heft 2-4. October, 1912).

The sera of eighty-eight animals obtained from various sources were tested by this author *re* complement fixation and agglutination with *Bacillus phlei*, human, bovine, and avian tubercle bacilli, and Johne's bacillus.

The results given show that such tests for diagnostic purposes would only be of value in cases in which the animals were known to be free from tuberculosis. The serum of a naturally infected bull (Johne's disease) gave a good positive agglutinin reaction on three different occasions up to a dilution of 1 : 15.

MEYER, K. F. Philadelphia Pathological Society, October 10, 1912.

The author has inquired into the prevalence of the disease in the United States of America, and has isolated the causal bacillus in pure culture by Twort's method from cases of the disease occurring in Philadelphia.

MCFADYEAN, SHEATHER AND EDWARDS. *Journal Comparative Pathology*, vol. xxv., p. 217 *et seq.*, October 19, 1912.

These authors report on eighteen cases in bovines and one in a Welsh ewe. Using the method of taking rectal scrapings suggested by Markus in 1904, they obtained very varied results, some examinations proving negative, other films being very rich in acid-fast bacilli. In microscopic examinations of fæces the same was found—viz., that the number of bacilli present seems to bear very little relation to the condition of the ileum and other parts of the intestine usually most severely affected.

Careful examinations were made of different portions of the bowels, with the object of determining which region is usually richest in acid-fast bacilli. The results, shown in graphic form, appear to indicate that, as a rule, the lower part of the ileum and the neighbourhood of the ileocæcal valve is usually the predilection seat of the bacilli.

O. Bang's avian tuberculin test was applied to fourteen animals. On the first test the temperature reached 106·4° F. in one case, but in no other did it exceed 104° F. On re-testing the case which reacted to 106·4° F. after a month's interval, there was no result. Four more of the fourteen cases were again tested after an interval of a month or more (one case receiving a large dose of avian tubercle bacilli intravenously), and in these the maximum temperatures were 105° (intravenous injection), 105·8°, 104·4°, and 105·4° F.

The ophthalmic tuberculin test with avian tuberculin, applied in six cases, gave two positive results and four negative. The intradermic test also gave no valuable information.

In the second portion of their report these authors confirm Twort's method of isolating the bacillus of Johne's disease by the incorporation of dead acid-fast bacilli or their extracts or products (tuberculins) in any good ordinary laboratory culture medium, and have succeeded in isolating the bacillus in twenty-three cases. They disagree with Twort and Ingram as to the relative value of the bovine and avian strains of tubercle bacilli for incorporation in media, and have obtained good growths using both these strains.

RABIES.

With reference to the footnote on p. 621, in connection with the quarantining of foxes imported into Great Britain from foreign countries, we find that the matter is provided for by the Importation of Canine Animals Order of 1909. This Order states that no fox shall be landed except by a licence of the Board.

TETANUS.

It is important to point out in connection with this disease that a relapse may occur in a case that appears to be approaching convalescence, or even when the horse seems fit to return to work. Instances of this kind are reported in the *American Journal of Veterinary Medicine*, November, 1912, under the heading of "An Unexpected Fatality from Tetanus." In one case, which lasted two weeks, all symptoms of the disease had abated, and the animal was ready to be put into harness, when symptoms of acute tetanus developed, and he succumbed in two hours. In another, the course of the disease was thirty days, and the mare had been in slings for three weeks. On removal from these she seemed perfectly cured, and was led back and forth to water. An acute attack of tetanus occurred on the thirty-second day, and she died the following morning.

Baker of Chicago stated that it is usual for 75 per cent. of cases of tetanus that live eighteen days to recover, and about 90 per cent. of those living twenty-one days recover, but he sometimes met with a fatality from the disease on the thirtieth or thirty-second day, and recorded an instance that on the twenty-second day had a relapse and died on the twenty-eighth day. He admitted that the cause for these relapses was mysterious, but was of opinion that when wounds or abrasions of the skin were produced by the harness of the slings, they constituted a new focus of infection, and generated more toxin, with the resultant fatal termination from it, in spite of the recovery from the primary infection.

However this may be, the matter is an important one from the point of view of prognosis, as we have seen cases that seemed to be approaching convalescence suddenly develop acute symptoms and succumb.

TRYPANOSOMES AND TRYPANOSOMIASIS.

Glossina fusca.

Newstead has shown that *G. fusca* is a West African species, and is distinct from that occurring in Eastern Africa, which is now known as *G. brevipalplis* Newstead.

Page 1056. **T. vivax** in small animals.

Torke and Blacklock (1911) succeeded in infecting rabbits and white rats with a strain of *T. vivax* originating in the Gambia. Only a small percentage of the inoculated animals became infected.

Page 1059. **Human Trypanosomiasis.**

A reference to Taute's original paper shows that this experiment was carried out with an animal trypanosome and not with *T. gambiense*, as the extract in the *Sleeping Sickness Bulletin* would infer.

Taute, however, subsequently (1911) proved that *G. morsitans* can transmit *T. gambiense*. Kleine's failure is ascribed to the work having been conducted at an elevation, since Kinghorn and Torke have conclusively shown that a certain degree of heat is essential to the cyclical development of *T. rhodesiense* within *G. morsitans*.

Page 1060. **Transmission of T. rhodesiense.**

Kinghorn and Torke (1912) have shown that *G. morsitans* can transmit *T. rhodesiense* in the Loangwa Valley, the period of development in the fly being about fourteen days. They further proved that both wild game and domestic stock harbour a trypanosome indistinguishable from *T. rhodesiense*, and may thereby act as reservoirs.

Page 1063. **T. brucei.**

It appears extremely probable that in Zululand, Bruce was working with more than one strain or species of trypanosome. The one sent to Europe and described by Plimmer and Bradford is clearly monomorphic, and remains so even after being maintained at various laboratories for upwards of fifteen years. None of the strains originating from the dog sent to Europe show the short forms now described by Bruce from his Zululand and Uganda specimens. It is known that several trypanosome species now occur in the neighbourhood of Umbombo, where the original investigations were carried out.

CORRIGENDA.

Page 47, line 13 from top, *for* "Charbert" *read* "Chabert."

,, 198, *for* "Jöhne" *read* "Johne," also throughout the section.

,, 471, line 12 from top, *for* "infectiöse" *read* "infektiöse."

,, 651, line 14 from bottom, *for* "cocco bacillus" *read* "coccobacillus."

,, 706, line 4 from bottom, *for* "Gaus" *read* "Gans."

,, 731, line 2 from top, *for* "*horoughly*" *read* "*thoroughly*."

,, 1031, lines 13 and 16 from bottom, *for* "Hæmophysalis" *read* "Hæmaphysalis."

,, 1036, line 11 from top, *for* "*Theilcri*" *read* "*Theileria*."

,, 1050, at end of second paragraph *add*, "and the destruction of all manure should be insisted on."

,, 1053, line 4 from top, *for* "1901" *read* "1910."

,, ,, "*Trypanosoma Congolense*" should be placed under a separate heading, as it constitutes a special group distinct from *T. Evansi.*

,, 1055, "*Trypanosoma Pecaudi*" should be in similar type to "*T. Dimorphon*," as it belongs to this group.

,, 1060, line 4 from bottom, *insert* "there" before "culpable."

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